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FINAL RISK ASSESSMENT
NIAGARA COUNTY REFUSE SITE
WHEATFIELD, NEW YORK
RISK ASSESSMENT
Work Assignment: C02089
(Ref. No. 1-635-259)

Prepared for:

U.S. Environmental Protection Agency

Contract No.: 68-W9-0003

TRC
TRC Environmental Corporation

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FINAL RISK ASSESSMENT
NIAGARA COUNTY REFUSE SITE
WHEATFIELD, NEW YORK

Prepared for

U.S. ENVIRONMENTAL PROTECTION AGENCY
Emergency and Remedial Response Division
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New York, New York 10278

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1.0 INTRODUCTION

1.1 Overview

The final rule of the National Oil and Hazardous Substances Pollution Contingency Plan (NCP, 1990) calls for conducting a baseline risk assessment as part of the Remedial Investigation (RI) at Superfund hazardous waste sites. The purpose of the baseline risk assessment is to determine whether contaminants identified at the site pose a current or potential future risk to public health or the environment in the absence of remediation. The analysis assists in evaluating whether remediation is necessary.

As part of the RI oversight effort at the Niagara County Refuse Landfill (NCR site) in Wheatfield, New York, TRC Environmental Corporation (TRC, formerly Alliance Technologies Corporation) conducted a baseline risk assessment, which includes both a public health and ecological risk assessment. This effort has been conducted under EPA Contract No. 68-W9-0003 (TES-6), Work Assignment C02089. The public health risk assessment presented in this report is primarily a quantitative analysis based on RI field sampling and analysis results and other information. The ecological risk assessment is both quantitative and qualitative and is based on previously published information in addition to information obtained during the RI.

The risk assessment evaluates actual or potential exposures to site contaminants under current and future land use scenarios. Existing site documents such as the RI report prepared by Conestoga-Rovers & Associates (CRA, 1991a) and EPA Biological Technical Assistance Group (BTAG) comments on the RI report (BTAG, 1991) have been utilized, and state and local officials have been consulted to determine likely receptors and exposure pathways for current and future land uses and demographics.

Receptors evaluated in the public health risk assessment include:

- Trespassers
- Residents
- Excavation Workers

There are five main components to the quantitative public health risk assessment. These are hazard identification, fate and transport evaluation, exposure assessment, toxicity evaluation, and risk characterization. The hazard identification step discusses the contamination at the site and includes the selection of contaminants of concern, i.e., those contaminants likely to pose the greatest risk to public health or the environment. The fate and transport of these contaminants in environmental media (e.g., soils and ground water) are then discussed. The exposure assessment uses available data on chemical releases from the site to estimate exposures to receptor populations. The toxicity evaluation describes the toxicological effects to public health from exposure to each contaminant and summarizes appropriate toxicity criteria. The risk characterization then estimates the carcinogenic and noncarcinogenic risks to human health attributable to site-related contaminants, based on toxicity data and calculated exposure doses. The ecological risk assessment generally includes the same components as the public health assessment.

This risk assessment was conducted in accordance with the following EPA guidance:

- *Risk Assessment Guidance for Superfund (RAGS). Volume 1 - Human Health Evaluation Manual (Part A)*, December 1989, Interim Final. (EPA, 1989a).
- *Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors"*. March 1991. Office of Solid Waste and Emergency Response. (EPA, 1991a).

- *Risk Assessment Guidance for Superfund (RAGS). Volume II - Environmental Evaluation Manual*, March, 1989, Interim Final. Office of Emergency and Remedial Response. (EPA, 1989b).

- *Superfund Exposure Assessment Manual.* April, 1988. Office of Remedial Response. (EPA, 1988b).

- *Guidance for Data Useability in Risk Assessment*, October 1990, Interim Final. Office of Emergency and Remedial Response. (EPA, 1990a).

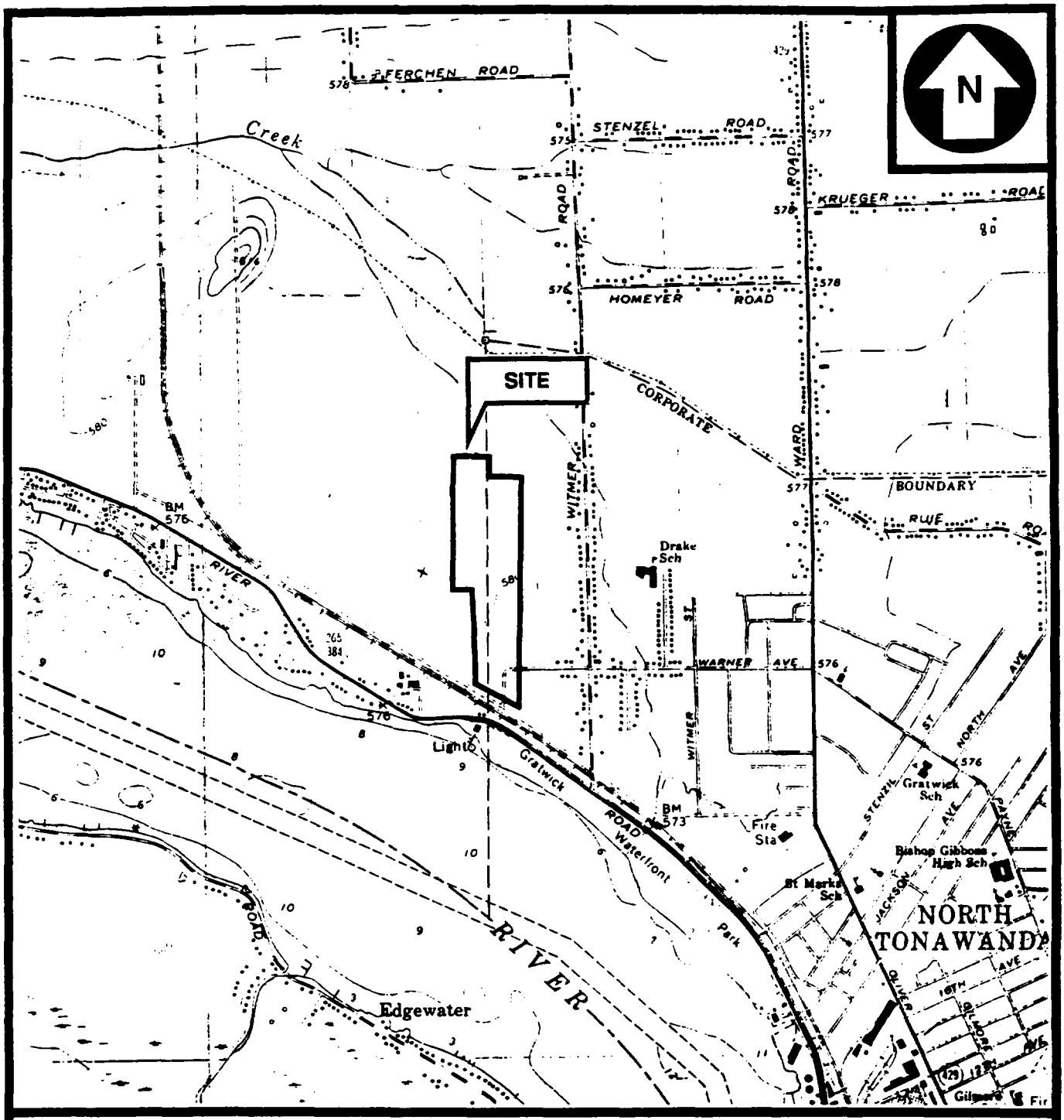
The report is organized into the following sections:

- Section 1 - Introduction
- Section 2 - Hazard Identification
- Section 3 - Environmental Fate and Transport
- Section 4 - Exposure Assessment
- Section 5 - Toxicity Evaluation
- Section 6 - Risk Characterization
- Section 7 - Uncertainty Analysis
- Section 8 - Ecological Risk Assessment
- Section 9 - Summary and Conclusions
- Section 10 - References

Appendices provide supporting information for relevant sections of the text.

1.2 Site Description

The Niagara County Refuse (NCR) site consists of approximately 50 acres in Wheatfield, Niagara County, New York. The NCR site is bounded to the east by low density residential land; to the north by woodlands and wetlands; to the west by agricultural land; and to the south by railroad tracks (CRA, 1991a). The Niagara River is located approximately 500 feet south of the NCR site. Figure 1-1 illustrates the area in which the site is located.



BASE MAP IS A PORTION OF THE FOLLOWING U.S.G.S. 7.5' SERIES QUADRANGLE: TONAWANDA WEST, NY, 1965

0 1000 2000 3000 feet

SITE LOCATION

NIAGARA COUNTY REFUSE SITE
WHEATFIELD, NEW YORK

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Figure 1-1.

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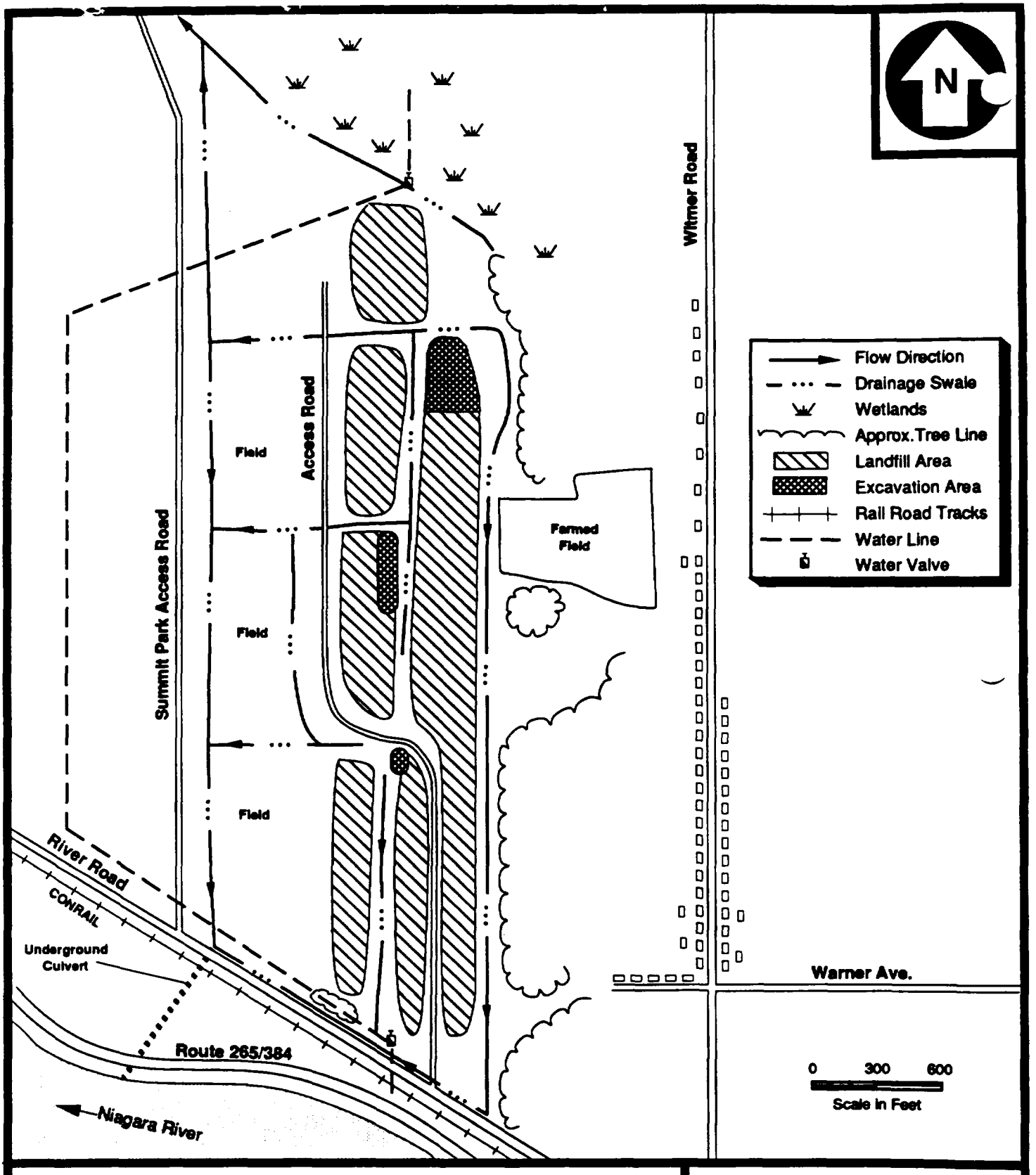
The landfill consists of six distinct cells which are separated by drainage swales and access roads. The cells are an average of twenty feet higher than the surrounding topography. The landfilled area is approximately 3,800 feet long, running north to south, and 720 feet wide. Refuse deposited in the landfill is reported to be between 15 and 39 feet thick (CRA, 1991a).

Drainage swales surround the perimeter of the landfill as well as the perimeters of each landfill cell. The swales channel surface water runoff to two discharge points. One discharge point is a 30-inch culvert at the southwestern corner of the site. The culvert runs under River Road and Route 265 and discharges directly into the Niagara River. The second discharge point is a 24-inch culvert which runs from the northwestern corner of the landfill, under the Summit Park access road, and apparently discharges into Black Creek (CRA, 1991a). During its operation, the surface of landfill had potholes or ditches that pooled surface water directly into the ground. Figure 1-2 illustrates the site and its immediate vicinity. A water main running through the center of the site (see Figure 1-2), was excavated and backfilled with waste prior to 1973. The main now runs west of the site.

During the RI, field inspectors noted approximately 30 leachate seeps emanating from the landfill slope and toe. No restrictive fences currently enclose the landfill. "Warning" signs were posted around the landfill perimeter; however, many of these signs were vandalized. Stressed vegetation was also noted during RI field work in a low lying wetland area immediately north of the site (CRA, 1991a).

1.3 Site History

The property on which the site is situated was part of Demler Farms until 1969. Historical aerial photographs indicate that the property was farmed as recently as 1951.



SITE VICINITY MAP

NIAGARA COUNTY REFUSE SITE
WHEATFIELD, NEW YORK

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Figure 1-2.

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In 1969, the Niagara County Refuse Disposal District (NCRDD) bought the property and began landfilling operations. NCRDD serviced all of Niagara County except for the towns of Niagara, Cambria, Wilson, and Newfane (CRA, 1991a). Until its closure in 1976, NCRDD accepted household, yard, agricultural, institutional, commercial, and industrial waste; demolition and construction debris; sewage treatment plant sludge; street sweepings; and used tires for disposal at the landfill. Municipal as well as privately operated waste collection firms disposed of waste at the site (CRA, 1991a). More than 100 industrial waste generators were known to have disposed of waste at the site. The types of waste disposed of at the site included heat-treatment salts, plating-tank sludge, tetrachloroethylene, polyvinyl chloride (PVC) skins and emulsion, thiazole polymer blends, polyvinyl alcohol, phenolic resins, and brine sludge containing mercury (EPA, 1988c).

In 1973, a landfill Operating Plan stated that approximately 39 of the 50 acres were landfilled to capacity. At that time, portions of three of the cells were being used for disposal. These areas are depicted as "excavation areas" in Figure 1-2. Five of the six landfill cells were excavated prior to 1973. The northernmost cell, which encompasses approximately 8 acres, was the only cell in use between 1974 and the landfill's closure in 1976. This cell has been described as "the industrial waste cell", although records and recent soil borings indicate that all of the cells received similar waste types (CRA, 1991a).

NCRDD reportedly excavated natural soils to a depth of approximately 11 feet and backfilled the ditches with refuse and industrial waste (CRA, 1991a). In 1973, as part of an effort to reduce leachate migration, NCRDD applied impervious cover material to the surface and sides of the inactive cells, sealed the landfill toe with clay, graded the landfill surface to reduce pooling, and removed all tires, barrels, and other loose material (CRA, 1991a). The landfill was closed in 1976.

1.4 Summary of Site Investigations

Investigations at the NCR site began as early as 1973, when the Niagara County Health Department (NCHD), working in conjunction with the New York State Department of Health (NYSDOH), collected leachate samples from the landfill's outfalls and surface water samples from the Niagara River immediately downstream of the outfalls. Samples contained high levels of heavy metals. However, a sample collected from an upstream location also contained significant concentrations of heavy metals (CRA, 1991a).

In 1980, EPA collected two surface water and five sediment samples from the onsite drainage swales, the ponded area north of the site, and the discharge point of the southernmost culvert. The surface water samples contained negligible concentrations of contaminants, but the sediments contained bis(2-ethylhexyl)phthalate (BEHP) (up to 6.9 mg/kg), polycyclic aromatic hydrocarbons (PAHs) (up to 3.2 mg/kg), and polychlorinated biphenyls (PCBs) (up to 0.32 mg/kg) (CRA, 1991a).

Eight additional surface water and sediment samples were collected from the drainage swales by New York State Department of Environmental Conservation (NYSDEC) in 1981. Surface water samples contained two volatile organic compounds (VOCs), toluene, and ethylbenzene; one base-neutral/acid extractable (BNA) organic compound, phenol; one pesticide, heptachlor; and two metals, copper and zinc (CRA, 1991a). In a separate study, Fred C. Hart Associates (Hart) collected surface water, sediment, and soil samples from the NCR site. Surface water samples contained methylene chloride, phthalates, phenols, and metals. Soils contained phthalates, PAHs, PCBs, and metals. Supplementary surface water samples were collected in 1981 by Recra Research. Elevated concentrations of phenols (34 mg/l) were detected in these samples (CRA, 1991a).

In 1982, the U.S. Geological Survey (USGS) collected sediment samples and drilled ten soil borings at the NCR site. Ground water monitoring wells were installed in two of the bore holes. Soil samples collected from borings drilled into the landfill cells contained organic contaminants whereas those collected from soil borings drilled along the perimeter of the landfill did not. Sediment and ground water samples contained three unspecified priority pollutants (CRA, 1991a).

In 1983, NUS Corp. Field Investigation Team (NUS/FIT), under contract to EPA, installed five bedrock ground water monitoring wells and collected and analyzed samples from each. In addition, NUS collected nine surface water and sediment samples from the landfill's drainage swales. Methylene chloride was detected in surface water and ground water samples, bis(2-ethylhexyl)phthalate (BEHP) was detected in one ground water sample at 35 µg/l, and endosulfan sulfate was detected in one sediment sample at 268 µg/kg. Concentrations of inorganic elements were reported to be comparable to background levels (CRA, 1991a).

In 1985, EA Science and Technology (EAST), under contract to NYSDEC, installed four ground water monitoring wells in four of the landfill cells. Phenols and numerous VOCs, including toluene, benzene, 4-methyl-2-pentanone, and ethylbenzene, were detected in samples collected from these wells. Samples collected from the northernmost wells contained the highest organic contaminant concentrations. Concentrations of metals, including arsenic, barium, chromium, iron, lead, manganese, and zinc, were detected at levels exceeding New York State's water quality criteria for drinking waters. Cyanide was also detected in samples collected by EAST (CRA, 1991a).

The NCR site was placed on the National Priorities List (NPL) in 1983. Pursuant to an Administrative Order on Consent (AOC) issued by EPA, a Remedial Investigation/

Feasibility Study (RI/FS) was initiated in May 1990. The Project Operations Plan (POP) for the RI/FS, submitted by CRA on behalf of the Potentially Responsible Party (PRP) Committee, was approved by EPA on May 23, 1990. The POP provided for:

- geophysical and topographic surveys;
- collection of air samples;
- completion of a quantitative biota survey;
- excavation of test pits; and
- installation of overburden and bedrock monitoring wells;
- collection of additional subsurface soil, leachate, surface water, sediment, and ground water samples.

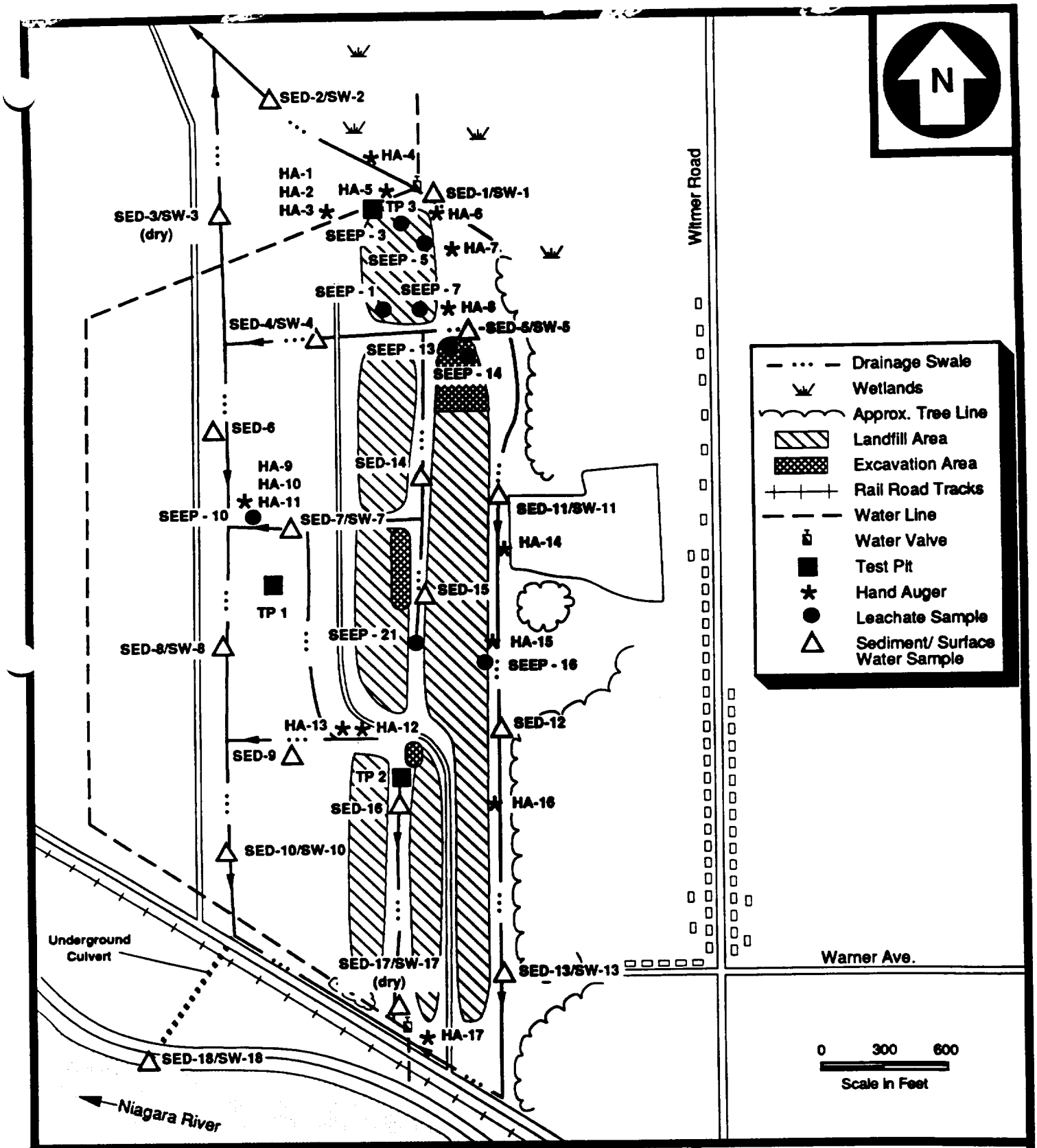
RI field activities were completed in August 1991. This risk assessment is based on data collected by CRA during RI field activities and on data collected by TRC during field oversight activities (TRC, 1991a). Sampling locations evaluated in this report are indicated in Figures 1-3 and 1-4.

1.5 Summary of Site Contamination

The assessment of site contamination is separated by medium. Contamination of surface soil, subsurface soil, leachate soil, sediments, ground water, surface water, leachate water, and air are summarized in this section.

1.5.1 Soil Contamination

For the purposes of assessing the risks associated with exposure to soils at the NCR site, data have been separated into surface (0 to 3 feet) and subsurface (2 to 15 feet)



SOIL, SURFACE WATER, AND SEDIMENT SAMPLING LOCATIONS

NIAGARA COUNTY REFUSE SITE
WHEATFIELD, NEW YORK

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Figure 1-3.

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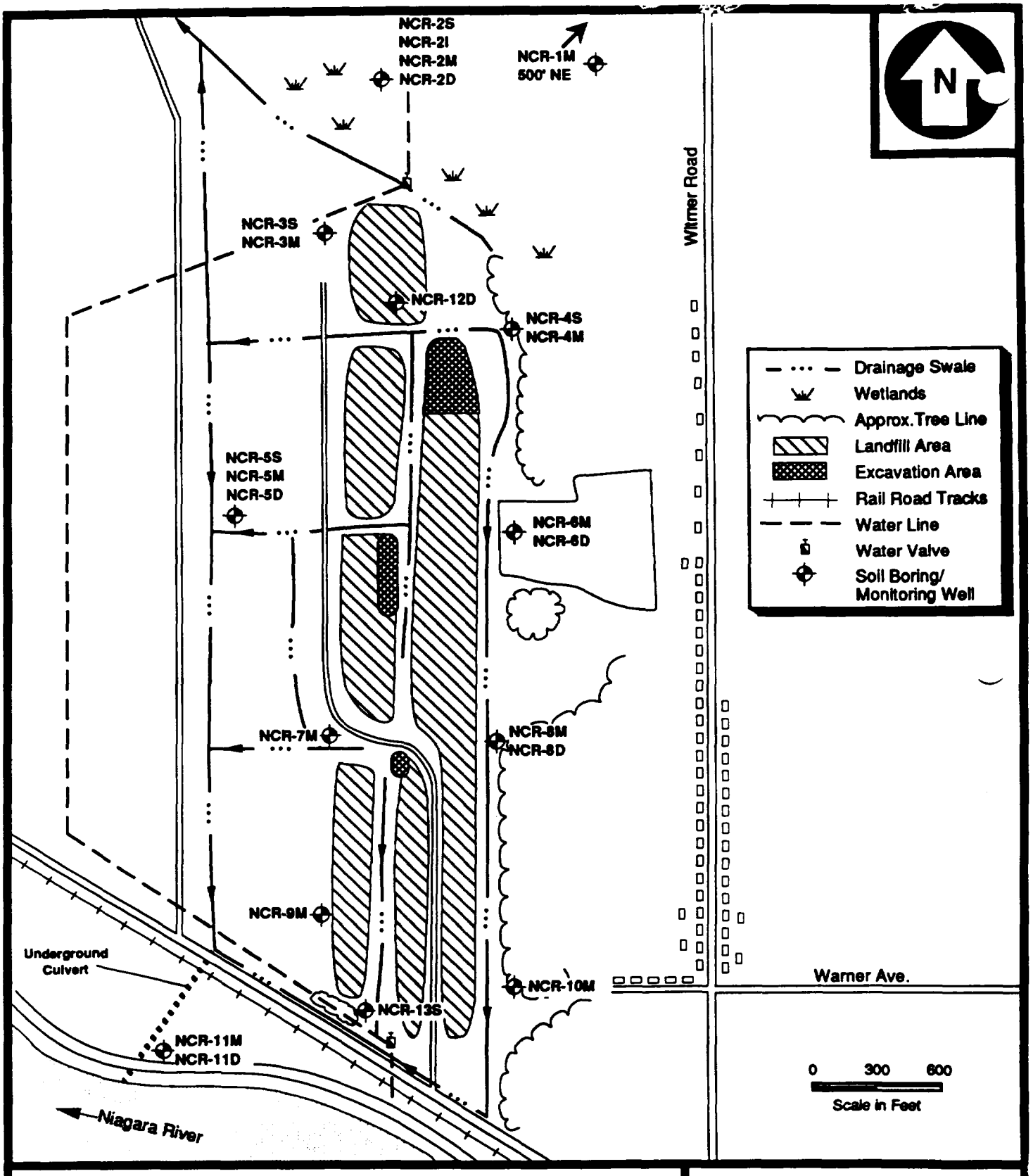

SOIL BORING AND GROUND WATER SAMPLING LOCATIONS

NIAGARA COUNTY REFUSE SITE
WHEATFIELD, NEW YORK

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Figure 1-4.

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depth profiles. Because soil borings were not collected at even two-foot intervals, there is a slight overlap between surface and subsurface soil depths. All samples collected from boring intervals which included soils at depths of one foot or less regardless if deeper soils were also included in the interval, were included in the surface soil grouping while all deeper soils were included in the subsurface or deep soil groupings. Although deep soil samples (> 15 feet) were collected, they are not expected to be encountered during present or potential future activities (utility service, excavation). Contaminants detected in deep surface soils could, however, leach into ground water under certain conditions and subsequently be encountered by ground water users.

Surface Soils

Surface soil samples were collected from twelve locations at NCR during RI field activities. Seven of these samples were collected from depths of 0 to 3 feet during monitoring well installation in July and September of 1990. The remaining 5 surface soil samples were collected from the shallow sand-silt unit using a hand bucket auger during November and December 1990. Seven VOCs and 13 metals were detected in these samples. In addition, BNAs and one pesticide were detected infrequently and at low concentrations.

- *Volatile Organic Compounds*

  The VOCs most frequently detected in surface soils were methylene chloride and acetone. Methylene chloride was detected in six samples at concentrations ranging between 6.5 and 22.0 micrograms per kilogram (µg/kg). Acetone was detected in four surface soil samples at concentrations ranging from 6.5 to 17

µg/kg. In addition, vinyl chloride was detected at 240 µg/kg in one surface soil sample (NCR-13).

- *Inorganics*

  All of the metals detected in the surface soils were present in at least 11 of the 12 samples collected. Barium, lead, manganese, and zinc were detected at concentrations exceeding 100,000 µg/kg. Arsenic, chromium, copper, nickel, and vanadium were detected at concentrations exceeding 20,000 µg/kg. Other metals detected include aluminum, beryllium, cobalt, and iron.

Subsurface Soils

Subsurface soils were collected from eight soil boring locations and the Test Pit 3 sampling location. Six VOCs, two pesticides, and 17 metals were detected in subsurface soil samples (2 to 15 feet) collected at the NCR site. Nine BNAs were detected in one sample (Testpit 3), but were not noted in the others.

- *Volatile Organic Compounds*

  Methylene chloride and trichloroethylene (TCE) were detected in four of ten subsurface soil samples collected at the site. Neither were detected at concentrations exceeding 17 µg/kg. Vinyl chloride was detected at one sampling point (NCR-13) at 210 µg/kg.

- *Pesticides*

  Two pesticides, alpha-BHC and heptachlor epoxide, were detected in one instance (separate samples) at 0.26 µg/kg and 0.58 µg/kg.

- *Inorganics*

  Aluminum and manganese concentrations in subsurface soils exceeded 1,000,000 µg/kg. Barium, copper, and zinc were present in concentrations exceeding 100,000 µg/kg. Arsenic, chromium, cobalt, lead, nickel, and vanadium were present at concentrations between 10,000 and 100,000 µg/kg.

Deep Subsurface Soils

Deep subsurface soil samples were collected from nine soil boring locations. Ten VOCs, eight BNAs, one pesticide, one PCB and seventeen metals were detected in soils collected at depths of greater than fifteen feet at the NCR site.

- *Volatile Organic Compounds*

  No volatile compounds were detected in more than two out of eleven samples analyzed. 1,1-Dichloroethylene (390 µg/kg) and vinyl chloride (190 µg/kg) were detected at the highest concentrations.

- *Base-Neutral/Acid Extractables*

  Nine BNAs were detected in deep subsurface soils. Benzylbutylphthalate and bis(2-ethylhexyl) phthalate were detected in 3 out of 10 samples analyzed. No other BNAs were detected more than once.

- *Pesticides/PCBs*

  One pesticide, delta-BHC, was detected in a single deep subsurface soil sample at a concentration of 25,000 µg/kg. Aroclor 1254 was detected in one out of eleven samples analyzed at an estimated value below the minimum detection limit.

- *Inorganics*

  Sixteen metals were detected in deep subsurface soils. Twelve of these; aluminum, arsenic, barium, chromium, cobalt, copper, iron lead, manganese, nickel, vanadium and zinc were detected in all samples analyzed. Antimony, cadmium, and mercury were detected in three or less samples out of eleven samples analyzed.

Leachate Soils

For this risk assessment, soils collected from visible leachate outbreaks were analyzed separately from other soils. Two leachate soil samples were collected at the NCR site from the northern most landfill cell during RI field activities.

- *Volatile Organic Compounds*

Two VOCs, methylene chloride and 1,4,-dichlorobenzene, were detected in one of the two leachate seeps at the site at concentrations of 67 and 1200 µg/kg, respectively.

- *Base-Neutral/Acid Extractables*

Nine PAHs, three phthalates, and 4-methylphenol were the only BNAs detected in leachate soils at the site. Pyrene (840 µg/l), benzylbutylphthalate (1200 µg/kg), and BEHP (750 µg/kg) were the only three BNAs whose maximum concentrations exceeded 700 µg/kg. Each of these were detected in one sample. Di-n-butylphthalate was detected in both samples but at concentrations less than 350 µg/kg.

- *Pesticides*

Five pesticides, delta-BHC, 4,4'-DDE, aldrin, dieldrin, and gamma-chlordane, were detected in one of two leachate soil sample (SEEP-1R). Concentrations ranged from 0.69 µg/kg (4,4'-DDE) to 3.2 µg/kg (dieldrin).

- *Inorganics*

Fifteen metals were detected in leachate soil samples collected at the site. Two of these, aluminum and iron, were detected at concentrations exceeding 1,000,000 µg/kg. Barium, lead, manganese, and zinc were detected at concentrations between 100,000 and 600,000 µg/kg. The remaining metals were detected at concentrations lower than 50,000 µg/kg. Of the nine

remaining metals, copper (47,800 µg/kg), vanadium (23,100 µg/kg), chromium (18,100 µg/kg), and nickel (18,700 µg/kg) were detected in the highest concentrations.

1.5.2 Sediment Contamination

The majority of the drainage swales at the site are seasonal. Twenty sediment samples were collected from the drainage swales transversing and encircling the landfill. Four VOCs, nine BNAs (primarily PAHs), nine pesticides, and 15 metals were detected in at least two sediment samples.

- *Volatile Organic Compounds*

  Methylene chloride and acetone were detected in greater than 50 percent of the sediment samples collected. Maximum concentrations of 73 and 89 µg/kg, respectively, were detected for the two VOCs. Benzene and 1,1,1-trichloroethane were detected infrequently and at low concentrations.

- *Base-Neutral/Acid Extractables*

  Eight of the nine BNAs detected at least twice were PAHs. Fluoranthene was detected at the highest concentration (330 µg/kg) and the greatest frequency (7 of 20). BEHP was detected in 11 samples of concentrations ranging from 110 to 3,900 µg/kg.

- *Pesticides*

  Delta-BHC and 4,4'-DDT were the only pesticides detected in greater than 20 percent of the sediment samples collected. Delta-BHC was detected in seven samples at concentrations ranging from 1.7 to 5.4 µg/kg and 4,4'-DDT was

detected in five samples at concentrations ranging from 6.9 to 77.0 µg/kg. The remaining pesticides, gamma-BHC, aldrin, heptachlor epoxide, dieldrin, 4,4'-DDE, endrin, and endosulfan II, were detected in three or fewer samples. Maximum concentrations ranged from 1.5 (gamma-BHC) to 28.0 µg/kg (4,4'-DDE).

- *Inorganics*

Fifteen metals were detected in at least 19 of the 20 sediment samples collected from drainage swales at the NCR site. Aluminum and iron, the maximum concentrations of which were 27,800 and 69,000 mg/kg respectively, were the most abundant metals detected. The maximum concentrations of the remaining thirteen metals were between 1.7 mg/kg (mercury) and 695 mg/kg (manganese). Barium was detected at concentrations ranging from 64.5 to 218 mg/kg, and lead was detected at a maximum concentration of 100 mg/kg.

1.5.3 Ground Water Contamination

For the purposes of this risk assessment, ground water samples collected at the NCR site were broken into "perimeter" (collected outside of the landfill cells) and "landfill cell" samples. Three landfill cell ground water samples (collected from one well) and forty-five perimeter ground water samples were collected at the site.

Landfill Cell Ground Water

The monitoring well from which the three landfill cell ground water samples were collected, NCR-12D, was drilled through the landfill cap in the northern cell. Samples were contaminated primarily with VOCs and metals. Concentrations of one pesticide,

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heptachlor, exceeded federally established maximum contaminant levels (MCLs). Heptachlor (MCL = 0.4 µg/l) was detected in one sample at a concentration of .0067 µg/l.

- *Volatile Organic Compounds*

  Twenty VOCs were detected in ground water samples collected from NCR-12D. Two of these were detected at concentrations exceeding MCLs. TCE (MCL = 5 µg/l) was detected in one sample at a concentration of 9.5 µg/l, and benzene (MCL = 5 µg/l) was detected in one sample at a concentration of 5 µg/l. Chloroform, tetrachloroethylene, toluene, and styrene were also detected, but at concentrations lower than MCLs.

- *Base-Neutral/Acid Extractables*

  Of the BNAs detected, phenol, 2-methylphenol, 4-methylphenol, and 2,4-dimethylphenol were detected in all three samples analyzed. The highest concentrations detected were reported for phenol and ranged from 775 µg/l to 2,650 µg/l.

- *Inorganics*

  Fifteen metals were detected in ground water samples collected from NCR-12D, two of which were present at levels equal to or exceeding MCLs. Beryllium (MCL = 1 µg/l) was detected in one sample at a concentration of

1 µg/l, and antimony (MCL = 5 µg/l was detected in two samples at concentrations of 26.6 and 44.75 µg/l.

Perimeter Ground Water

The primary contaminants in the perimeter ground water samples collected at the NCR site were VOCs, pesticides, and metals. Although numerous contaminants were detected, many were not consistently detected. Six BNAs were detected; however, none were present at levels exceeding MCLs.

- *Volatile Organic Compounds*

  Seven VOCs were detected in perimeter ground water samples, including benzene, methylene chloride, and acetone at low frequencies. Concentrations of benzene did not exceed MCLs, however, MCLs have not been established for methylene chloride or acetone.

- *Pesticides*

  Seventeen pesticides were detected at the NCR site in perimeter ground water samples. Seven were maintained as COCs. MCLs have been established for only two of these pesticides, heptachlor (MCL = 0.4 µg/l) and alpha-chlordane (MCL = 2 µg/l (chlordane)). Heptachlor was detected in 18 out of the 45 samples analyzed at concentrations ranging from .0007 to .09 µg/l. Alpha-chlordane was present in four of 45 samples at concentrations ranging from .00062 to .0022 µg/l. Delta-BHC, gamma-BHC, 4,4'-DDE, endosulfan sulfate, and endrin aldehyde do not have established MCLs for comparison.

- *Inorganics*

Twenty-one metals were detected in perimeter ground water samples collected at the NCR site. Five were detected at concentrations exceeding MCLs. The most prevalent of these were total chromium, nickel, and beryllium. Total chromium (MCL = 100 µg/l) was detected in 17 samples at levels ranging from 7.5 to 134 µg/l and beryllium (MCL = 1 µg/l) was detected in 8 samples at concentrations ranging from 1 to 3.1 µg/l. Nickel (MCL = 100 µg/l) was detected in 18 samples at levels ranging from 9.5 to 155 µg/l. Antimony (MCL = 0.5 µg/l; range - 23.1 to 69.8 µg/l) and cadmium (MCL = 5 µg/l; range - 4.4 to 5 µg/l) were detected less frequently.

Iron was detected in 42 of 45 samples at levels ranging from 59.5 to 108,000 µg/l. A secondary MCL (SMCL), which is a standard designed to control aesthetics and odor, of 300 µg/l is established for iron. In addition, an action level (a suggested standard), of 15 µg/l has been established for lead. Lead was detected in 11 samples at concentrations between 2.6 and 77.9 µg/l.

1.5.4 Surface Water Contamination

Surface water samples were collected from drainage swales and from visible leachate seeps at the NCR site. Eleven samples from drainage swales and eight samples from seeps were collected.

Surface Water (Drainage Swales)

VOCs, BNAs, pesticides, and metals were detected in the drainage swales at the site.

- *Volatile Organic Compounds*

Seven VOCs were detected in surface water samples collected from the drainage swales at the NCR site. Carbon disulfide was detected most frequently (three samples) and at the highest concentrations (maximum 8 µg/l). 1,1,1-Trichloroethane, 4-methyl-2-pentanone, tetrachloroethylene, toluene, ethylbenzene, and xylenes were detected at low frequencies and concentrations.

- *Base-Neutral/Acid Extractables*

Six BNAs were detected in drainage swales at the site. Only one of these, bis(2-ethylhexyl phthalate) (BEHP), was detected in more than two of the samples. BEHP was detected in eight of 11 samples at concentrations ranging from 0.7 to 1000 µg/l. Phenol, 2,4-dimethyl-phenol, diethylphthalate, and di-n-butylphthalate were also detected but infrequently and at low concentrations.

- *Pesticides*

One pesticide, delta-BHC, was detected in two drainage swale surface water samples collected at the NCR site. The maximum concentration of delta-BHC detected was .021 µg/l. 4,4'-DDT was detected in one sample at 0.04 µg/l. Gamma-BHC and heptachlor epoxide were detected at lower concentrations in one sample.

- *Inorganics*

Fifteen metals and cyanide were detected in surface water samples collected from the drainage swales. Aluminum, barium, lead, and manganese were detected most frequently. Aluminum, manganese, iron, barium, and zinc were detected at the highest maximum concentrations (> 450 µg/l). Arsenic,

chromium, copper, nickel, and vanadium were detected at maximum concentrations ranging from 30 to 100 µg/l.

Cyanide was detected in four of 11 samples. Concentrations ranged from 15.6 to 40.6 µg/l.

Leachate Surface Water

Water samples were collected from eight seeps emanating from the slopes of the landfill. Samples were found to contain numerous VOCs, BNAs, pesticides, and metals.

- *Volatile Organic Compounds*

  Five VOCs, 2-butanone (MEK), 4-methyl-2-pentanone, toluene, ethylbenzene, and xylenes, were detected in at least 50 percent of the leachate water samples collected at the NCR site. MEK (96 to 1400 µg/l), xylenes (12 to 1400 µg/l), ethylbenzene (2 to 600 µg/l), and toluene (3 to 410 µg/l) were detected in the highest concentrations. Of the VOCs detected less frequently, acetone (490 to 2200 µg/l) and methylene chloride (470 µg/l) were detected at the highest concentrations. Benzene was detected in three of eight samples at concentrations ranging from 36 to 50 µg/l.

- *Base-Neutral/Acid Extractables*

  Fourteen BNAs were detected in enough leachate water samples to be considered COCs. Four phenolic compounds, three PAHs, and four phthalates were detected. The four phenolic compounds were detected in at least 50 percent of the samples at maximum concentrations ranging from 960 to 3750 µg/l. Three other BNAs, naphthalene, diethylphthalate, and BEHP were

detected in at least half of the samples. Naphthalene concentrations ranged from 0.6 to 200 µg/l. Concentrations of diethylphthalate and BEHP did not exceed 55 µg/l. Maximum concentrations of seven other BNAs did not exceed 51 µg/l, with the exception of 4-chloroaniline, which was detected in one sample at 160 µg/l.

- *Pesticides*

Five pesticides were detected in leachate waters collected at the NCR site. Two of these were detected in three of the samples. Delta-BHC was present at concentrations between .019 and .18 µg/l and 4,4-DDT was detected in concentrations ranging from .044 to 0.11 µg/l. Gamma-BHC, heptachlor, and aldrin were detected less frequently. Heptachlor was detected in one sample at 1.1 µg/l.

- *Inorganics*

Fifteen metals were detected in leachate water samples collected at the site. All six were detected in all of the samples. Aluminum and iron were detected at concentrations exceeding 300,000 µg/l. Barium was detected at a maximum concentration of 7,610 µg/l. Manganese and zinc were detected between 1400 and 2700 µg/l. Of the remaining metals, lead (maximum concentration, 1010 µg/l) and nickel (maximum concentration, 157 µg/l) were detected at the highest concentrations.

1.5.5 Air Emissions

Emissions from two leachate seep locations (Seep-5 and Seep-16) were sampled for volatile organic contaminants in July of 1990. Benzene, methylene chloride, and toluene were detected and reported at estimated values at least one order of magnitude

below the New York state ambient air levels (CRA, 1991a). However, due to problems in sampling, much of the data were rejected during data validation. Insufficient data remained for use in the quantitative risk assessment.

2.0 HAZARD IDENTIFICATION

2.1 Data Evaluation

The following section describes the sources of the analytical data used in the risk assessment, and the methods used to statistically analyze these data.

2.1.1 Data Sources

Environmental sampling data used in this risk assessment were collected during the RI conducted by Conestoga-Rovers & Associates (CRA, 1991a). Prior to evaluation TRC reviewed the analytical results and accompanying documentation to ensure that all appropriate, validated data were included in the risk assessment data base. A number of questions about the data arose which were resolved through direct communication with EPA and CRA (CRA, 1991b). Based on discussions with EPA, the data from samples collected by TRC field oversight personnel, in addition to the data from samples collected by CRA during the RI, were included in the data base. TRC oversight samples consisted of true splits (collected at the same time and location as CRA samples), with results consistent with RI samples, and samples determined to not be true splits. True splits were treated as duplicates of the corresponding RI samples, while the other splits were treated as discrete samples.

2.1.2 Data Analysis

Analytical results were provided to TRC on laboratory summary sheets (generally Form Is); results of data validation were provided on CRA summary tables and had to be transcribed to the laboratory summary sheets. Data were manually entered from the Form Is into the TRC data base. Data for all environmental media sampled during

the RI were available. As mentioned previously, much of the data gathered during the air monitoring program, were rejected during data validation; thus there was not sufficient air data available for use in this risk assessment.

Samples from the following media were considered for evaluation:

- Surface Soil
  - 0 to 3 feet*
- Subsurface Soil
  - 2 to 15 feet*

  Deep subsurface soil
  - > 15 feet
- Ground water (shallow overburden, deep overburden, and bedrock)
  - Northernmost landfill cell (NCR-12D)
  - Landfill perimeter
- Drainage swale surface waters
- Drainage swale sediments
- Leachate soil
- Leachate water

*Due to slight overlap between surface and subsurface depths, all samples collected from boring intervals at depths of one foot or less were included in the surface soil group while deeper soils were included in the subsurface group.

Table 2-1 presents a list of all samples evaluated for the risk assessment and types of analytical results available. This risk assessment contains data from two ground water sampling events and one sampling event for all other media. The sample number listed on the table for each oversight and duplicate sample contains a suffix of SP and/or DUP, respectively. Suffixes I and II in all ground water sample numbers refer to the first and second sampling rounds, respectively. All sample numbers listed correspond to sampling points shown on Figures 1-3 and 1-4.

TABLE 2-1. SAMPLES INCLUDED IN THE NCR RISK ASSESSMENT

Medium/Area	Sample Number	Volatile Organics	BNAs	Pesticides/ PCBs	Inorganics
Surface Soil	HA-13	X	X	X	X
Surface Soil	HA-14	X	X	X	X
Surface Soil	HA-3	X	X	X	X
Surface Soil	HA-8	X	X	X	X
Surface Soil	HA-9	X	X	X	X
Surface Soil	HA-9DUP	X	X	X	X
Surface Soil	NCR-1 (1-3')	X	X	X	X
Surface Soil	NCR-11A (0-3.2')	X	X	X	X
Surface Soil	NCR-11B (0-3.2')	X	X	X	X
Surface Soil	[NCR-23 (0-2')] NCR-11B (0-3.2')DUP	X	X	X	X
Surface Soil	NCR-13 (0-0.8')	X	X	X	X
Surface Soil	NCR-4 (1-3')	X	X	X	X
Surface Soil	NCR-7 (0.0-2.0')	X	X	X	X
Surface Soil	NCR-8 (0.0-2.0')	X	X	X	X
Subsurface Soil	NCR-10 (2-4')	X	X	X	X
Subsurface Soil	NCR-10 (2-4')DUP [NCR-21 (6-8')]	X	X	X	X
Subsurface Soil	NCR-13 (2.5-3.5)	X	X	X	X
Subsurface Soil	NCR-2 (2.0-3.0')	X	X	X	X
Subsurface Soil	NCR-3M (4-6')	X	X	X	X
Subsurface Soil	NCR-5 (5.4-7.0')	X	X	X	X
Subsurface Soil	NCR-6 (2.0-6.0')DUP [NCR-6(2-4')SP]	X	X	X	
Subsurface Soil	NCR-6 (2.0-6.0')	X	X	X	X
Subsurface Soil	NCR-8 (3.0-5.0')	X	X	X	X
Subsurface Soil	NCR-9M (2.0-6.0')	X	X	X	X
Subsurface Soil	TESTPIT3R	X	X	X	X
Subsurface Soil	TESTPIT3SP	X	X	X	X
Deep Subsurface Soils	NCR-1 (44-46')	X	X	X	X
Deep Subsurface Soils	NCR-10 (26-28')	X	X	X	X
Deep Subsurface Soils	NCR-10 (44-46')	X	X	X	X
Deep Subsurface Soils	NCR-12 (22-26')	X	X	X	X
Deep Subsurface Soils	NCR-12 (44-46')	X	X	X	X
Deep Subsurface Soils	NCR-12 (44-46')DUP [NCR-12A(44-46')]	X	X	X	X
Deep Subsurface Soils	NCR-12 (22-26')DUP [NCR-12D(23')SP]		X	X	X
Deep Subsurface Soils	NCR-2 (24-24.7')	X	X	X	X
Deep Subsurface Soils	NCR-3M(24-26')DUP [NCR-3M(24-28')SP]	X	X	X	X
Deep Subsurface Soils	NCR-3M (24-26')	X	X	X	X
Deep Subsurface Soils	NCR-4 (55-57')	X	X	X	X

TABLE 2-1 (CONTINUED)

Medium/Area	Sample Number	Volatile Organics	BNAs	Pesticides/ PCBs	Inorganics
Deep Subsurface Soils	NCR-5 (44-47')	X	X	X	X
Deep Subsurface Soils	NCR-6 (38-40')	X	X	X	X
Deep Subsurface Soils	NCR-7 (44-46')	X	X	X	X
Landfill Perimeter Ground Water	NCR-10M-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-10M-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-11D-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-11D-IDUP	X	X	X	X
Landfill Perimeter Ground Water	NCR-11D-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-11DSP	X	X	X	X
Landfill Perimeter Ground Water	NCR-11M-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-11M-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-13S-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-13S-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-1M-I	X	X	X	X
Landfill Perimeter Ground Water	NR-1M-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-2D-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-2D-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-2I-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-2I-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-2M-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-2M-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-2S-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-2S-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-3M-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-3M-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-3S-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-3S-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-4M-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-4M-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-4S-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-4S-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-5D-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-5D-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-5M-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-5M-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-5S-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-5S-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-6D-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-6D-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-6M-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-6M-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-7M-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-7M-IDUP	X	X	X	X

TABLE 2-1 (CONTINUED)

Medium/Area	Sample Number	Volatile Organics	BNAs	Pesticides/ PCBs	Inorganics
Landfill Perimeter Ground Water	NCR-7M-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-7M-IIDUP	X	X	X	X
Landfill Perimeter Ground Water	NCR-8D-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-8D-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-8M-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-8M-II	X	X	X	X
Landfill Perimeter Ground Water	NCR-8M-IIDUP	X	X	X	X
Landfill Perimeter Ground Water	NCR-9M-I	X	X	X	X
Landfill Perimeter Ground Water	NCR-9M-II	X	X	X	X
Northernmost Landfill Cell Ground Water	NCR-12D-I	X	X	X	X
Northernmost Landfill Cell Ground Water	NCR-12D-IDUP	X	X	X	X
Northernmost Landfill Cell Ground Water	NCR-12D-II	X	X	X	X
Northernmost Landfill Cell Ground Water	NCR-12D-IIDUP	X	X	X	X
Northernmost Landfill Cell Ground Water	NCR-12DSP	X	X	X	X
Northernmost Landfill Cell Ground Water	NCR-12DUPSP	X	X	X	X
Drainage Swale Surface Water	SW-1-R	X	X	X	X
Drainage Swale Surface Water	SW-10-R	X	X	X	X
Drainage Swale Surface Water	SW-11-R	X	X	X	X
Drainage Swale Surface Water	SW-11-RDUP	X	X	X	X
Drainage Swale Surface Water	SW-11SP	X	X	X	X
Drainage Swale Surface Water	SW-13	X	X	X	X
Drainage Swale Surface Water	SW-18-R	X	X	X	X
Drainage Swale Surface Water	SW-2-R	X	X	X	X
Drainage Swale Surface Water	SW-4-R	X	X	X	X
Drainage Swale Surface Water	SW-5-R	X	X	X	X
Drainage Swale Surface Water	SW-7-R	X	X	X	X
Drainage Swale Surface Water	SW-8-R	X	X	X	X
Drainage Swale Sediments	SED-1-R	X	X	X	X
Drainage Swale Sediments	SED-10-R	X	X	X	X
Drainage Swale Sediments	SED-11-R	X	X	X	X
Drainage Swale Sediments	SED-11-RDUP	X	X	X	X
Drainage Swale Sediments	SED-12-R	X	X	X	X
Drainage Swale Sediments	SED-12SP	X	X	X	X
Drainage Swale Sediments	SED-13-R	X	X	X	X
Drainage Swale Sediments	SED-14-R	X	X	X	X
Drainage Swale Sediments	SED-15-R	X	X	X	X
Drainage Swale Sediments	SED-16-R	X	X	X	X
Drainage Swale Sediments	SED-17-R	X	X	X	X
Drainage Swale Sediments	SED-18-R	X	X	X	X
Drainage Swale Sediments	SED-2-R	X	X	X	X
Drainage Swale Sediments	SED-3-R	X	X	X	X
Drainage Swale Sediments	SED-4-R	X	X	X	X
Drainage Swale Sediments	SED-4DUPSP	X	X	X	X

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TABLE 2-1 (CONTINUED)

Medium/Area	Sample Number	Volatile Organics	BNAs	Pesticides/ PCBs	Inorganics
Drainage Swale Sediments	SED-4SP	X	X	X	X
Drainage Swale Sediments	SED-5-R	X	X	X	X
Drainage Swale Sediments	SED-6-R	X	X	X	X
Drainage Swale Sediments	SED-7-R	X	X	X	X
Drainage Swale Sediments	SED-7-RDUP	X	X	X	X
Drainage Swale Sediments	SED-8-R	X	X	X	X
Drainage Swale Sediments	SED-9-R	X	X	X	X
Leachate Soils	SEEP-1-R	X	X	X	X
Leachate Soils	SEEP-3-R	X	X	X	X
Leachate Water	SEEP-10-R	X	X	X	X
Leachate Water	SEEP-13-R	X	X	X	X
Leachate Water	SEEP-14-R	X	X	X	X
Leachate Water	SEEP-16-R	X	X	X	X
Leachate Water	SEEP-21-R	X	X	X	X
Leachate Water	SEEP-5-R	X	X	X	X
Leachate Water	SEEP-5-RDUP	X	X	X	X
Leachate Water	SEEP-5SP	X	X	X	X
Leachate Water	SEEP-7-R	X	X	X	X

Notes: (1) All water samples analyzed were unfiltered.
(2) Deep subsurface soils were not evaluated further in the risk assessment.

Prior to analysis, site data were reviewed for the following:

- Validation qualifiers on concentration values,
- Sample duplicates,
- Sample dilutions, and
- Sample re-extracts.

Validation qualifiers were treated according to EPA guidance (EPA, 1989a). Rejected samples ("R" qualifiers) were not included in the data base for the risk assessment. Non-detect results ("U" qualifier) were included only if other results for a given chemical in a particular medium/area indicated the chemical was present. In these instances, half the reported sample quantitation limit was used. This procedure is described further below. Estimated results, usually indicated by a "J" qualifier, were included in summary statistics (see Appendix A).

Duplicates of the following nineteen samples were included with the site sampling data:

HA-9
NCR-11B (0-3.2')
NCR-6 (2.0-6.0')
NCR-10 (2-4')
NCR-3M (24-26')
NCR-12 (22-26')
NCR-12 (44-46')
NCR-7M-I
NCR-7M-II
NCR-8M-II
NCR-11D-I
NCR-12D-I
NCR-12D-II
NCR-12DSP
SW-11-R
SED-4SP
SED-7-R
SED-11-R
SEEP-5-R

In most cases, results of the duplicate sample and the original sample were averaged. The resulting value was the arithmetic mean of positive results or the arithmetic mean of the reported detection limits if both samples showed non-detects. Conservatively, if one sample showed a positive result and the other a negative result, the positive result was used.

The site data included several samples that required dilution during analysis. Where initial results indicated the instrument calibration range was exceeded for a given analyte ("E" qualifier), the dilution result was considered more appropriate. In addition, if a chemical was detected in the dilution but not the initial sample, the dilution value was used. Otherwise, the original result was used.

The site data also included some samples that required re-extraction during analysis. Based on notations in the data package, the data from the re-extracts were considered more appropriate than the initial result. In these cases, the data from the initial analyses were deleted and replaced with the results of the re-extraction.

Summary statistics for all chemicals detected in each medium/area evaluated in the public health risk assessment are presented in Appendix A. Appendix A tables list frequency of detection, number of samples analyzed (excluding rejected samples), the lowest and highest detected concentrations, the mean concentration, the upper 95-percent confidence limit of the mean, and the lowest and highest detection limits for non-detects. (Appendix B contains summary statistics for somewhat different data groupings for the ecological risk assessment). All data were analyzed using SAS™, a widely-used statistical software package (SAS Institute Inc., 1988).

As agreed with EPA Region II and in accordance with current Superfund risk assessment guidance (EPA, 1989a), risk calculations for the "reasonable maximum exposure" scenario were based on either the upper 95-percent confidence limit (95% UCL) of the mean concentration or the maximum concentration; the lower of these

two values was used in risk calculations. The 95% UCL was obtained using the method developed by Land (1975) as described by Gilbert (1987). This method is preferred for log normally distributed data, which are typical of environmental sampling programs, and is one of the methods currently recommended by EPA.

The equation used to calculate the 95% UCL concentration is:

$$95\%\ UCL = \exp\left[\overline{Y} + 0.5(s^2) + (s)(H)/(n-1)^{1/2}\right]$$

where:

exp = inverse natural log function;

$\overline{Y}$ = arithmetic mean of log-transformed data;

s^2 = variance of the mean;

s = standard deviation;

H = H statistic (Land, 1975); and

n = sample size (number of samples analyzed).

The H statistic for the above equation is dependant on sample size n and the standard deviation s. Land (1975) provides tables of H values for representative values of n and s. As recommended by Land, values of H not appearing in the tables were derived by cubic (four-point Lagrangian) interpolation (Hornbeck, 1975).

2.2 Selection of Contaminants of Concern

2.2.1 Background

This section describes the methodology used to select contaminants of concern (COCs) for the NCR site for all medium evaluated. The selected COCs are a subset of the total list of contaminants detected and represent those contaminants likely to contribute most to the overall human health risk at the site. COCs were chosen for surface soils,

subsurface soils, landfill perimeter ground water, northernmost landfill cell ground water, drainage swale surface water, drainage swale sediments, leachate soils and leachate water.

2.2.2 Methodology

A wide variety of hazardous substance list chemicals were detected at the NCR site in all media evaluated in the risk assessment including over 20 VOCs, 37 BNAs, 19 pesticides and 24 inorganic chemicals. The concentration and frequency of detection for all these chemicals are summarized in Appendix A. Based on the heterogeneous nature of contamination expected due to the site's history, contaminants with suspected natural sources could not be eliminated. In addition, although analytes such as human nutrients (calcium, magnesium, potassium and sodium) are typically eliminated as COCs, site-specific background concentrations of inorganics were unknown and could not be used to justify the selection or elimination of other inorganic COCs. A risk assessment based on this lengthy list of potential site-related chemicals, however, would contribute little to the accuracy of the risk estimate and would not facilitate the identification of the significant contaminants. As a result, a quantitative method termed "concentration-toxicity screening" was employed to select COCs.

The concentration-toxicity screening procedure (EPA, 1989a) was utilized to identify the most "important" chemicals, i.e., chemicals contributing 99 percent of the risk for each medium at the site, based on concentration and toxicity. The screening procedure involved the following four steps: 1) calculation of individual chemical risk factors, 2) calculation of the sum of the risk factors for each medium, 3) calculation of the ratio of the chemical-specific risk factor to the total risk factor for each medium, and 4) elimination of all chemicals with ratios of less than 0.01 for each medium. Separate ratios were calculated for carcinogenic and noncarcinogenic effects. In step 1), a risk factor (Ri_j) for each chemical in each medium was obtained from the product

of the concentration of chemical i in medium j (C_{ij}) and the toxicity value for chemical i in medium j (T_{ij}) (i.e., either the slope factor or 1/RfD) as shown below:

$$R_{ij} = (C_{ij})(T_{ij}).$$

Available toxicity values used in step one (slope factors, RfDs) for chemicals detected at the NCR site are summarized in Appendix C. Throughout the screening procedure, only oral toxicity values were used because ingestion is the only exposure route fully evaluated for each medium.

In step 2), individual chemical risk factors were summed to obtain a total risk factor for each medium (R_j) as shown below:

$$R_j = R1_j + R2_j + \ldots + R_{ij}.$$

The ratio of the risk factor for each chemical (R_{ij}) to the total risk factor (R_j) was calculated as R_{ij}/R_j in Step 3). This ratio approximates the relative risk for each chemical in medium j.

In step 4), all chemicals with R_{ij}/R_j ratios of less than 0.01 in all media detected were eliminated as COCs. Conversely, if the ratio based on either noncarcinogenic or carcinogenic effects for a specific chemical was equal to or exceeded 0.01 in any medium, that chemical was retained for evaluation in the risk assessment for all media in which detected. The results of the concentration-toxicity screen performed for the NCR site are summarized in Appendix D.

Several contaminants detected at the site lack appropriate toxicity values for use in quantitative risk assessment and could not be incorporated into the concentration-toxicity screening. Of these chemicals, phenanthrene, delta-BHC, cobalt, and lead

were retained for further evaluation. 2-Methylnaphthalene, benzo(g,h,i)perylene, endrin aldehyde, endrin ketone, endosulfan sulfate, and isopropylbenzene were not retained due to their infrequent detection in only one or two site media.

Chromium was evaluated in the concentration-toxicity screening using a weighted-average value of the trivalent chromium and hexavalent chromium RfDs comprised of 7 parts tri to 1 part hex (EPA, 1992f). Although chromium was detected in most soil media at maximum concentrations of approximately 31,000 µg/kg and at a maximum concentration in leachate water of 118 µg/l, it was not shown to be a contaminant of concern in any site medium.

The following carcinogenic chemicals were shown to contribute less than 1% of the risk in each medium detected: chloroform, tetrachloroethylene, n-nitrosodiphenylamine, pentachlorophenol, alpha-BHC, gamma-BHC, alpha-chlordane, gamma-chlordane, 4,4-DDD, 4,4-DDE, 4,4-DDT, dieldrin, and heptachlor epoxide. However, based on their carcinogenic weight of evidence classification, occurrence in media where exposure is most likely to occur (sediments and soils), frequency of detection, and slightly elevated concentrations, the following chemicals were retained as COCs: 4,4-DDE, 4,4-DDT, dieldrin, and heptachlor epoxide. The toxicity screen also excluded several carcinogenic PAHs (cPAHs) as COCs as a result of the application of toxic equivalency factors (TEFs) (See Section 5.2.2).

Table 2-2 lists all COCs selected and all media in which the COCs were detected.

In addition to the Target Compound List (TCL) of organic compounds detected at the NCR site, other organic compounds may have been detected that are not on the TCL. These additional compounds are referred to as tentatively identified compounds (TICs). Their assigned identity is highly uncertain. Similarly, the reported concentrations are only estimations, are highly questionable, and may be orders of magnitude higher or lower than the actual concentrations (EPA, 1989a).

TABLE 2-2. NIAGARA COUNTY REFUSE SITE: CONTAMINANTS OF CONCERN								
	Surface Soil	**Subsurface Soils**	**Landfill Perimeter Ground Water**	**NCR 12D Ground Water**	**Drainage Swale Surface Water**	**Drainage Swale Sediments**	**Leachate Soil**	**Leachate Water**
Volatiles								
Acetone	X	X	X	X		X		X
Benzene			X	X		X		X
2-Butanone				X				X
1,4-Dichlorobenzene				X			X	
Methylene Chloride	X	X	X	X		X	X	X
Styrene	X			X				
Trichloroethylene	X	X		X				
1,2,4-Trimethylbenzene				X				
Vinyl Chloride	X	X						
BNAs								
Benzo(a)anthracene						X	X	
Benzo(a)pyrene						X		
Bis(2-ethylhexyl)phthalate	X	X	X	X	X	X	X	X
4-Chloroaniline								X
2,4-Dimethylphenol				X	X			X
2,6-Dinitrotoluene								X
2-Methylphenol				X				X
4-Methylphenol		X		X			X	X
Naphthalene		X		X			X	X
Phenanthrene		X				X	X	X
Phenol			X	X	X			X
Pesticides								
Aldrin			X			X	X	X
Delta-BHC	X		X		X	X	X	X

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TABLE 2-2. (CONTINUED)

	Surface Soil	Subsurface Soils	Landfill Perimeter Ground Water	NCR 12D Ground Water	Drainage Swale Surface Water	Drainage Swale Sediments	Leachate Soil	Leachate Water
4,4'-DDE			X			X	X	
4,4'-DDT			X		X	X		X
Dieldrin			X			X	X	
Heptachlor			X	X				X
Heptachlor Epoxide		X	X		X	X		
Inorganics								
Aluminum	X	X	X	X	X	X	X	X
Antimony		X	X	X		X		
Arsenic	X	X	X	X	X	X	X	X
Barium	X	X	X	X	X	X	X	X
Beryllium	X	X	X	X	X	X	X	X
Cadmium			X		X	X	X	X
Cobalt	X	X	X	X	X	X	X	X
Copper	X	X	X	X	X	X	X	X
Cyanide					X			
Iron	X	X	X	X	X	X	X	X
Lead	X	X	X		X	X	X	X
Manganese	X	X	X	X	X	X	X	X
Mercury		X				X	X	X
Nickel	X	X	X	X	X	X	X	X
Silver			X	X				
Thallium		X			X			
Vanadium	X	X	X	X	X	X	X	X
Zinc	X	X	X	X	X	X	X	X

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TICs were not selected as COCs at the NCR site for the following reasons:

- a large degree of uncertainty is associated with both the identity and estimated concentrations of TICs;
- identification of TICs was typically insufficient to gather appropriate toxicity data because specific identities were rarely assigned, only general chemical class (e.g., chlorinated alkanes); and
- comparatively fewer TICs than TCL compounds were present.

3.0 CONTAMINANT FATE AND TRANSPORT

3.1 Introduction

This section describes the fate and transport of chemical contaminants detected at the NCR site and selected as COCs. The discussion integrates the geology, hydrology, and nature and extent of contamination (summarized in Section 1) with physical and chemical characteristics of the contaminants detected. The evaluation presented here is qualitative and focuses on the contaminants that are of primary concern from a human health risk perspective. Groups of chemicals (e.g., VOCs) are evaluated together when physical and chemical characteristics are similar. The discussion provides a separate analysis for the following chemical classes: VOCs, BNAs, pesticides, and inorganics. Table 3-1 presents a summary of the physical and chemical properties of the organic COCs based on values obtained from the literature.

3.2 Potential Routes of Migration

Contamination at the site will be transported from a potential source area to uncontaminated areas by the movement of contaminated media via natural processes. In the case of the NCR site, the primary source areas are waste deposited in landfill cells and contaminated subsurface soils. In general, contaminant movement will occur as ground water, surface water runoff, and air move away from source areas. Site-specific features will influence this movement.

The features which influence the transport and fate of the detected contaminants are medium-specific. Ground water transport will depend on the nature of the geologic materials as well as the direction and velocity of ground water flow. The locations of ground water recharge and discharge will also influence the anticipated exposure routes. In the case of surface water, the site features of most concern are surface

TABLE 3-1. PHYSICAL AND CHEMICAL PROPERTIES OF CONTAMINANTS OF CONCERN

Detected Compounds	Chem. Class	Water Solubility (mg/L)	K_{oc} Organic Carbon Partition Coeff. (ml/g)	Log K_{ow} Octanol Water Partition Coeff.	Fish Bioconc. Factor (L/Kg)	Vapor Pressure (mm Hg)	Henry's Law Constant ($atm*m^3/mol$)	Soil Half-Life (days)	Surface Water Half-Life (days)	Ref.
Acetone	VOC	1000000	2.2	-0.24		2.70e+02	2.06e-05		1.9	A
Aldrin	Pest/Herb	0.18	96000	5.30	28	6.00e-06	1.60e-05			H
Benzene	VOC	1750	83	2.12	5.2	9.52e+01	5.59e-03		1-6	A
Benz(a)anthracene	PAH	0.0057	1e+06	5.6		2.20e-08	1.16e-06		0.1-5	A
Benzo(a)pyrene	PAH	0.0012	6e+06	6.06		5.60e-09	1.55e-06	420-480	0.4	A
Bis(2-ethylhexyl)phthalate	BN	0.4		8.73		2.00e-07	2.57e-07			E
BHC (delta-)	Pest/Herb	31.4	6600	4.10	130	1.70e-05	2.07e-07			H
Butanone (2-)	VOC	268000	4.5	0.26	0	7.75e+01	2.74e-05		10	A
Chloroaniline (4-)	BN					1.50e-02				G
DDE (4,4'-)	Pest/Herb	0.04	4e+06	7	51000	6.50e-06	6.80e-05			H
DDT (4,4'-)	Pest/Herb	0.005	2e+05	6.19	54000	5.50e-06	5.13e-04	1000 - 5500	56 - 110	H
Dichlorobenzene (1,4-)	VOC	79	1700	3.60	56	1.18e+00	2.89e-03			H
Dieldrin	Pest/Herb	0.195	1700	3.5	4760	1.78e-07	4.58e-07			A
Dimethylphenol (2,4-)	A	4200	222	2.42		6.21e-02	2.38e-06			H
Dinitrotoluene (2,6-)	BN	240	45	2	3.8	5.10e-03	5.09e-06			H
Heptachlor	Pest/Herb	0.18	12000	4.40	15700	3.00e-04	8.19e-04		0.96	H
Heptachlor epoxide	Pest/Herb	0.35	220	2.70	14400	3.00e-04	4.39e-04			H
Methylene chloride	VOC	20000	8.8	1.3	5	3.62e+02	2.03e-03		1.2-5.8	A
Methylphenol (2-)**	A	25000	500	1.95	0	2.43e-01	1.50e-06			H

TABLE 3-1. (CONTINUED)

Detected Compounds	Chem. Class	Water Solubility (mg/L)	K_{oc} Organic Carbon Partition Coeff. (ml/g)	Log K_{ow} Octanol Water Partition Coeff.	Fish Bioconc. Factor (L/Kg)	Vapor Pressure (mm Hg)	Henry's Law Constant ($atm*m^3/mol$)	Soil Half-Life (days)	Surface Water Half-Life (days)	Ref.
Methylphenol (4-)**	A	25000	500	1.93	0	1.14e-01	1.10e-06			H
Naphthalene	PAH	30.6		3.35		2.30e-01	4.48e-04			C
Phenanthrene	PAH	1	14000	4.46	2630	6.80e-04	1.59e-04		0.38-2.00	A
Phenol	A	93000	14.2	1.46	1.4	3.41e-01	4.54e-07		0.62 - 9	H
Styrene	VOC	300				4.50e+00	2.05e-03			H
Trichloroethylene	VOC	1100	126	2.38	10.6	5.79e+01	9.10e-03		1.0-90.0	A
Trimethylbenzene (1,2,4-)	VOC	57.6				2.03e+00	5.57e-03			H
Vinyl chloride	VOC	2670	57	1.38	1.17	2.66e+03	8.19e-02		1.0-5.0	A

LEGEND:

VOC	- Volatile Organic Compound
A	- Acid Extractable Organic Compound
BN	- Base/Neutral Extractable Organic Compound
PAH	- Polycyclic Aromatic Hydrocarbon
PCB	- Polychlorinated Biphenyl
Pest/Heb	- Pesticide/Herbicide
*	- Estimated value
**	- Water solubility + Koc are reported for 2-methylphenol
***	- Properties are reported for the alpha-isomer.

REFERENCES:

A - EPA Superfund Public Health Evaluation Manual, October, 1986.
B - EPA Water Quality Assessment: A Screening Procedure for Toxic and Conventional Pollutants in Surface and Ground Water Part I, September 1985.
C - Miller, M.M. and S.P. Wasik, 1985. Environ. Sci. Technol. 19, 552-529.
D - Mackay, D. and W.Y. Shiu, 1981. J. Phys. Chem. Ref. Data, 19(4).
E - EPA Water-Related Environmental Fate of 129 Priority Pollutants, December, 1979.
F - EPA Treatability Manual, Volume I: Treatability Data, September, 1981.
G - Handbook of Environmental Data on Organic Chemicals, Verschueren, 1977.
H - EPA Basics of Pump-and-Test Ground-Water Remediation Technology, March, 1990.

drainage patterns, topography, and surface cover. For emissions of contaminants from the soil into air, ground cover plays the most significant role at the site.

3.2.1 Ground Water Transport

The hydrogeologic environment at the site is complex. Two water bearing units (aquifers) underlie the NCR site, an overburden unit and a bedrock unit. The 1991 RI report states that silty clay and till units separate the two aquifers and act as semi-confining layers (CRA, 1991a). The overburden aquifer appears to be a seasonal manifestation that, when present, directly contacts solid waste material in some landfill cells (see figure 5.4 of RI Report). The direction of ground water flow was not determined in the overburden aquifer which lies perched atop the semi-confining layers. Flow patterns are expected to be controlled by the surface topography of the semi-confining layers. Ground water (and possibly dissolved contaminants) may flow downward from the overburden aquifer to the bedrock aquifer since a vertical flow component does exist. However, vertical ground water flow was predicted to be minimal given the massive thickness (20 to 40 feet) and low hydraulic conductivities of the semi-confining layers (CRA, 1991a). Flow within the bedrock aquifer, also predicted to be minimal, is predominantly in a horizontal direction through a fracture network. The Niagara River may recharge this aquifer.

Contamination has been documented in both aquifers. Dissolved contaminants may be leached from the solid waste to the overburden aquifer. Heavier-than-water contaminants may accumulate and migrate along the upper surface of the semi-confining layers or they may sink further through the semi-confining layer to the bedrock aquifer. Contaminants that reach the bedrock aquifer will be transported in the direction of ground water flow. The direction of ground water flow is generally to the north toward Lake Ontario (CRA, 1991a), but may also be to the south toward the Niagara River based on seasonal fluctuations, etc. (TRC, 1992).

3.2.2 Surface Transport

The surface topography of the site is dominated by man-made features that control drainage. The six landfill cells are expressed as distinct mounded areas (typically 20 feet high) that are separated by drainage swales and access roads. A perimeter drainage swale also surrounds the entire landfill. Surface water runoff from precipitation is expected to be directed away from the landfill mounds into the drainage swales. Runoff from most areas is directed to an underground culvert that eventually discharges into the Niagara River. Runoff from the northern-most areas is directed to a second culvert which discharges to a creek in the northern wetlands. Since many of the swales are vegetated or clogged with heavy accumulations of silt, surficial transport of contaminants will be limited.

Contaminants from areas where the landfill cover is eroded, exposing solid waste or contaminated materials, may migrate and be deposited in the drainage swales. Contaminants may also seep out of the landfill toes along the drainage swales. Field personnel noted both exposed solid waste and numerous leachate seeps during RI activities (CRA, 1991a).

3.2.3 Contaminant Releases to Air

Most areas of the site are vegetated with grasses, although bare soil does exist on parts of the landfill cap. The low soil concentrations of contaminants and nature of the ground cover are likely to preclude organic vapor and dust emissions from soils.

3.3 Contaminant Persistence and Migration

Organic and inorganic contaminants were identified in ground water, surface and subsurface soils, surface water from leachate seeps and drainage swales, and sediments

from leachate seeps and drainage swales. Intermedia transfer of contaminants may occur by a variety of processes. The general processes associated with each medium and contaminant type are discussed below.

3.3.1 Surface Soils

Volatile Organics

Five VOCs were selected as COCs in surface soils; these include: acetone, methylene chloride, styrene, TCE, and vinyl chloride. These compounds have high vapor pressures which favor volatilization. However, it is unlikely that significant airborne concentrations will result from the typically low detected levels and infrequent occurrences of these VOCs. With the exception of vinyl chloride, these moderately to highly soluble compounds are unlikely to be transported to ground water given their low surface concentrations. Vinyl chloride concentrations reach 240 ug/kg, indicating that this chemical may be transported to the subsurface. Little attenuation is expected since this compound has a very low propensity for sorption to soils (Koc<1,000 ml/g).

Base-Neutral/Acid Extractable Organics

Bis(2-ethylhexyl)phthalate was the only detected BNA in surface soils. It is unlikely to migrate to the subsurface given its low water solubility, high sorption propensity, and limited environmental concentrations (i.e., near detection limit). Since this contaminant has a tendency to bind strongly to soils, the most probable fate of this contaminant is overland transport with eroded soil particulates during storm and meltwater events. Contaminated soils will be deposited in the drainage swales and transported downstream to the Niagara River or Black Creek.

Pesticides/Herbicides

The only pesticide detected, delta-BHC, exhibits transport properties that are similar to bis(2-ethylhexyl)phthalate, except that it has an even greater propensity for adsorbing to soils. It too will be deposited in the drainage swales, and perhaps eventually transported to the Niagara River or the creek in the northern wetlands area, depending on the force of the surface runoff.

Inorganics

Inorganics in onsite surface soil will be transported in much the same way as organic contaminants. The inorganics can be leached by rainfall infiltrating through surface soils and into the ground water. Ion exchange and desorption are the two primary mechanisms which can result in the movement of inorganics from soil to ground water. Inorganics-contaminated soils can also be eroded by surface processes and subsequently deposited into the drainage swales. It is unlikely that inorganics-contaminated soil may act as a source for airborne emissions of particulates because a majority of the site is covered by grasses.

3.3.2 Subsurface Soils

Volatile Organics

The contamination profile of VOCs in subsurface soils was similar to that observed in surface soils; concentrations were also comparable. The four VOCs selected as COCs in subsurface soils include acetone, methylene chloride, TCE, and vinyl chloride. These highly water soluble compounds are readily leached from soils and may be transported to ground water. It is doubtful that the low soil concentrations will result

in significant ground water concentrations. The low Koc values (<1,000 mL/g) suggest that these VOCs will migrate slowly through the semi-confining layer with little sorption to the geologic material.

Base-Neutral/Acid Extractable Organics

Only very low levels (below or near detection limits) of BNAs were detected. Their low water solubilities, high sorption propensities, and limited environmental concentrations will limit their downward transport. Only 4-methylphenol is likely to be leached from soils and transported to ground water. However, resulting ground water concentrations would not be significant given the low soil concentrations. The semi-confining layer with typically high sorptive capacities will retard the downward migration of all detected BNAs but 4-methylphenol.

Pesticides/Herbicides

Heptachlor epoxide has a low water solubility and low sorptive capacity and is likely to remain near its point of deposition. The trace concentrations and presence of the semi-confining layer significantly limit any downward migration.

Inorganics

Subsurface soils contaminated by inorganics can act as a source of contamination to ground water through leaching. The extent of this process cannot be evaluated at this time because the pH and eH (oxidation/reduction potential) of the soils and ground water are unknown. These measurements provide information on the acidity and oxidation state of the different media and can be used to predict the equilibrium state of the inorganics in the soil and ground water. Additional processes that will affect

the movement of inorganics through soils include ion exchange and desorption. These processes also cannot be predicted because the CEC (cation-exchange capacity) of soils is unknown.

3.3.3 Ground Water

Similar ground water contamination was reported in both the overburden and bedrock aquifers. Since these two aquifer are hydraulically connected, at least minimally, they are considered to be one unit.

Volatile Organics

Volatile contaminants were identified in ground water from the interior of the northern landfill cell at concentrations which were somewhat greater than those in perimeter monitoring wells. Fewer volatile contaminants, at low concentrations, were reported in the perimeter monitoring wells. Volatile ground water contaminants detected in the northern landfill cell included, acetone, 2-butanone, 1,4-dichlorobenzene, methylene chloride, styrene, trichloroethylene, 1,2,3-trimethylbenzene, and 1,3,5-trimethylbenzene. The moderate to high water solubilities of these VOCs indicate they will be transported in the direction of ground water flow. The presence of these compounds in the bedrock aquifer underlying the northern landfill cell suggests that VOCs have migrated from the overburden aquifer through the semi-confining layer. The most likely cause of this transport is the natural downward hydraulic gradient present in the site's aquifer. Concentrations are predicted to significantly diminish during horizontal transport from the site as a result of dispersion and dilution within the water column and by adsorption to geologic materials.

Base-Neutral/Acid Extractable Organics

Of the organic contaminants detected in ground water, phenolic compounds were identified at concentrations nearly an order-of-magnitude greater than other organics. With high water solubilities, 2-methylphenol, 4-methylphenol, and 2,4-dimethylphenol can be readily transported in the direction of ground water flow. Little attenuation will occur from adsorption given their low Koc and log Kow values as listed in Table 3-1. Their presence in the bedrock aquifer is evidence of their low sorptive properties since these phenolic compounds migrated through the semi-confining layer. Concentrations of these contaminants will most likely diminish as a result of dispersion and dilution during horizontal transport from the site.

Pesticides/Herbicides

Heptachlor, delta-BHC, and 4,4'-DDE were identified in both the overburden and bedrock aquifers. They were predominantly detected in monitoring wells along the landfill perimeter, with only trace concentrations identified in ground water from the landfill interior. These compounds typically have limited mobilities because of their low water solubilities, and high propensities for adsorbing to soils. The semi-confining layer, with high binding capacities, is expected to restrict the downward migration of these compounds so presence in the bedrock aquifer is unexpected.

Inorganics

Metals migration in ground water is a complex process and is dependent upon the phase in which the metals exists (e.g., dissolved species or suspended particulate). At the NCR site, aluminum, beryllium, cobalt, copper, iron, lead, vanadium, and zinc primarily occur in solid phase as indicated by higher concentrations in unfiltered samples than filtered (CRA, 1991a). These metals are expected to be less mobile than

those in the dissolved phase since their movement as suspended particulates will be impeded by geologic material. Dissolved metals (arsenic, barium, cadmium, and nickel) will be transported in the direction of ground water flow although concentrations will be lessened by dispersion, dilution, and adsorption to geologic materials.

3.3.4 Drainage Swale Sediments

Volatile Organics

Though few VOCs were detected in the drainage swale sediments, some were detected in greater than half of the samples analyzed. Acetone, benzene, and methylene chloride have high vapor pressures which favor volatilization. The fact that these drainage swales contain water seasonally or after storm events increases the likelihood of volatilization since they are usually exposed to the ambient air. However, it is unlikely that significant airborne concentrations will result from the low levels detected. Contaminants that do not volatilize may be transported to the subsurface given the high water solubilities. Little attenuation is expected since these compounds have low propensities for sorption to soils (Koc<1,000 mL/g).

Base-Neutral/Acid Extractable Organics

PAHs were the most abundant BNAs detected in the drainage swale sediments. Fluoranthene, benzo(a)pyrene, and chrysene are a few of the detected PAHs. These compounds are unlikely to migrate to the subsurface due to their low water solubilities, high sorption propensities, and trace environmental concentrations (i.e., below detection limits). The most probable fate of these contaminants is overland transport as eroded soil particulates further downstream in the drainage swales, with possible subsequent deposition into the Niagara River or the northern wetlands area.

Pesticides/Herbicides

The detected pesticides retained as COCs include aldrin, delta-BHC, dieldrin, 4,4'-DDE, 4,4-DDT, and heptachlor epoxide. All have limited water solubilities, and are moderately to highly sorbed to soils. These compounds will most likely be transported downstream with eroded materials, then deposited in downstream locations within the drainage swale, Niagara River, and/or the northern wetlands area.

Inorganics

Inorganics will be transported much the same way as BNAs and pesticides with deposition in downstream locations.

3.3.5 Drainage Swale Surface Water

Numerous organic contaminants were detected in drainage swale surface water samples. However, concentrations were typically near the detection limits. Bis(2-ethylhexyl)phthalate (BEHP) was notably different from this trend since it was detected at concentrations 100 times greater than the detection limit. With detected concentrations exceeding its water solubility value, this compound is most likely present as a suspended particulate or colloid rather than a dissolved contaminant. These particulates will most likely be deposited at location further downstream (i.e., drainage swale, Niagara River, and/or northern wetlands).

The RI did not distinguish between suspended and dissolved inorganic species in surface water. Those species present as suspended particulates will be subject to transport processes that are similar to BEHP. Dissolved inorganic species will be deposited to the surface waters of the Niagara River and/or the northern wetlands area.

Limited concentrations may be leached from soils to ground water. However, the pH and eH of the sediments are unknown so the possible extent of leaching cannot be evaluated.

3.3.6 Leachate Soil

Two groups of compounds represent the dominant contaminants detected in the soils underlying the leachate seeps. These were PAHs (e.g., naphthalene, chrysene, and phenanthrene) and pesticides/herbicides (e.g., 4,4'-DDE, dieldrin, and delta-BHC). These contaminants exhibit similar physical and chemical properties and are therefore expected to be transported by similar processes. All have limited water solubilities, and are moderately to highly sorbed to soils. These compounds, with low water solubilities and high sorptive tendencies, will most likely remain bound to soils, and transported only as eroded materials. They will likely be deposited in the drainage swale, Niagara River, and/or the northern wetlands area.

The soils associated with the leachate seeps can act as a source of inorganic contamination to ground water through leaching or to downstream drainage swale sediments by erosion. As described previously for subsurface soils the extent of these processes cannot be evaluated at this time because the pH and eH of these soils and leachate are unknown. These measurements, are required in order to predict the equilibrium state of the inorganics in the soil and leachate water. Additional processes which will affect the movement of inorganics through soils include ion exchange and desorption. These processes also cannot be predicted because the CEC of soils is unknown.

3.3.7 Leachate Water

Volatile Organics

Several VOCs were detected at elevated concentrations (>1,000 ug/L). Benzene and methylene chloride are most likely to volatilize from these surface seeps (Henry's Law Constants>10^{-3}); whereas, acetone and 2-butanone have tendencies to remain in solution. Since all of these VOCs are moderately to highly soluble in water with little sorptive propensities, those that do not completely volatilize from the seeps will be transported to the drainage swales. These drainage swales only contain surface water seasonally, so once the water evaporates, each of these VOCs is likely to volatilize. Portions may be transported to the subsurface as the water infiltrates the soils. Therefore, it is unlikely that these VOCs will be transported from the drainage swales to ground water.

Other Organic Contaminants

Though numerous organic compounds were detected in the leachate water, most were present at or below the detection limits. This is most likely a result of the limited water solubilities of the BNA contaminants, including pesticides and herbicides. These compounds are most likely present as suspended particulates and colloids. Erosion is predicted to be the most likely transport mechanism. Transport of the low concentrations being discharged by the leachate seeps will be restricted to the immediate seep vicinity.

Inorganics

The RI did not distinguish between suspended and dissolved inorganic species in leachate water. Assumed transport processes are similar to those identified for leachate soils.

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4.0 EXPOSURE ASSESSMENT

4.1 Introduction

This section evaluates the likelihood, magnitude, and frequency of exposure to the contaminants of concern at the Niagara County Refuse site. In the exposure assessment, pathways and routes by which receptors may contact contaminants are identified. The specific steps involved in the exposure assessment include the following:

- Characterization of Exposure Setting (Section 4.2)
  - description of the physical setting
  - identification of potentially exposed populations
- Identification of Exposure Pathways (Section 4.3)
  - identification of media of concern
  - identification of actual and potential exposure routes
- Development of Exposure Scenarios (Section 4.4)
  - present and future scenarios
  - exposure parameters
- Quantification of Exposure (Section 4.5)
  - estimation of exposure point concentrations
  - estimation of exposure doses

The physical characteristics of the site were examined in order to adequately assess the pathways by which human receptors may become exposed to site contaminants. Exposure scenarios were then developed with consideration of demographics, land use, and human behavior patterns. Estimates of exposure doses were calculated for each actual and potential exposure pathway and receptor population, considering both present and future use of the site. In accordance with current EPA guidance, the

reasonable maximum exposure (RME) was assessed. Values for intake variables (e.g., consumption rates) were selected so that the combination of all values used to calculate exposure doses will result in conservative but reasonable estimates. As such, not all intake variables represent maximum values. Compounding maximum values for all inputs would result in unrealistically high exposure estimates.

4.2 Characterization of Exposure Setting

The physical characteristics of the site and characteristics of the human population on and near the site were evaluated to determine which parameters might influence exposure to site contaminants and to help identify exposure pathways. The physical setting of the NCR site is described in Section 1.0. This section focuses on actual and potential receptors of site contaminants.

Demographics and land use were evaluated in assessing present and potential future populations which live, work, or otherwise spend time at or in the area of the NCR site. The purpose of this analysis was to assess the likelihood of various groups, including sensitive subpopulations, of becoming exposed to site contaminants.

Receptors under Present Land Use: The Niagara County Disposal District operated the Niagara County Refuse Landfill until 1976 at which time the landfill was officially closed. The site is located in an industrial zoning district; however, adjacent land is zoned for residential, commercial, and industrial uses. The area is provided with a municipal water supply.

Residences, including one with a large vegetable garden, are located adjacent to the eastern boundary of the site. Access to the site is currently not restricted and trespassers, along with signs of previous trespassing activity (trails, vandalism), have

been observed on-site (CRA, 1991a; TRC, 1991b). The site is bounded to the north by wooded wetlands and a clay-mining operation; to the west by active farmland for the production of wheat and corn; and to the south by railroad tracks, River Road, and a park near the Niagara River. The Niagara River is used for recreational fishing, swimming, and boating (TRC, 1991b). The present use of the site and adjacent land in addition to the existence of a municipal water supply indicate that trespassers are the most probable receptors to site contaminants under current site conditions.

Receptors Under Future Land Use: Continued residential use of areas directly adjacent to the NCR site seems likely; however, future residential use of the site itself is unlikely to occur because (1) the area is zoned industrial and, as a result, is unlikely to be rezoned residential and (2) the site's prior use as a landfill will most likely preclude its consideration for residential development. Trespassers who live adjacent to the site, excavation workers engaged in utility or landfill maintenance, and residents adjacent to the site who elect to install a private drinking water well rather than use the municipal water supply are possible receptors under future site conditions.

4.3 Identification of Exposure Pathways

The purpose of this step is to identify complete exposure pathways to be evaluated in the risk assessment. To be complete, a pathway must consist of the following four elements:

- a source and mechanism of chemical release into the environment;
- a transport medium by which the released chemical may reach a receptor (e.g., ground water);

- a point of potential contact of the human receptor with the contaminated medium (e.g., individual accesses the site and contacts the contaminated medium); and
- an exposure route (e.g., ingestion, dermal contact).

The sources and mechanisms of chemical release are discussed in Sections 1.0, 2.0, and 3.0 of this report. Transport mediums, points of potential contact, and exposure routes are discussed in the following sections.

4.3.1 Media and Exposure Routes of Concern

Potential exposures to site-related contaminants of concern were examined for the following media:

- surface soils,
- subsurface soils,
- ground water,
- sediments,
- surface water,
- leachate soils,
- leachate water,
- air, and
- home-grown produce.

The discussion that follows provides a rationale for the inclusion or exclusion of different environmental media in the risk assessment. The exposure routes relevant to each medium are also summarized.

Surface and Subsurface Soils

Surface and subsurface soil exposures are assessed separately in the risk assessment. Surface soils are evaluated because trespassing has been observed on the site (TRC, 1991b) and these soils are contaminated. While the landfill was reported covered with a "clean" cap (CRA, 1991a), organics and toxic inorganics were detected in surface soil borings at levels that may be greater than background. Surface soils included all samples collected between 0 feet and 3 feet. Trespasser exposure incidental ingestion of surface soils sampled on the site is evaluated.

Exposure via inhalation of volatilized chemicals or contaminated particulates is not evaluated because contaminant concentrations in surface soils are relatively low and the frequency and duration of exposure is expected to be minimal. VOCs and BNAs were detected infrequently in surface soils. Most compounds were detected in one or two (of 12) samples. Upper confidence limit concentrations for all VOCs were less than 100 µg/kg; mean concentrations were less than 10 µg/kg. The upper confidence limit concentration for BEHP (a BNA) was less than two times EPA's Contract Required Quantitation Limit for BEHP. While no site-specific background concentrations were available for soils, the inorganics concentrations all appear reasonable for background levels in the Northeast. For these reasons, it is believed that significant inhalation exposures to site contaminants will not occur.

Subsurface soils are evaluated because future excavation of landfill subsurface soils may occur during landfill maintenance (fence installation, well/vent installation) or during maintenance of the water line utility which runs near the south, west, and north boundaries of the site. The exposure assessment evaluates incidental ingestion of subsurface soils by future excavation workers. Inhalation of volatilized contaminants and/or contaminated particulates is not evaluated because contaminant concentrations

are low and the magnitude of exposure is expected to be less significant than through other subsurface soil exposure routes.

Ground Water

Ground water in the vicinity of the site is considered potable. However, local sulfur concentrations may be higher than what is considered acceptable for drinking without treatment (Tucket, 1991; McMahon, 1991). Drinking water for the cities of Tonawanda, North Tonawanda, and Niagara Falls, and for the towns of Wheatfield and Lockport is currently obtained from the Niagara River (CRA, 1991a). All intakes, except that for the city of Niagara Falls, are located upgradient of the NCR site. The drinking water supply intake for the city of Niagara Falls is located approximately three miles downstream of the site (CRA, 1991a).

There are no known private wells used for drinking, industrial, or irrigation water supplies in the town of Wheatfield. Several industries in Niagara City, downstream of the site, use the Niagara River as a source for industrial water. Water for industrial or agricultural use in the town of Wheatfield is obtained from the municipal water supply (Gwazdak, 1991); however, there may be some unidentified industrial or agricultural users of the Niagara River (McMahon, 1991).

Because adjacent areas are zoned residential, ground water at the site is classified as potable, and private drinking wells can be installed, future ingestion of ground water by residents is evaluated in the exposure assessment. Two distinct evaluations are performed: one for ground water surrounding the perimeter of the landfill and outside the clay barrier surrounding the landfill cells, and the other for ground water from the interior of the northernmost industrial landfill cell.

Although the hydrogeological connection between ground water from the perimeter of the landfill and ground water from the interior of the landfill is not completely understood, a clay barrier is believed to surround the landfill and may inhibit the horizontal and vertical migration of ground water contaminants away from the site (CRA, 1991a). A natural downward hydraulic gradient present in the site's aquifer, however, may facilitate the migration of contaminants from the overburden aquifer through the semi-confining layer and into the bedrock aquifer. Horizontal transport of contaminants away from the site is more likely in the bedrock aquifer although contaminant concentrations are predicted to significantly diminish during horizontal transport as a result of dispersion and dilution in the water column.

Separate evaluations of ground water from the perimeter and the interior of the landfill were performed because (1) the hydrogeological connection between the perimeter and interior of the landfill appears to be limited to a certain degree, (2) the contaminant composition of ground water analyzed from the perimeter wells differs from the contaminant composition of ground water from the interior landfill cell and (3) the contaminant composition of ground water from the interior landfill cell may most accurately reflect site related contamination.

Resident exposure via dermal contact with contaminated ground water is not evaluated because the magnitude of such exposure is expected to be low compared to other ground water exposures. This is due to the short duration of exposure, and the low permeability of the skin to compounds that may be present at dilute levels in tap water. Exposure of residents to ground water contaminants through the inhalation of volatilized contaminants during bathing, showering and/or cooking, and the inhalation of volatilized contaminants which have migrated from ground water through basement foundations and accumulated in indoor air, are also not evaluated. Inhalation exposures for both perimeter and interior ground water scenarios are not evaluated because in both sample groups, VOCs were detected at low average

concentrations, and as a result, exposures via other routes are expected to be more significant. Few VOCs were detected in the perimeter ground water samples. In addition, horizontal movement of ground water away from the site is expected to be limited to the bedrock layer which is approximately 45 to 50 feet below ground surface and significantly below the depth of building foundations.

Users of the drinking water supply intake for the city of Niagara Falls, located approximately three miles downstream of the site, are potential receptors for contamination migrating off-site via ground water to the Niagara River. However, exposure to these receptors is expected to be insignificant because contaminant concentrations in ground water are low, horizontal movement of ground water away from the site is minimal, and contaminants migrating off-site are expected to degrade and become diluted over time.

Sediments

Sediment samples were collected from on site and nearby drainage ditches including the drainage ditch running along the wetlands on the north side of the site and the outfall of the culvert discharging to the Niagara River from the site. Exposure of trespassers and excavation workers to sediments via dermal contact and incidental ingestion is evaluated because trespassing occurs on site, neighboring areas are zoned residential, and future excavation of the drainage swales to improve drainage flow may occur. Exposure via inhalation of volatilized contaminants or particulate emissions is not evaluated. The vegetative cover in the drainage swales is expected to limit volatilization and particulate emissions. However, during future excavation of the drainage swales, workers may be exposed via inhalation of volatiles or contaminated particulates. This pathway is expected to be infrequent and therefore, insignificant compared to other exposures and is not evaluated.

Surface Water

Samples were collected from the drainage swales, the Niagara River culvert discharge point and along a drainage swale in the northern wetlands area. Trespassers may contact site contaminated surface waters; however, exposure to surface waters via incidental ingestion and dermal contact is not evaluated. Surface water at the site is mostly intermittent, except for the ditch along the east side of the site, and contaminant concentrations in surface water are low. Anticipated activities (e.g., wading) involve no exposure via the oral route and, due to low contaminant concentrations, insignificant exposure via dermal contact.

Leachate Soils

Approximately 30 leachate seeps have been identified at the site. Soil samples were collected from two of these leachate seeps during the November 1990 sampling round. Under present and future conditions, exposure to chemicals found in leachate soils could occur to youth trespassers who may occasionally contact leachate seeps while engaged in hiking, hunting, or other recreational activities at the site. Exposure via dermal contact and ingestion may occasionally occur and is evaluated. Exposure via inhalation of particulate emissions is not expected to be significant due to the usually wet conditions and isolated locations of leachate seeps.

Leachate Water

Seven leachate water samples were collected during the November 1990 sampling round. The anticipated activities at the site (hiking, hunting, etc.) involve no exposure via incidental ingestion of leachate water. Occasional exposure to leachate water contaminants may occur through dermal contact or inhalation of volatilized chemicals. However, because the seeps are periodic and present in isolated locations throughout

the site, exposure to potential receptors is expected to be insignificant. Data collected during the RI on volatile emissions from leachate seeps is not of sufficient quality to allow a quantitative evaluation of air exposures but these are also expected to be minimal.

Locally Grown Produce

Agricultural fields used for the cultivation of corn and wheat are located along the western boundary of the landfill and between the drainage ditches. A large garden plot used for the cultivation of vegetables by an adjacent resident is located along the eastern boundary of the landfill, adjacent to the north to south running drainage swale. The drainage swale elevation is below the elevation of the farm field. Exposure to human receptors through the ingestion of produce contaminated as a result of uptake of chemicals by roots, or deposition of contaminated dust on plant surfaces is not expected to be significant at the NCR site. The corn and wheat cultivated in the western field may be intended for wide commercial distribution, thus reducing individual exposure. Surface soils at the site have low concentrations of chemicals; therefore, deposition of contaminated dust on plant surfaces is expected to be minimal. Uptake by roots of contaminated ground water emanating from the site is not expected to occur because ground water at the site appears to have minimal horizontal movement in the upper aquifer layers (CRA, 1991a). Finally, application of pesticides on surrounding fields and garden plots is expected to greatly exceed levels of pesticides detected in site media which may contact and/or be absorbed by food crops.

Large scale, uncontrolled future excavation activities, might result in significant deposition of contaminated dust on the eastern farm field. Although no ground water wells used to irrigate farm fields are known to exist in the area, future use of contaminated ground water from the site for irrigation might also result in the

absorption by plants of site contaminants. Both scenarios, are considered unlikely and are not evaluated.

4.3.2 Summary of Exposure Pathways Considered

Table 4-1 summarizes the exposure pathways considered in the risk assessment for both present and future land use and the rationale for their inclusion or exclusion. The pathways retained for the purposes of developing exposure scenarios used for quantitative evaluation include:

- ingestion of ground water, surface and subsurface soils, sediments, and leachate soils; and

- dermal contact with sediments and leachate soils.

4.4 Exposure Scenarios

Multiple exposure scenarios were developed for both present and future use of the NCR site. The following factors were considered in developing these scenarios:

- whether sufficient quantitative data exist to evaluate exposure;
- the frequency and duration of likely exposures; and
- the relative contribution of the exposure to the site-wide total exposure.

The following subsection discusses the specific exposure parameters used to calculate exposure doses.

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TABLE 4-1. NIAGARA COUNTY REFUSE SITE: SUMMARY OF EXPOSURE PATHWAYS

Pathway	Receptor	Time-Frame Evaluated		Degree of Assessment		Rationale for Selection or Exclusion	Data Grouping
		Present	Future	Quant.	Qual.		
Ground Water							
Ingestion of Ground Water	Resident	No	Yes	X	X	Adjacent areas are zoned residential. Although residents currently rely on municipal water, ground water is potable. No private domestic or industrial use wells are known to exist.	All ground water samples (NCR 12D ground water evaluated separately. See 5. on page 2). Impact on downstream water supplies evaluated qualitatively.
Inhalation of Ground Water Contaminants During Showers	Resident	No	No			Concentrations of volatiles in ground water are low.	
Inhalation of Contaminants that Volatilize from Ground Water and Seep into Basements	Resident	No	No			Concentrations of volatiles in ground water are low. Horizontal flow of ground water away from the site appears to be limited.	
Dermal Contact with Ground Water	Resident	No	No			Considered insignificant compared to other ground water exposures.	
Surface Soils							
Incidental Ingestion of Onsite Surface Soils	Trespasser	Yes	Yes	X		Onsite trespassers have been observed.	All surface soils (0'- 3').
Dermal Contact with Onsite Surface Soils*	Trespasser	No	No			Onsite trespassers have been observed. Exposures expected to be minimal.	
Inhalation of VOC Emissions and Particulates from Surface Soils	Trespasser	No	No			Concentrations in surface soils are low.	

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TABLE 4-1 (CONTINUED)

Pathway	Receptor	Time-Frame Evaluated: Present	Time-Frame Evaluated: Future	Degree of Assessment: Quant.	Degree of Assessment: Qual.	Rationale for Selection or Exclusion	Data Grouping
Subsurface Soils							
Incidental Ingestion of Onsite Subsurface Soils	Excavation Worker	Yes	Yes	X		Exposure to subsurface soils (2' to 15') may occur during excavations for utility and landfill maintenance.	All subsurface soils less than or equal to 15'.
Dermal Contact with Onsite Subsurface Soils*	Excavation Worker	No	No			Exposure to subsurface soils (2' to 15') may occur during excavations for utility and landfill maintenance. Exposures expected to be minimal.	
Sediments							
Incidental Ingestion of Drainage Swale Sediments	Trespasser	Yes	Yes	X		Onsite trespassers have been observed.	All sediment samples.
	Excavation Worker	No	Yes	X		Exposure to drainage swale sediments may occur during utility and landfill maintenance.	
Dermal Contact with Drainage Swale Sediments**	Trespasser	Yes	Yes	X		Onsite trespassers have been observed.	All sediment samples.
	Excavation Worker	No	Yes	X		Exposure to sediments may occur during utility and landfill maintenance.	
Inhalation of VOC Emissions and Particulates from Drainage Swale Sediments	Trespasser	No	No			Moisture content, absence of physical disturbance and vegetation limit release of particulates. Vegetative cover limits VOC emissions.	
	Excavation Worker	No	No				
Surface Water							
Incidental Ingestion of Surface Water	Trespasser	No	No			Anticipated activity involves negligible exposure via the oral route.	

TABLE 4-1 (CONTINUED)

Pathway	Receptor	Time-Frame Evaluated		Degree of Assessment		Rationale for Selection or Exclusion	Data Grouping
		Present	Future	Quant.	Qual.		
Dermal Contact with Surface Water	Trespasser	No	No			Anticipated activity involves limited exposure. Concentrations in surface water are low.	
Leachate Soils							
Incidental Ingestion of Leachate Soils	Trespasser	Yes	Yes	X		Onsite trespassers have been observed and may occasionally contact leachate seep.	All leachate soil samples.
Dermal Contact with Leachate Soils	Trespasser	Yes	Yes	X		Onsite trespassers have been observed and may occasionally contact leachate seep.	All leachate soil samples.
Inhalation of VOC Emissions and Particulates from Leachate Soils	Trespasser	No	No			Generally wet conditions of leachate limits particulate emissions. Low VOC concentrations in soil (water) suggest insignificant air releases.	
Leachate Water							
Incidental Ingestion of Leachate Water	Trespasser	No	No			Anticipated activity involves negligible exposure. Seeps are periodic.	
Dermal Contact with Leachate Water	Trespasser	No	No			Anticipated activity involves negligible exposure. Seeps are periodic.	
Inhalation of VOC Emissions	Trespasser	No	No			Low VOC concentrations in soil (water) suggest insignificant air releases.	
Home Grown Produce							
Ingestion of Contaminated Home Grown Produce	Adjacent Resident	No	No			Farm field adjacent to the site. Existence of home grown produce contaminated by contaminants emanating from the site is unknown.	

*not evaluated quantitatively, per EPA guidance (no cadmium, PCBs, dioxin detected).
**cadmium only

4.4.1 Exposure Parameters

The parameters used to calculate exposure doses are summarized in Tables 4-2 through 4-10 at the end of Section 4. The tables correspond to the scenarios presented in Table 4-1. Note that parameters are summarized only for those scenarios evaluated quantitatively. Note also that only dermal contact with cadmium is quantitatively evaluated. Due to the inadequacy of data to evaluate this pathway, EPA currently recommends quantitatively evaluating dermal contact exposures to only cadmium, PCBs, and dioxin (EPA Region II, 1992). Cadmium was detected in site sediments and leachate soil but not in surface or subsurface soil. PCBs and dioxin were not detected in any site media. As a result, only scenarios involving dermal contact with sediments and leachate soils are quantitatively evaluated.

Values for exposure parameters used generally reflect reasonable maximum assumptions. Where EPA Headquarters guidance (EPA, 1989a; EPA, 1991a) was prescriptive, these values were adopted. If specific inputs were not recommended by EPA Headquarters guidance, the following sources were used: *Superfund Exposure Assessment Manual* (EPA, 1988b), and *The Exposure Factors Handbook* (EPA, 1989c).

The residential population for future scenarios involving ground water was assumed to be a 70 kg adult. Residents were assumed to be exposed to ground water for a period of 30 years (90th percentile for time at a single residence, EPA, 1989a). For ground water exposures, residents were assumed to ingest contaminated ground water on a daily basis, excluding two weeks spent away from home (350 days/year, EPA, 1991a).

The trespasser was assumed to be a youth, age nine to eighteen years old, weighing 50 kilograms (kg). Trespassing activity was assumed to occur over a period of 10 years (total years in the age group) and exposure to contaminated surface soils and

sediments was assumed to occur frequently, especially during summer months (total of 117 days per year). Exposure by contaminated leachate soils was assumed to occur less frequently, for a total of five days/year. Dermal contact scenarios assumed exposed areas of arms, hands, and legs.

The excavation worker was assumed to be a 70 kg adult exposed to subsurface soils 5 days a week for a period of three months. The duration of exposure was assumed to be one year as recommended by EPA (EPA, 1991a). Dermal contact scenarios assumed soil contact with hands and arms only.

4.5 Quantification of Exposure

The purpose of this section is to describe the methodology and approach for determining exposure point concentrations of COCs and chemical-specific intakes (dose) for the receptors and pathways selected for quantitative evaluation.

4.5.1 Estimation of Exposure Point Concentrations

The exposure point concentration is the measured or estimated amount of a chemical in the environmental medium of concern at the point of human contact. Exposure point concentrations were developed for each exposure pathway based on available site sampling data (Section 2.0). Conservatively, concentrations at exposure points for present and future scenarios were assumed to be those measured during the RI. In general, no dilution or degradation was assumed.

The exposure point concentrations for soils are expressed in mass per unit weight (mg/kg) and for water in mass per unit volume (mg/L). To represent the reasonable maximum exposures (as defined by EPA, 1989a), the upper 95 percent confidence limit of the mean concentration or the maximum concentration was used as the

exposure point concentration. The methodology for determining these values was described in Section 2.0. In brief, the upper 95 percent confidence limit concentration was calculated and compared to the maximum concentration; the lower of the two values was used as the exposure concentration. When the upper 95 percent confidence limit was greater than the maximum, it usually indicated a small sample size and widely distributed data. In these cases, it was believed that the upper 95 percent confidence limit did not adequately represent the available sampling data.

For averaging purposes, values of one-half the detection limit were used for concentrations below the detection limit if there were other positive results for a chemical in a particular medium.

4.5.2 Exposure Doses

The following standard EPA equation (EPA, 1989a) was used to estimate exposure doses received by the receptor populations for all scenarios:

$$I = \frac{C \times CR \times EF \times ED}{BW \times AT}$$

Where:

I	=	Intake Dose (mg/kg/day)
C	=	Concentration (mg/kg or mg/L)
CR	=	Contact Rate (kg/day or L/day)
EF	=	Exposure Frequency (days/year)
ED	=	Exposure Duration (years)
BW	=	Body Weight (kg)
AT	=	Averaging Time (days)

Each of the exposure parameters from Tables 4-2 through 4-10 were applied to this general equation. The specific exposure dose calculations are presented in Appendix E.

To evaluate noncarcinogenic health effects, exposure point concentrations were calculated for subchronic (short-term), and chronic (long-term) exposure periods. Subchronic and chronic doses were calculated for exposures over a variety of exposure periods depending on the receptor population. These population-specific exposure periods are identified in Tables 4-2 through 4-10. When assessing carcinogenic health effects, only chronic doses were calculated. These were averaged over a 70-year lifetime.

TABLE 4-2. EXPOSURE PATHWAY: INGESTION OF GROUND WATER BY LOCAL RESIDENT FOR FUTURE SCENARIO

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Local Residents	
Body Weight (kg)					
Adult Resident	--	--	70	Per EPA Guidance	RAGS Suppl.
Duration of Exposure (years)					
Adult Resident	1 - 70	35	30	90th percentile for time at a single residence	RAGS Suppl.
Exposure Frequency (days/year)	1 - 365	182.5	350	Per EPA Guidance	RAGS Suppl.
Ingestion Rate (l/day)					
Adult Resident	--	--	2	Per EPA Guidance	RAGS Suppl.
Averaging Time (days)					
noncarcinogens	--	--	10950	Values used are based on exposure duration for noncarcinogens and lifetime exposure for carcinogens	RAGS
carcinogens	--	--	25550		

RAGS Suppl.: U.S. EPA, *Risk Assessment Guidance for Superfund, Vol. I. Supplemental Guidance: Standard Default Exposure Factors,* Interim Final. Office of Emergency and Remedial Response. March 1991.
RAGS: U.S. EPA, *Risk Assessment Guidance for Superfund, Volume I,* EPA 540/1-89/002. Office of Emergency and Remedial Response. December 1989.

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TABLE 4-3. EXPOSURE PATHWAY: INGESTION OF SURFACE SOILS BY YOUTH TRESPASSER FOR PRESENT AND FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Youth Trespasser	
Body Weight (kg) Youth (Age 9-18)	30.7-66.7	48.7	50	Value used is average of all age groups in range	EFH
Duration of Exposure (years) Youth	1 - 10	5	10	Total years in age group	
Exposure Frequency (days/year) Youth	1-273	136.5	117	Assumes youth trespasses 5 d/wk during summer, and 2 d/wk during spring and fall (39 weeks total).	
Ingestion Rate (mg/day) Youth	--	--	100	Value used is specified for children more than 6 years old	RAGS Sup
Fraction Ingested from Contaminated Source (unitless)	--	--	1	Assume that all soil contacted is contaminated	RAGS
Averaging Time (days) noncarcinogenic carcinogenic	-- --	-- --	3650 25550	Values used are based on exposure duration for noncarcinogens and lifetime exposure for carcinogens	RAGS

RAGS Suppl.: *Risk Assessment Guidance for Superfund, Vol. I Supplemental Guidance: Standard Default Exposure Factors, Interim Final.* Office of Emergency and Remedial Response. March 1991.
EFH: *Exposure Factors Handbook*, EPA 600/8/-89/043. Exposure Assessment Group, Office of Health and Environmental Assessment. 1989.
RAGS: *Risk Assessment Guidance for Superfund, Volume I*, EPA 540/1-89/002. Office of Emergency and Remedial Response. December 1989.

TABLE 4-4. EXPOSURE PATHWAY: INGESTION OF ONSITE SUBSURFACE SOILS BY EXCAVATION WORKER FOR FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Excavation Worker	
Body Weight (kg)					
Excavation Worker	--	--	70	Per EPA Guidance	RAGS Suppl.
Duration of Exposure (years)					
Excavation Worker	1 - 30	15	1	Per EPA Guidance	RAGS Suppl.
Exposure Frequency (days/year)					
Excavation Worker	1 - 365	182.5	65	Assume excavation occurs 5 days/week for 3 months	
Ingestion Rate (mg/day)					
Excavation Worker	--	--	480	Value used is specified for adults	RAGS Suppl.
Fraction Ingested from Contaminated Source (unitless)	--	--	1	Assume that all soil contacted is contaminated	RAGS
Averaging Time (days)					
noncarcinogens	--	--	91	Values used are based on exposure duration for noncarcinogens and lifetime exposure for carcinogens	RAGS
carcinogens	--	--	25550		

RAGS Suppl.: *Risk Assessment Guidance for Superfund, Vol. I. Supplemental Guidance: Standard Default Exposure Factors*, Interim Final. Office of Emergency and Remedial Response. March 1991.
RAGS: *Risk Assessment Guidance for Superfund, Volume I*, EPA 540/1-89/002. Office of Emergency and Remedial Response. December 1989.

TABLE 4-5. EXPOSURE PATHWAY: INCIDENTAL INGESTION OF SEDIMENTS BY YOUTH TRESPASSER FOR PRESENT AND FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Youth trespasser	
Body Weight (kg) Youth (Age 9-18)	30.7-66.7	48.7	50	Value used is average of all age groups in range	EFH
Duration of Exposure (years) Youth	1 - 10	5	10	Total years in age group	
Exposure Frequency (days/year) Youth	1 - 273	136.5	117	Assumes youth trespasses 5 days/wk during summer, and 2 day/wk during spring and fall (39 weeks total)	
Ingestion Rate (mg/day) Youth	--	--	100	Value used is specified for children more than 6 years old	RAGS Suppl.
Fraction Ingested from Contaminated Source (unitless)	--	--	1	Assumes that all soil contacted is contaminated	RAGS
Averaging Time (days) noncarcinogenic carcinogenic	 -- --	 -- --	 3650 25550	Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS

RAGS Suppl.: U.S. EPA, *Risk Assessment Guidance for Superfund, Vol. I. Supplemental Guidance: Standard Default Exposure Factors,* Interim Final. Office of Emergency and Remedial Response. March 1991.
RAGS: U.S. EPA, *Risk Assessment Guidance for Superfund, Volume I,* EPA 540/1-89/002. Office of Emergency and Remedial Response. December, 1989.
EFH: *Exposure Factors Handbook,* EPA 600/8-89/043. Exposure Assessment Group, Office of Health and Environmental Assessmen 1989.

TABLE 4-6. EXPOSURE PATHWAY: INCIDENTAL INGESTION OF SEDIMENTS BY EXCAVATION WORKERS FOR FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Excavation Workers	
Body Weight (kg) Excavation Worker	--	--	70	Per EPA Guidance	RAGS Suppl.
Duration of Exposure (years) Excavation Worker	1 - 30	15	1	Per EPA Guidance	RAGS Suppl.
Exposure Frequency (days/year) Excavation Worker	1 - 365	183	65	Assumes excavation occurs 5 days/week for 3 months	
Ingestion Rate (mg/day) Excavation Worker	--	--	480	Value used is specified for adults	RAGS Suppl.
Fraction Ingested from Contaminated Source (unitless)	--	--	1	Assumes that all soil contacted is contaminated	RAGS
Averaging Time (days) noncarcinogenic carcinogenic	 -- --	 -- --	 91 25550	Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS

RAGS Suppl.: U.S. EPA, *Risk Assessment Guidance for Superfund, Vol. I. Supplemental Guidance: Standard Default Exposure Factors*, Interim Final. Office of Emergency and Remedial Response. March 1991.
RAGS: U.S. EPA, *Risk Assessment Guidance for Superfund, Volume I*, EPA 540/1-89/002. Office of Emergency and Remedial Response. December 1989.

TABLE 4-7. EXPOSURE PATHWAY: DERMAL CONTACT WITH SEDIMENTS BY YOUTH TRESPASSER FOR PRESENT AND FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Youth Trespasser	
Body Weight (kg) Youth (Age 9-18)	30.7-66.7	48.7	50	Value used is average of all age groups in range	EFH
Duration of Exposure (years) Youth	1 - 10	5	10	Total years in age group	
Exposure Frequency (days/year) Youth	1 - 273	136.5	117	Assume youth trespasses 5 days/wk during summer and 2 days/wk during spring and fall (39 weeks total)	
Skin Surface Area Contacted (sq. cm) Arms Hands Legs Total Area of These Limbs	 1430-3060 610-990 3330-5880 5370-9930	 2250 800 4610 7660	 2130 810 4780 7720	50th percentile values averaged over entire age group	EFH
Soil Skin Adherence Factor (mg/sq. cm)	--	--	0.6	EPA Region II	
Absorption Factor Cadmium	0.001-0.01	0.005	.005	Value used is midpoint of range given by EPA, Region II	EPA Region II
Averaging Time (days) noncarcinogenic carcinogenic	 -- --		 3650 25550	Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS

RAGS Suppl.: U.S. EPA, *Risk Assessment Guidance for Superfund, Vol. I Supplemental Guidance: Standard Default Exposure Factors*, Interim Final. Office of Emergency and Remedial Response. March 1991.
RAGS: U.S. EPA, Region I, *Supplemental Risk Assessment Guidance for the Superfund Program*. Draft Final, EPA 901/5-89-001. June 1989.
SEAM: U.S. EPA, *Superfund Exposure Assessment Manual*, EPA 540/1-88/001. Office of Remedial Response. December 1989.
EFH: U.S. EPA, *Exposure Factors Handbook*, EPA 600/8-89/043. Exposure Assessment Group, Office of Health and Environmental Assessment. 1989
RAGS: U.S. EPA, *Risk Assessment Guidance for Superfund, Volume I*, EPA 540/1-89/002. Office of Emergency and Remedial Response. December 1989.
EPA Region II: Telephone conversations between TRC and Mark Maddaloni and Peter Grevatt, EPA Region II Risk Group, on February 4, 1992 and January 23, 1992, respectively.

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TABLE 4-8. EXPOSURE PATHWAY: DERMAL CONTACT WITH SEDIMENTS BY EXCAVATION WORKERS FOR FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Excavation Workers	
Body Weight (kg) Excavation Worker	--	--	70	Per EPA Guidance	RAGS Suppl.
Duration of Exposure (years) Excavation Worker	1 - 30	15	1	Per EPA Guidance	RAGS Suppl.
Exposure Frequency (days/year) Excavation Worker	1 - 365	183	65	Assumes excavation occurs 5 days/week for 3 months	
Skin Surface Area Contacted (sq. cm) Arms Hands Total Area of These Limbs	 -- -- --	 -- -- --	 2300 820 3120	Per EPA Guidance	RAGS
Soil Skin Adherence Factor (mg/sq. cm)	--	--	0.6	EPA Region II	
Absorption Factor Cadmium	0.001-0.01	0.005	.005	Value used is midpoint of range given by EPA Region II	EPA Region II
Averaging Time (days) noncarcinogenic carcinogenic	 -- --		 91 25550	Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS

RAGS Suppl.: U.S. EPA, *Risk Assessment Guidance for Superfund, Vol. I Supplemental Guidance: Standard Default Exposure Factors*, Interim Final. Office of Emergency and Remedial Response. March 1991.
EFH: U.S. EPA, *Exposure Factors Handbook*, EPA 600/8-89/043. Exposure Assessment Group, Office of Health and Environmental Assessment. 1989
RAGS: U.S. EPA, *Risk Assessment Guidance for Superfund, Volume I*, EPA 540/1-89/002. Office of Emergency and Remedial Response. December 1989.
EPA Region II: Telephone conversations between TRC and Mark Maddaloni and Peter Grevatt, EPA Region II Risk Group, on February 4, 1992 and January 23, 1992, respectively.

TABLE 4-9. EXPOSURE PATHWAY: INGESTION OF LEACHATE SOILS BY YOUTH TRESPASSER FOR PRESENT AND FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Youth Trespasser	
Body Weight (kg) Youth (Age 9-18)	30.7-66.7	48.7	50	Value used is average of all age groups in range	EFH
Duration of Exposure (years) Youth	1 - 10	5	10	Total years in age group	
Exposure Frequency (days/year) Youth			5	Assumes youth trespasses leachate seeps 5 days/year.	
Ingestion Rate (mg/day) Youth	--	--	100	Value used is specified for children more than 6 years old	RAGS Suppl.
Fraction Ingested from Contaminated Source (unitless)	--	--	1	Assume that all soil contacted is contaminated	RAGS
Averaging Time (days) noncarcinogenic carcinogenic	-- --	-- --	3650 25550	Values used are based on exposure duration for noncarcinogens and lifetime exposure for carcinogens	RAGS

RAGS Suppl.: *Risk Assessment Guidance for Superfund, Vol. I Supplemental Guidance: Standard Default Exposure Factors, Interim Final.* Office of Emergency and Remedial Response. March 1991.
EFH: *Exposure Factors Handbook*, EPA 600/8/-89/043. Exposure Assessment Group, Office of Health and Environmental Assessment. 1989.
RAGS: *Risk Assessment Guidance for Superfund, Volume I*, EPA 540/1-89/002. Office of Emergency and Remedial Response. December 1989.

TABLE 4-10. EXPOSURE PATHWAY: DERMAL CONTACT WITH LEACHATE SOILS BY YOUTH TRESPASSER FOR PRESENT AND FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Youth Trespasser	
Body Weight (kg) Youth (Age 9-18)	30.7-66.7	48.7	50	Value used is average of all age groups in range	EFH
Duration of Exposure (years) Youth	1 - 10	5	10	Total years in age group	
Exposure Frequency (days/year) Youth			5	Assume youth trespasses leachate seeps 5 days/year	
Skin Surface Area Contacted (sq cm)					
	1430-3060	2250	2130	50th percentile values averaged over entire age group	EFH
Arms	610-990	800	810		
Hands	3330-5880	4610	4780		
Legs					
	5370-9930	7660	7720		
Total Area of These Limbs					
Soil Skin Adherence Factor (mg/sq cm)	--	--	0.6	EPA Region II	
Absorption Factor Cadmium	0.001-0.01	0.005	.005	Value used is midpoint of range given by EPA, Region II	EPA Region II
Averaging Time (days)					
noncarcinogenic	--	--	3650	Values used are based on exposure duration for noncarcinogens and lifetime exposure for carcinogens	RAGS
carcinogenic	--	--	25550		

EFH: *Exposure Factors Handbook*, EPA 600/8-89/043. Exposure Assessment Group, Office of Health and Environmental Assessment. 1989

RAGS: *Risk Assessment Guidance for Superfund, Volume I*, EPA 540/1-89/002. Office of Emergency and Remedial Response. December 1989.

EPA Region II: Telephone conversations between TRC and Mark Maddaloni and Peter Grevatt, EPA Region II Risk Group, on February 4, 1992 and January 23, 1992, respectively.

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5.0 TOXICITY AND DOSE-RESPONSE ASSESSMENT

5.1 Introduction

This section presents scientific evidence of toxicity and information that relates chemical exposure (dose) to anticipated health effects (responses) for each COC. Health criteria derived from dose-response data are used in the next section to estimate the carcinogenic and noncarcinogenic risks associated with exposure to these COCs.

Toxicity information was obtained from the scientific literature and EPA's Integrated Risk Information system (IRIS) on-line data base. Health criteria were obtained from the following sources, listed in descending order of use:

- IRIS (EPA, 1991d),
- Health Effects Assessment Summary Tables (HEAST) (EPA, 1991f),
- Direct Communication with EPA's Environmental Criteria and Assessment Office (ECAO),
- EPA Criteria documents, and
- Agency for Toxic Substances and Disease registry (ATSDR) Toxicological Profiles.

A summary of the relevant dose-response criteria for all COCs used in the risk assessment appears in Table 5-1. The table contains available oral slope factors for evaluating carcinogenic risks, and oral chronic reference doses (RfDs) and subchronic reference doses (RfDs) used to evaluate noncarcinogenic risks. Interim dose-response criteria obtained from ECAO (EPA, 1991c; EPA, 1992a; EPA, 1992b; EPA, 1992c; and EPA, 1992d) are also included in this table for certain RfDs which were not available in IRIS or HEAST.

TABLE 5-1. TOXICITY VALUES FOR THE NCR SITE CONTAMINANTS OF CONCERN.

CHEMICAL	CARCINOGENIC				CHRONIC		SUBCHRONIC	
	Weight of Evidence Classification		Oral Slope Factor (mg/kg/day)-1		Chronic Oral RfD (mg/kg/day)		Subchronic Oral RfD (mg/kg/day)	
Volatiles								
Acetone	D	a			1.00E-01	a	1.00E+00	b
Benzene	A	a	2.90E-02	a				
2-Butanone (MEK)	D	a			5.00E-02	b	5.00E-01	b
1,4 Dichlorobenzene (para)	C	b	2.40E-02	b	1.00E-01	d	1.00E-01	i
Methylene chloride	B2	a	7.50E-03	a	6.00E-02	a	6.00E-02	b
Styrene	B2	b	3.00E-02	b	2.00E-01	a	2.00E+00	b
Trichloroethylene	B2	b	1.10E-02	b	6.00E-03	d	6.00E-03	i
1,2,4 Trimethylbenzene	D	l			6.00E-04	d	6.00E-04	i
Vinyl chloride (chloroethylene)	A	b	1.90E+00	b				
BNAs								
Benzo(a)anthracene	B2	a	5.79E-01	e				
Benzo(a)pyrene	B2	a	5.79E+00	a				
Bis(2-ethylhexyl)phthalate	B2	a	1.40E-02	a	2.00E-02	a	2.00E-02	b
4-Chloroaniline	--				4.00E-03	a	4.00E-03	b
2,4-Dimethylphenol					2.00E-02	b	2.00E-01	b
2,6-Dinitrotoluene	B2	b	6.80E-01	b, k				
2-Methylphenol (o-cresol)	--	b			5.00E-02	a	5.00E-01	b
4-Methylphenol (p-cresol)	C	a			5.00E-02	b	5.00E-01	b
Naphthalene	D	a			4.00E-03	b	4.00E-02	b
Phenanthrene	D	a						
Phenol	D	a			6.00E-01	a	6.00E-01	b
Pesticides								
Aldrin	B2	a	1.70E+01	a	3.00E-05	a	3.00E-05	b
delta-BHC	--							
4,4' DDE	B2	a	3.40E-01	a				
4,4' DDT	B2	a	3.40E-01	a	5.00E-04	a	5.00E-04	b
Dieldrin	B2	a	1.60E+01	a	5.00E-05	a	5.00E-05	b
Heptachlor	B2	a	4.50E+00	a	5.00E-04	a	5.00E-04	b
Heptachlor epoxide	B2	a	9.10E+00	a	1.30E-05	a	1.30E-05	i
Inorganics								
Aluminum	D	d			1.00E+00	d	1.00E+00	i
Antimony	--	a			4.00E-04	a	4.00E-04	b
Arsenic	A	a	1.75E+00	f	3.00E-04	a	1.00E-03	b
Barium	--	a			5.00E-02	b	5.00E-02	b
Beryllium	B2	a	4.30E+00	a	5.00E-03	a	5.00E-03	b
Cadmium (j)	B1	a			5.00E-04	a, g	5.00E-04	i
Cobalt	--					d		
Copper	D	c			4.00E-02	d	4.00E-02	i
Cyanide	D	a			2.00E-02	a	2.00E-02	b
Iron	D	d			5.00E-01	d	5.00E-01	i
Lead	B2	a						

TABLE 5-1. TOXICITY VALUES FOR THE NCR SITE CONTAMINANTS OF CONCERN. (cont.).

CHEMICAL	CARCINOGENIC		CHRONIC		SUBCHRONIC	
	Weight of Evidence Classification	Oral Slope Factor (mg/kg/day)-1	Chronic Oral RfD (mg/kg/day)		Subchronic Oral RfD (mg/kg/day)	
Manganese	D a		1.00E-01	a	1.00E-01	b
Mercury	D a		3.00E-04	b	3.00E-04	b
Nickel	A a		2.00E-02	a, h	2.00E-02	b
Silver	D a		5.00E-03	a	3.00E-03	b
Thallium	--		7.00E-05	b	7.00E-04	b
Vanadium	D c		7.00E-03	b	7.00E-03	b
Zinc	D a		2.00E-01	b	2.00E-01	b

a. From Integrated Risk Information System (IRIS) 5/1/92.
b. From Health Effects Assessment Summary Tables (HEAST) FY 1991.
c. From Drinking Water Regulations and Health Advisories, April 1992.
d. Interim value from ECAO. See text for specific reference.
e. Oral slope factor for B(a)P used for B(a)A (classified as a B2 carcinogen) with a TEF of 0.1 applied.
f. Arsenic oral slope factor derived from unit risk in IRIS.
g. Cadmium RfD is for water; 1.0E-03 mg/kg/day is RfD for food.
h. Value is for nickel soluble salts.
i. Chronic RfD used as Subchronic RfD if no Subchronic value is available per RAGS.
j. Dermal toxicity values for cadmium have been derived from oral toxicity values applying an absorption factor of 0.01 (10%) per EPA guidance (see text for specific reference). The RfD for both chronic and subchronic dermal exposure is 5.00E-05 mg/kg/day.
k. Value used applies to mixture of 2,4- and 2,6-dinitrotoluene.
l. Carcinogenic Weight of Evidence Classification obtained from Health Effects Assessment document, not IRIS or HEAST.

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Sections 5.2 and 5.3 describe the dose-response information used to evaluate potential carcinogenic and noncarcinogenic effects of COCs for the NCR site. Full toxicity profiles for all COCs are presented in Appendix F.

5.2 Carcinogenic Effects

5.2.1 General Method

The most current EPA carcinogenicity criteria were used to evaluate the effects of known or suspected carcinogenic COCs (Class A, B1, or B2). Carcinogenic risks were estimated using slope factors (also known as cancer potency factors). The slope factor is generally defined as the upper 95 percent confidence limit of the slope of the dose-response curve and is the result of the application of a low-dose extrapolation procedure. If slope factors for a given COC were not available, the applicable exposure pathways for that COC were not assessed quantitatively.

A summary of the available data for carcinogenic effects for each COC is presented in Table 5-2. This table includes the COC, the type of cancer and species from which the oral slope factors were derived, the source of the oral slope factors, and the primary studies used in the derivation of the oral slope factors. Table 5-3 presents an overview of the EPA weight-of-evidence categories for human carcinogenicity. Table 5-4 presents the criteria used to assess human and animal data for each EPA weight-of-evidence category in Table 5-3.

TABLE 5-2. POTENTIAL CARCINOGENIC EFFECTS OF NCR COCS

Chemical	Type of Cancer	Species	Source of Data
VOLATILES			
Acetone	lack of data in humans and animals		IRIS (5/1/92)
Benzene	nonlymphocytic leukemia	human	IRIS (5/1/92)
2-Butanone	no human data/inadequate animal data	--	IRIS (5/1/92)
1,4-Dichlorobenzene	hepatocellular adenomas/carcinomas	rodent	HEAST, 1991
Methylene Chloride	hepatocellular adenomas or carcinomas and hepatocellular cancer and neoplastic nodules	mouse	IRIS (5/1/92)
Styrene	lung and bronchi tumors	mouse	HEAST (1991)
Trichloroethylene	liver tumors	mouse	HEAST (1991)
1,2,4-Trimethylbenzene	lack of data in humans and animals		--
Vinyl chloride	lung tumors	rat	HEAST (1991)
BASE NEUTRAL/ ACID EXTRACTABLES			
Benzo(a)anthracene	pulmonary adenoma/hepatoma	mouse	IRIS (5/1/92)
Benzo(a)pyrene	stomach tumors	rodent	IRIS (5/1/92)
bis(2-Ethylhexyl)phthalate	liver tumors	rodent	IRIS (5/1/92)
4-Chloroaniline	lack of data in animals and humans	--	IRIS (5/1/92)

TABLE 5-2 (CONTINUED)

Chemical	Type of Cancer	Species	Source of Data
2,4-Dimethylphenol	lack of data in animals and humans	--	IRIS (5/1/92)
2,6-Dinitrotoluene	lack of data in animals and humans	--	IRIS (5/1/92)
2-Methylphenol	--	--	--
4-Methylphenol	skin papillomas	mouse	IRIS (5/1/92)
Naphthalene	no human data/inadequate animal data		IRIS (5/1/92)
Phenanthrene	no human data/negative animal data	rat	IRIS (5/1/92)
Phenol	no human data/inadequate animal data		IRIS (5/1/92)
PESTICIDES			
Aldrin	liver carcinomas	mouse	IRIS (5/1/92)
delta-BHC	--	--	--
4,4'-DDT	liver tumors	rodent	IRIS (5/1/92)
4,4'-DDE	liver/thyroid tumors	rodent	IRIS (5/1/92)
Dieldrin	hepatocarcinomas/pulmonary metastases	mouse	IRIS (5/1/92)
Heptachlor	liver tumors	mouse	IRIS (5/1/92)
Heptachlor epoxide	liver carcinoma	mouse	IRIS (5/1/92)
INORGANICS			
Aluminum	NA		---

TABLE 5-2 (CONTINUED)

Chemical	Type of Cancer	Species	Source of Data
Antimony	lack of data in humans and animals	--	IRIS (5/1/92)
Arsenic	skin cancer	human	IRIS (5/1/92)
Barium	lack of data in humans and animals		---
Beryllium	osteosarcomas	rabbits	IRIS (5/1/92)
Cadmium	no human data/inadequate animal data	rat	IRIS (5/1/92)
Cobalt	NA		---
Copper	no human data/inadequate animal data	--	IRIS (5/1/92)
Cyanide, total	lack of data in animals and humans		IRIS (5/1/92)
Iron	NA		---
Lead	renal tumors	human	IRIS (5/1/92)
Manganese	inadequate data in humans and animals		IRIS (5/1/92)
Mercury	inadequate data in humans and animals		IRIS (5/1/92)
Nickel (nickel carbonyl)	lung cancer	rat	IRIS (5/1/92)
Silver	questionable animal data		IRIS (5/1/92)
Thallium	lack of data in humans and animals		IRIS (5/1/92)
Vanadium	NA		
Zinc	inadequate data in humans and animals		IRIS (5/1/92)

** Pending on IRIS.
-- Unclassified by EPA as to carcinogenicity
NA Not Available
IRIS: Integrated Risk Information System on-line database. May 1, 1992.
HEAST: Health Effects Assessment Summary Tables, FY 1991.

TABLE 5-3. THE EPA WEIGHT-OF-EVIDENCE FOR HUMAN CARCINOGENICITY (EPA, 1986b)

Category	Description of Evidence
Group A Human Carcinogen	• Sufficient evidence exists from epidemiological studies to support a causal association between exposure to a given agent and cancer.
Group B Probable Human Carcinogen	
B1	• Limited human evidence and sufficient animal evidence.
B2	• Sufficient animal evidence and no or inadequate human evidence.
Group C Possible Human Carcinogen	• Limited animal evidence and no or inadequate human evidence.
Group D Not Classifiable as to Human Carcinogenicity	• Inadequate animal and human data.
Group E Probable Noncarcinogen	• Evidence of noncarcinogenicity in humans.

TABLE 5-4. EPA CARCINOGENICITY WEIGHT-OF-EVIDENCE CRITERIA FOR HUMAN AND ANIMAL DATA (EPA, 1986b)

Category	Description of Evidence
Human Evidence	
Sufficient	• Evidence indicates a causal relationship between the agent and human cancer.
Limited	• Evidence indicates that a causal relationship is credible, but that alternative explanations, such as chance, bias, or confounding could not be adequately excluded.
Inadequate	• There were few pertinent data, or the available studies, while showing evidence of an association, did not exclude chance, bias, or confounding and therefore a causal interpretation is not credible.
No Data	• Data are not available.
No Evidence	• No association between exposure and an increased risk of cancer in well designed and well conducted independent analytical epidemiological studies.
Animal Evidence	
Sufficient	• Evidence indicates that there is an increased incidence of malignant tumors in (a) multiple species or strains; (b) multiple experiments (e.g., with different routes of administration or using different dose levels; or (c) a single experiment with an unusually high incidence and unusual site or type of tumor, or early age at onset.

TABLE 5-4. (CONTINUED)

Category	Description of Evidence
Limited	• A carcinogenic effect is suggested by the data, but are limited because: (a) the studies involve a single species, strain, or experiment and do not meet criteria for sufficient evidence; (b) the experiments are restricted by inadequate dosage levels, inadequate duration of exposure to the agent, inadequate period of follow-up, poor survival, too few animals, or inadequate reporting; or (c) an increase in the incidence of benign tumors only.
Inadequate	• Evidence indicates that because of major qualitative or quantitative limitations, the studies cannot be interpreted as showing either the presence or absence of carcinogenic effects.
No Data	• Data are not available.
No Evidence	• No increased incidence of neoplasms observed in at least two well-designed and well-conducted animal studies in different species.

5.2.2 *Chemical-Specific Considerations*

PAHs

A number of PAHs, several of which have been demonstrated to be carcinogenic, were detected in the media sampled at the NCR site. All PAHs detected at the site which are class B2 carcinogens were grouped together and are listed on Table 5-5. Per EPA guidance, the slope factor for benzo(a)pyrene (B(a)P) was used for this group of compounds. Toxic equivalency factors (TEFs) provided by EPA Region II (EPA, 1992e), were applied to the B(a)P slope factor as follows:

benzo(a)anthracene	0.1
benzo(b)fluoranthene	0.1
benzo(k)fluoranthene	0.1
chrysene	0.01

The B(a)P slope factor of 5.79 $(mg/kg\text{-}day)^{-1}$ currently available on IRIS was used.

5.3 Noncarcinogenic Effects

5.3.1 *General Method*

Noncarcinogenic human health risks were evaluated by analyzing long-term exposures (chronic), and short-term exposures (subchronic) to contaminants of concern. To evaluate long-term exposures, chronic reference doses (RfD) were used. A chronic reference dose is defined as an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure concentration for the human population over a lifetime, including sensitive subpopulations, that is likely to be without an adverse health effect.

TABLE 5-5. CARCINOGENICITY OF PAHs DETECTED AT THE NCR SITE

PAHs for which there is sufficient evidence of carcinogenicity (B2 carcinogens):

Benzo(a)anthracene
Benzo(a)pyrene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Chrysene

PAHs for which the evidence is inadequate to assess their carcinogenicity:

Acenaphthene
Anthracene
Fluoranthene
Fluorene
2-Methylnaphthalene
Naphthalene
Phenanthrene
Pyrene

Source: EPA, 1991d.

Chronic RfDs are derived from the following equation:

$$\text{RfD (mg/kg/day) or (mg/cu.m)} = \frac{\text{NOAEL or LOAEL}}{\text{(UF) (MF)}}$$

Where:

NOAEL = The "No Observable Adverse Effects Level" which represents a chemical dose at which there is no statistically or biologically significant difference in frequency of an adverse effect between the exposed and control populations.

LOAEL = The "Lowest Observable Adverse Effects Level" which represents the lowest dose at which a statistically significant difference in the frequency of an adverse effect is observed.

UF = Uncertainty Factor; the UF is included to account for differences between species, variation in human sensitivity, and extrapolations from the subchronic to the chronic NOAEL or from the LOAEL to the NOAEL.

MF = Modifying Factor; an additional uncertainty factor that accounts for uncertainties in the overall validity of the study and data base.

To evaluate short-term exposures, subchronic reference doses (RfDs) were used. A subchronic RfD is similar to a chronic RfD except that the duration of exposure is defined by EPA as being "substantially less-than-lifetime," from 2 weeks to 7 years (EPA, 1989a). Subchronic RfDs are derived in the same manner as chronic values unless the uncertainty factor applied to derive a chronic RfD has been applied to

adjust for the extrapolation from subchronic exposure to chronic exposure. In these cases, the uncertainty factor is not applied in deriving the subchronic RfD.

A summary of the available data for chronic noncarcinogenic effects is presented in Table 5-6. Included in the table is the COC, the confidence level of the study used to determine the chronic oral RfD, a description of the critical physiological effect and the species of animal used in the study, the uncertainty/modifying factor, the source of the study used to derive the chronic oral RfD, and the study itself. The confidence levels were provided by IRIS as either high, medium, or low.

Table 5-7 contains a summary of the available data for subchronic noncarcinogenic effects. Included in this table is the COC, a description of the critical physiological effect and the species of animal used in the study, the uncertainty factor, the source of the study used to derive the subchronic oral RfD and the study itself.

In cases where a subchronic oral RfD has not been developed by EPA, the chronic oral RfD was adopted as the subchronic oral RfD per EPA guidance (EPA, 1989a).

If neither the chronic oral RfD nor the subchronic oral RfD was available for a given COC, long-term and short-term oral exposure pathways were not assessed quantitatively.

5.3.2 Chemical-Specific Considerations

PAHs

Several noncarcinogenic PAHs were detected at the site. Toxicity values exist for all noncarcinogenic PAH COCs except phenanthrene. The lack of toxicological data

TABLE 5-6. POTENTIAL CHRONIC NONCARCINOGENIC EFFECTS OF NCR COCS

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
VOLATILES					
Acetone	low	increased liver weight and nephrotoxicity	rat	UF: 1000 MF: 1	IRIS (5/1/92)
Benzene	**	NA			
2-Butanone	--	fetotoxicity	rat	UF: 1000	HEAST (1991)
1,4-Dichlorobenzene	NA	NA			HEAST, 1991
Methylene Chloride	NA	liver toxicity	rat	UF: 100	HEAST (1991)
Styrene	medium	red blood cell and liver effects	dog	UF: 1000 MF: 1	IRIS (5/1/92)
Trichloroethylene	**	NA		UF: -- MF: --	IRIS (5/1/92)
1,2,4-Trimethylbenzene	NA	NA			HEAST, 1991
Vinyl Chloride	NA	NA		UF: -- MF: --	---
BASE NEUTRAL/ACID EXTRACTABLES					
Benzo(a)anthracene	NA	NA			IRIS (5/1/92)
Benzo(a)pyrene	NA	NA			IRIS (5/1/92)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
bis(2-Ethylhexyl)phthalate	medium	increased relative liver weight	guinea pig	UF: 1000 MF: 1	IRIS (5/1/92)
4-Chloroaniline	low	non-neoplastic lesions of splenic capsule	rat	UF: 3000 MF: 1	IRIS (5/1/92)
2,4-Dimethylphenol	low	lethargy, prostration, ataxia, and hematological changes	mouse	UF: 3000 MF: 1	IRIS (5/1/92)
2,6-Dinitrotoluene	**	--	--	UF: -- MF: --	IRIS (5/1/92)
2-Methylphenol	--	--	--	UF: -- MF: --	--
4-Methylphenol	--	--	--	UF: MF:	
Naphthalene	**	decreased body weight gain	rat	UF: 10,000	IRIS (5/1/92), HEAST (1991)
Phenanthrene	NA	NA			IRIS (5/1/92)
Phenol	low	reduced fetal body weight	rat	UF: 100 MF: 1	IRIS (5/1/92)
PESTICIDES					
Aldrin	medium	liver toxicity	rat	UF: 1000 MF: 1	IRIS (5/1/92)
delta-BHC	--	lack of data in animals and humans	--	UF: --	HEAST (1991)
4,4'-DDT	medium	liver lesions	rat	UF: 100 MF: 1	IRIS (5/1/92)
Dieldrin	medium	liver lesions	rat	UF: 100 MF: 1	IRIS (5/1/92)

TABLE 5-6 (CONTINUED)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
4,4'-DDE	NA	NA			IRIS (5/1/92)
Heptachlor	low	liver weight increases	rat	UF: 300 MF: 1	IRIS (5/1/92)
Heptachlor epoxide	low	increased liver-to-body weight ratio	dog	UF: 1000 MF: 1	IRIS (5/1/92)
INORGANICS					
Aluminum	medium	decreased body weight gain; neurotoxicity	mouse	UF: 100	EPA, 1992d
Antimony	low	longevity/blood glucose/cholesterol	rat	UF: 1000 MF: 1	IRIS (5/1/92)
Arsenic	medium	keratosis/hyperpigmentation	human	UF: 3 MF: 1	IRIS (5/1/92)
Barium	medium	increased blood pressure	human	UF: 3 MF: 1	IRIS (5/1/92)
Beryllium	low	no adverse effects	rat	UF: 100 MF: 1	IRIS (5/1/92)
Cadmium	high	significant proteinuria	human	UF: 10 MF: 1	IRIS (5/1/92)
Cobalt	NA	NA			---
Copper	NA	NA			---

TABLE 5-6 (CONTINUED)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
Cyanide, total		weight loss, thyroid effects and myelin degeneration	rat	UF: 100 MF: 5	IRIS (5/1/92)
Iron	NA	NA			---
Lead	NA	CNS effects			HEAST, 1991
Manganese	medium	CNS effects	human	UF: 1 MF: 1	IRIS (5/1/92)
Mercury	**	neurotoxicity; kidney effects	rat	UF: 1000 MF:	IRIS (5/1/92) HEAST, 1991
Nickel (sol. salts)	medium	decreased body and organ weights	rat	UF: 100 MF: 3	IRIS (5/1/92)
Silver	NA	argyria	human	UF: 2	HEAST, 1991
Thallium	NA	increased blood enzyme levels/alopecia	rat	UF: 3000	HEAST, 1991
Vanadium	NA	no adverse effects observed	rat	UF: 100	HEAST,1991
Zinc	**	anemia	human	UF: 10	IRIS (5/1/92) HEAST, 1991

** Pending on IRIS
NA Not Available
--- No data source available
IRIS: Integrated Risk Information System. May 1, 1992.
HEAST: Health Effects Assessment Summary Tables, FY 1991.

TABLE 5-7. POTENTIAL SUBCHRONIC NONCARCINOGENIC EFFECTS OF NCR COCS

Chemical	Critical Effect	Species	Uncert. Factor
VOLATILES			
Acetone	increased liver and kidney weight/nephrotoxicity	rat	100
Benzene	--		
2-Butanone	fetotoxicity	rat	1000
1,4-Dichlorobenzene	--		
Methylene Chloride	liver toxicity	rat	100
Styrene	red blood cell and liver effects	dog	100
Trichloroethylene	--		
1,2,4-Trimethylbenzene	--		
Vinyl Chloride	--		
BASE NEUTRAL/ACID EXTRACTABLES			
Benzo(a)anthracene	--		
Benzo(a)pyrene	--		
bis(2-Ethylhexyl)phthalate	increased relative liver weight	guinea pig	1000
4-Chloroaniline	--		
2,4-Dimethylphenol	neurological signs and hematological changes	mouse	300

TABLE 5-7 (CONTINUED)

Chemical	Critical Effect	Species	Uncert. Factor
2,6-Dinitrotoluene	lack of data in animals and humans		
4-Methylphenol	--		
2-Methylphenol	--		
Naphthalene	decreased body weight gain	rat	1000
Phenanthrene	--		
Phenol	reduced fetal body weight	rat	100
PESTICIDES			
Aldrin	liver lesions	rat	1000
delta-BHC	lack of data in animals and humans		
4,4'-DDT	liver lesions	rat	100
Dieldrin	liver lesions	rat	100
Heptachlor	increased liver weight	rat	300
Heptachlor epoxide	--		
INORGANICS			
Aluminum	--		
Antimony	reduced lifespan, altered blood chemistries	rat	1000
Arsenic	keratosis and hyperpigmentation	human	1

TABLE 5-7 (CONTINUED)

Chemical	Critical Effect	Species	Uncert. Factor
Barium	increased blood pressure	rat	100
Beryllium	no adverse effects observed	rat	100
Cadmium	--		
Cobalt	--		
Copper	local GI irritation	human	NA
Cyanide, total	weight loss, thyroid effects and myelin degeneration	rat	500
Iron	--		
Lead	--		
Manganese	no adverse effects observed	human	1
Mercury	kidney effects	rat	1000
Nickel	reduced body and organ weight	rat	300
Silver	argyria	human	2
Thallium	increased SGOT and serum LDH levels, alopecia	rat	300
Vanadium	no adverse effects observed	rat	100
Zinc	anemia	human	10

Note: All subchronic information is from the *Health Effects Assessment Summary Tables*, FY 1991.
(a) Information is for endosulfan
(b) Information is for chlordane

precludes the derivation of a systemic toxicity value. Per EPA, a surrogate RfD was not applied to phenanthrene due to the uncertainties associated with assuming noncarcinogenic PAHs cause similar effects (EPA, 1991j).

Cadmium

Two oral RfDs are available to evaluate cadmium exposures: 5×10^{-4} mg/kg/day for water consumption and 1×10^{-3} mg/kg/day for food consumption. In this risk assessment, the food consumption RfD was used for soil and sediment exposures and the water consumption RfD for water exposures.

As recommended by EPA Region II (EPA Region II, 1992), dermal exposure to cadmium was assessed quantitatively. In order to do so, a dermal RfD was derived from the oral RfD for water consumption by applying an oral absorption factor of 0.10, as follows:

Oral RfD: 5.0×10^{-4} mg/kg/day

Dermal RfD: 5.0×10^{-4} mg/kg/day x 0.10 = 5.0×10^{-5} mg/kg/day

6.0 RISK CHARACTERIZATION

6.1 Introduction

The goal of the risk characterization is to quantify the increased probability of developing cancer or suffering an adverse acute, subchronic, or chronic noncarcinogenic effect as a result of exposure to site contaminants. The risk information will ultimately be used in evaluating whether remedial action at the site is necessary.

The present and potential future public health risks attributable to the site COCs are discussed in this section. The risk characterization integrates data developed from the hazard identification (Section 2.0), the exposure assessment (Section 4.0), and the toxicity and dose-response assessment (Section 5.0) to derive numerical estimates of carcinogenic and noncarcinogenic risk. Risk from site contaminants is assessed for each potential exposure medium (e.g., soil, ground water) under the "reasonable maximum exposure" conditions described previously.

6.2 General Methodology

Risk is a function of chemical toxicity and the route and duration of exposure. EPA's cancer slope factors, RfDs, and health advisories, discussed in Section 5.0, were used as indicators of toxicity in the risk characterization. The chemical- and pathway-specific doses calculated in accordance with the methods outlined in Section 4.0 are used to represent exposure.

Exposure and risk calculation worksheets are presented in Appendix E of this document. Summary risk tables are presented within the text of this section.

6.2.1 Carcinogenic Risk

The incremental carcinogenic (CA) risk associated with exposure to NCR contaminants was calculated according to the following equation:

Incremental CA Risk = Slope Factor x Dose

where the incremental CA risk represents the probability of developing cancer over a 70-year lifetime from exposure to the contaminants associated with the site. Cancer risk is unitless and is expressed here in scientific notation. For example, a risk of 1×10^{-6} indicates that an individual has one chance in 1,000,000 of developing cancer as a result of exposure to onsite contaminants during a lifetime.

The slope factor represents the carcinogenic potency of a chemical (see Table 5-1). The dose, or intake, represents the amount of contaminant to which a receptor is exposed, as described in Section 4.0. When evaluating carcinogenic risks, the dose in all exposure scenarios is the estimated daily intake of each contaminant, received during the specified period of exposure, and averaged over a 70-year lifetime.

Incremental CA risk was calculated for each COC having a designated slope factor and all applicable exposure pathways (ingestion of ground water and soil media). Risk values for all contaminants assessed were summed by exposure pathway to provide total pathway-specific risks.

EPA has not identified a single value that represents a significant incremental cancer risk. However, the NCP acceptable risk range for Superfund sites has been set at 10^{-4} to 10^{-6} per environmental medium (NCP, 1990). In other words, the goal of the NCP is to reduce the cancer risk associated with site contaminants in a given medium to within or below a range of 1 in 10,000 to 1 in 1,000,000.

6.2.2 Noncarcinogenic Effects

Potential noncarcinogenic effects were evaluated based on a comparison of chemical-specific subchronic or chronic exposure doses (depending on the scenario-specific exposure duration) with corresponding protective doses derived from health criteria, as described in Section 5.0. The result of this comparison is expressed as the Hazard Quotient:

$$\text{Hazard Quotient} = \frac{\text{Exposure Dose}}{\text{Protective Dose}}$$

A Hazard Quotient (HQ) that exceeds unity suggests a greater likelihood of developing an adverse subchronic, or chronic toxic effect. However, the uncertainty factors built into the protective doses result in conservative protective dose values. Therefore, the protective dose is likely well below that for which adverse effects will be seen.

Hazard Quotients were calculated for each contaminant for which health criteria are currently available. The HQs for each contaminant were summed to produce a rough estimate of the exposure pathway-specific risk, the Hazard Index (HI). Where appropriate, Hazard Indices for all soil media exposure pathways were summed to provide a total medium-specific risk. In estimating total noncarcinogenic risk, potential responses were conservatively assumed to be additive. However, all COCs do not have the same or similar toxic endpoints and responses may not be additive. Therefore, in those cases where the HI exceeded one, further analyses were undertaken to evaluate which specific chemicals might exhibit toxic effects.

6.3 Risk Summary

An overall summary of NCR site carcinogenic and noncarcinogenic risks is presented in Tables 6-1 and 6-2, respectively. These tables include cumulative cancer risk values and HIs for each exposure pathway and receptor population, and for present and future land use scenarios. Medium-specific risk, for which the NCP acceptable risk range of 10^{-4} to 10^{-6} applies, are derived by adding all pathways for a given medium. Chemical-specific risk values are presented in Appendix E.

The quantitative risk assessment for dermal contact exposures was limited (EPA Region II, 1992). The high degree of uncertainty in inputs for this pathway limits the ability to quantify dermal risks to only three contaminant types: cadmium, PCBs, and dioxins. Of these, only cadmium was detected at the NCR site. A quantitative assessment for cadmium dermal contact risks is therefore presented here. Dermal contact risks associated with other contaminants are assumed to be no higher than ingestion risks and may be lower.

A narrative detailing the results of the quantitative risk assessment is presented below. In addition, a qualitative discussion is provided for risks associated with exposure pathways not evaluated (e.g., dermal contact and inhalation) and for lead, aluminum, cobalt, and other chemicals for which no toxicity values are currently available.

The greatest carcinogenic risk values are associated with the highly conservative ground water ingestion scenarios. The perimeter ground water scenario produced an incremental risk of 2E-04 while the northern landfill cell ground water scenario produced an incremental risk of 1E-04. Two other exposure pathways produced carcinogenic risk in excess of 10^{-6}; ingestion of surface soils by youth trespassers (4E-06) and ingestion of sediments by youth trespassers (5E-06). These values are all,

TABLE 6-1. SUMMARY OF CARCINOGENIC RISK ESTIMATES FOR THE NCR SITE

Scenario	Receptor	Present/Future	Incremental Risk
Ground Water - Perimeter			
Ingestion	Resident	F	2E-04**
Ground Water - Northern Landfill Cell			
Ingestion	Resident	F	1E-04*
Surface Soil			
Ingestion	Youth Trespasser	P/F	4E-06*
Subsurface Soil			
Ingestion	Excavation Worker	F	7E-07
Sediments			
Ingestion	Youth Trespasser	P/F	5E-06*
Ingestion	Excavation Worker	F	9E-07
Leachate Soils			
Ingestion	Youth Trespasser	P/F	9E-08

*Exceeds 10^{-6} risk
**Exceeds 10^{-4} risk

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TABLE 6-2. SUMMARY OF NONCARCINOGENIC HAZARD INDICES (HI) FOR THE NCR SITE

Scenario	Receptor	Present/Future		Chronic HI
Ground Water - Perimeter				
Ingestion	Resident	F		5E+00*
Ground Water - Northern Landfill Cell				
Ingestion	Resident	F		4E+00*
Surface Soil				
Ingestion	Youth Trespasser	P/F		9E-02
Subsurface Soil				
Ingestion	Excavation Worker	F		7E-01a
Sediments				
Ingestion	Youth Trespasser	P/F		1E-01
Dermal Contact	Youth Trespasser	P/F		2E-03
			Total	1E-01
Ingestion	Excavation Worker	F		7E-01a
Dermal Contact	Excavation Worker	F		1E-03a
			Total	7E-01a
Leachate Soils				
Ingestion	Youth Trespasser	P/F		3E-03
Dermal Contact	Youth Trespasser	P/F		9E-05
			Total	3E-03

TABLE 6-3. SUMMARY OF CUMULATIVE CARCINOGENIC RISK ESTIMATES FOR THE NCR SITE

Scenario	Cumulative Incremental Risk
Cumulative Carcinogenic Risk Estimates For Each Receptor	
Future Youth Trespasser Living in Adjacent Residence:	
Perimeter Ground Water Ingestion	2E-04**
Surface Soil Ingestion	4E-06*
	2E-04**
Perimeter Ground Water Ingestion	2E-04**
Sediments Ingestion	5E-06*
	2E-04**
Perimeter Ground Water Ingestion	2E-04**
Leachate Soils Ingestion	9E-08
	2E-04**
Future Excavation Worker Living in Adjacent Residence:	
Perimeter Ground Water Ingestion	2E-04**
Sediments Ingestion	9E-07*
	2E-04**
Perimeter Ground Water Ingestion	2E-04**
Subsurface Soils Ingestion	7E-07
	2E-04**

(Note: Cumulative risks were calculated based on perimeter ground water ingestion which showed a higher incremental risk than northern landfill cell ground water ingestion.

*Exceeds 10^{-6} risk
**Exceeds 10^{-4} risk

TABLE 6-4. SUMMARY OF CUMULATIVE NONCARCINOGENIC HAZARD INDICES (HI) FOR THE NCR SITE

Scenario	Cumulative Chronic HIs
Cumulative Noncarcinogenic Hazard Indices For Each Receptor	
Future Youth Trespasser Living in Adjacent Residence:	
Perimeter Ground Water Ingestion	5E+00*
Surface Soil Ingestion	9E-02
	5E+00*
Perimeter Ground Water Ingestion	5E+00*
Sediments Ingestion	1E-01
Sediments Dermal Contact	2E-03
	5E+00*
Perimeter Ground Water Ingestion	5E+00*
Leachate Soil Ingestion	3E-03
Leachate Soil Dermal Contact	9E-05
	5E+00*
Future Excavation Worker Living in Adjacent Residence:	
Perimeter Ground Water Ingestion	5E+00*
Subsurface Soil Ingestion	7E-01a
	5E+00
Perimeter Ground Water Ingestion	5E+00*
Sediments Ingestion	7E-01a
Sediments Dermal Contact	1E-03a
	6E+00*

*Hazard Index exceeds one (1).
a - Subchronic HIs were calculated for this scenario.

however, within the acceptable risk range established by the NCP. Cumulative carcinogenic risk estimates for each receptor did not exceed the incremental risk level for future resident ingestion of perimeter ground water.

Noncarcinogenic HIs, summed across chemicals for each exposure route, exceeded one for perimeter ground water ingestion (HI = 5) and for northern landfill cell ground water ingestion (HI = 4). No other exposure pathways produced noncarcinogenic HIs greater than one. The cumulative noncarcinogenic Hazard Index for a future excavation worker living in an adjacent residence totaled 6.

6.3.1 Ground Water

Perimeter Ground Water

Carcinogenic Risks

Carcinogenic risks associated with ingestion of ground water located outside the landfill perimeter were attributed primarily to concentrations of arsenic (5.73 ug/L), and beryllium (1.05 ug/L). Arsenic was detected in 20 out of the 44 samples analyzed with a maximum concentration detected of 16.40 ug/L from sample location NCR-1 (see Appendix A). Other sample locations with high arsenic concentrations in both sampling rounds were NCR-2 (15.4 ug/L) and NCR-11 (13 ug/L). Beryllium was detected in 8 out of 45 samples with a maximum concentration of 3.1 ug/L attributable to sample location NCR-2I.

Noncarcinogenic Risks

The chronic HI exceeded one for the perimeter ground water ingestion scenario. Two inorganics resulted in chronic HQs greater than one: iron (30,400 ug/L) and antimony (16.3 ug/l). The iron HQ is based on an interim RfD obtained from ECAO. The HQs for aluminum (19,100 µg/L), arsenic (5.73 ug/L), and manganese (1870 ug/L) were

slightly less than one. Antimony was detected in 4 out of 41 samples analyzed. All detections for antimony occurred in samples collected from location NCR-11 with a maximum concentration detected of 69.80 ug/L. Iron was detected in 42 of the 45 samples analyzed at concentrations up to 108,000 ug/L. The maximum concentration detected occurred a sample collected from location NCR-2. Aluminum occurred at high maximum concentrations in ground water in 32 of the 44 samples analyzed. The maximum and upper 95 percent confidence limit concentrations calculated for aluminum are 80,800 ug/L (sample location NCR-2) and 19,125 ug/L, respectively. The frequency and range of arsenic detections are noted under Carcinogenic Risks. Manganese was detected in 43 of the 45 samples analyzed at concentrations up to 3,930 ug/L and with a mean of 135.41 ug/L. The maximum concentration detected is attributed to sample location NCR-2.

Other Potential Risks

Lead was detected in 11 out of 43 samples from the perimeter ground water monitoring wells. The maximum concentration detected, 77.90 ug/L (sample location NCR-2), exceeds the MCL/action level of 15 ug/L for drinking water sources. The upper 95 percent confidence limit of 13.01 ug/L, however, did not exceed the action level.

Cobalt, which was detected in 9 out of the 45 samples with a maximum concentration of 43.90 ug/L detected in NCR-2, also could not be assessed due to a lack of toxicity data. However, calculated dose estimates (see Appendix E) are well below 0.06 mg/kg/day, a value typical of dietary intake (EPA, 1992b). All other contaminants detected but not assessed due to lack of toxicity data occurred infrequently (in less than 5% of the samples analyzed) and at low concentrations.

Carcinogenic Risks

Carcinogenic risks associated with ingestion of groundwater from the northern most landfill cell are attributable primarily to concentrations of arsenic (2.5 ug/L), beryllium (1.0 ug/L), and styrene (.67 ug/L). Arsenic and beryllium were both detected in one out of the three samples analyzed. Styrene was detected in all three samples; however, one sample, NCR-12DSP, collected by the TRC oversight team, exceeded the other two styrene levels detected by an order of magnitude.

Noncarcinogenic Risks

Chronic HQs exceeded one only for antimony which was detected in 2 out of the 3 samples analyzed and at a maximum concentration of 44.7 ug/L.

Other Potential Risks

The following chemicals, and their associated frequencies of detection, were detected in northern landfill cell ground water but could not be assessed due to lack of appropriate toxicity data: isopropylbenzene (1 out of 1 sample), endosulfan sulfate (1 out of 3 samples), and cobalt (1 out of 3 samples). Cobalt dose estimates were below a typical dietary intake level of 0.06 mg/kg/day.

Neither the dermal contact or inhalation exposure routes were quantitatively evaluated in the perimeter and northern landfill cell exposure scenarios. As explained in Section 5.0, exposure via dermal contact or inhalation during household use would be expected to be minimal due to low contaminant concentrations and short exposure duration. Depth to ground water which may contain site contaminants precludes the possible infiltration of volatile organics from ground water through basement foundations and into indoor air.

Like all other exposure scenarios evaluated in this risk assessment, the ground water scenarios for both perimeter and northern landfill cell ground water are based on maximum concentrations or the upper 95 percent confidence limit concentrations, which ever is lower. This approach provides a conservative estimate of the reasonable maximum concentration. In addition, the scenarios are based on the conservative assumptions that ground water in the vicinity of the site will be used as a drinking water supply in the future or that contamination will enter the existing supply. These assumptions are unlikely for several reasons; (1) public water is available to all residents and industries adjacent to the site, (2) the intake of the city of North Tonawanda and the town of Wheatfield public water supply is located upgradient of the site, and (3) ground water movement away from the site appears to be minimal (CRA, 1991a). These factors suggest that the ground water scenarios presented above are very conservative (i.e., health protective).

6.3.2 Soils

Surface Soils

Carcinogenic Risks

Ingestion of site surface soils for the youth trespasser scenario produced a risk level exceeding 10^{-6} but within the EPA's target range of 10^{-6} to 10^{-4}. The risk was attributable primarily to arsenic which was detected in all twelve surface soil samples analyzed at concentrations ranging from 2.7 mg/kg to 28 mg/kg. The maximum concentration detected was from sample location NCR-11.

Noncarcinogenic Risks

Chronic HQs did not exceed one for the youth trespasser ingestion scenario evaluated.

Other Potential Risks

Cobalt was detected in all twelve samples analyzed with concentrations ranging from 3 mg/kg to 14 mg/kg (NCR-8). Delta-BHC was also detected in one surface soil sample at 1.4 ug/kg. Toxicity data are unavailable for these chemicals, or is not adequately characterized, and therefore, an assessment of their potential effects is not possible. However, cobalt dose estimates are below a typical dietary intake level of 0.06 mg/kg/day.

Lead was detected in all samples analyzed. Concentrations in surface soils ranged from 4.9 mg/kg to 175 mg/kg (NCR-11) which was below the EPA's target cleanup level for lead of 500 mg/kg to 1000 mg/kg (EPA, 1989e).

Exposures via dermal contact were not quantitatively evaluated. The reason for this is that, as discussed previously, EPA considers there to be sufficient data to evaluate the dermal pathway for only three contaminants: cadmium, PCBs, and dioxins. None of these contaminants were detected in soils at the site. Inhalation was also not evaluated quantitatively due to low contaminant concentrations in surface soils. In general, exposure via both of these routes is expected to be insignificant due to the low contaminant concentrations and low frequency of exposure.

Subsurface Soils

Carcinogenic Risks

All carcinogenic risk estimates for the subsurface soil scenario were below 10^{-6}. Therefore, based on quantitative risk estimates, carcinogenic risk from ingestion of subsurface soils are assumed to insignificant.

Noncarcinogenic Risks

All noncarcinogenic risk estimates for the subsurface soil ingestion pathway resulted in a total HI of less than one.

Other Potential Risks

Lead was detected in all subsurface soil samples analyzed at concentrations ranging up to 20 mg/kg (Testpit 3). However, this concentration is below the EPA's target clean-up level for lead of 500 to 1000 mg/kg (EPA, 1989e).

Cobalt was also detected frequently and at a maximum concentration of 15 mg/kg. This maximum concentration is from sampling location NCR-3. Toxicity data are unavailable for this inorganic therefore an assessment of the potential effects is not possible. However, dose estimates are below accepted safe levels.

Exposures via dermal contact and/or inhalation were not quantitatively evaluated. Exposure via inhalation is expected to be minimal due to the short duration of expected exposure. Exposure via dermal contact could not be evaluated quantitatively because inadequate data is available on the dermal toxicity of the contaminants detected. Exposure via dermal contact may result in added risk. However, in the scenario evaluated, worker exposure via dermal contact is expected to be minimized by the wearing of appropriate work clothing such as gloves, hard hats, and long slacks.

Chemical contaminants in soils at the site, including deep subsurface soils, may potentially leach into ground water and contribute to risks posed by exposure to contaminated ground water. However, the resulting ground water concentrations are expected to be minimal due to the reported semi-confining layer at the site and the low soil concentrations.

Sediments

Carcinogenic Risks

Incidental ingestion of sediments produced a risk exceeding 10^{-6} for the youth trespasser scenario. However, this risk fell within EPA's acceptable risk range of 10^{-6} to 10^{-4}. Risk was attributable primarily to arsenic detected at a maximum concentration of 27.6 mg/kg. Arsenic was detected in all samples analyzed with concentrations ranging from 0.71 mg/kg to 27.6 mg/kg at sampling location SED-10.

Noncarcinogenic Risks

Ingestion of and dermal contact with (for cadmium only) sediments for both scenarios evaluated did not result in chronic or subchronic HIs which exceeded one.

Other Potential Risks

Per EPA recommendations, dermal contact was evaluated only for exposure to cadmium. However, other chemicals detected in site sediments may pose additional risk from dermal contact.

COCs which were detected but could not be evaluated due to a lack of toxicity data are cobalt, lead, phenanthrene, and delta-BHC. Cobalt and lead were detected in all sediment samples analyzed. The maximum concentration detected for cobalt is 17.7 mg/kg at location SED-18. The maximum concentration for lead, detected in sample SED-8-R, is 100 mg/kg which is below the EPA's target clean-up level for lead of 500 to 1000 mg/kg (EPA, 1989e).

Phenanthrene was detected in 6 out of 20 samples at a maximum concentration of .18 mg/kg. Delta-BHC was detected in 7 out of 20 samples with a maximum concentration of 5.4 ug/kg.

Leachate Soils

Carcinogenic Risk

The total carcinogenic risk estimate for the leachate soil scenario was below 10^{-6}. Therefore, carcinogenic risks from ingestion of leachate soils are assumed to be insignificant.

Noncarcinogenic Risk

Ingestion of and dermal contact with (cadmium only) leachate soils did not result in chronic HIs above one for the youth trespasser scenario.

Other Potential Risks

As explained previously, dermal contact with contaminants detected but not evaluated could pose an added risk to receptors. In addition, chemicals selected as COCs and detected in leachate soils but for which no appropriate toxicity data exist could also pose additional risks. Such chemicals detected in site leachate soils include phenanthrene, delta-BHC, cobalt, and lead. Cobalt was detected in both samples analyzed at a maximum concentration of 6.8 mg/kg. Phenanthrene and delta-BHC were each detected in 1 out of 2 samples and at the following respective maximum concentrations: 0.47 mg/kg, and 2.1 mg/kg. Lead was detected in both samples analyzed and at a maximum concentration of 110 mg/kg which is below the EPA's target clean-up level for lead.

Inhalation of volatilized organics or contaminated particulates from leachate soils (seeps) may also pose added risk to site receptors. However, due to the low concentrations detected in leachate soils, the intermittent nature of the seeps, and isolated locations of seeps, exposure via inhalation of leachate contaminants is expected to be insignificant.

6.3.3 Surface Water, Leachate Water

The risk associated with exposure to surface water and leachate water were not assessed quantitatively for the reasons discussed in Section 4.0. Risks from exposure to contaminants in surface water and leachate water are expected to be insignificant due to limited or negligible opportunities for exposure via ingestion, inhalation, or dermal contact.

6.3.4 Locally-Grown Produce

As explained in Section 4.0 of this report, risk to human receptors through the ingestion of locally-grown produce contaminated through root uptake of site ground water or deposition of contaminated dust on plant surfaces is not expected to be significant. Deposition of contaminated dust on plant surfaces is expected to be minimal because contaminant concentrations in site surface soils are low. Root uptake of contaminants through contact with site ground water is not expected to occur because (1) ground water at the site appears to have minimal horizontal movement in the upper aquifer layers (CRA, 1991a) and (2), water for irrigation is most likely obtained from the public water supply or directly from the Niagara River (Gwazdak, 1991; McMahon, 1991).

7.0 DISCUSSION OF UNCERTAINTIES

7.1 Introduction

The carcinogenic and noncarcinogenic risk estimates presented in this report are not intended to be calculations of absolute risk to individuals who reside adjacent to and/or frequent the NCR site. Uncertainties in underlying data prevent exact determination of risk to receptor populations. The goal of the risk assessment is to provide reasonable, conservative risk estimates to guide decisionmaking. By using standardized methodology guidelines, in particular, *Risk Assessment Guidance for Superfund* (EPA, 1989a), and standardized default exposure factors, provided in EPA (1991a), risk assessments for Superfund sites provide a basis for determining whether remediation needs to be considered.

The NCP (1990) establishes an acceptable medium-specific cancer risk range of 10^{-6} to 10^{-4}, indicating that a range of risk estimates is appropriate. Moreover, EPA guidance (EPA, 1989a) acknowledges that uncertainty in a risk assessment can cause differences in the numerical results of more than an order of magnitude. Therefore, it is important to document and discuss the types of uncertainties that may affect the risk estimates calculated in the previous section.

Risk is broadly a function of exposure and toxicity. Therefore, uncertainties in characterizing either of these lead to inaccuracy in risk estimates. Specific sources of uncertainty can be divided into two groups: methodological and site-specific. These types of uncertainties are described in the following subsections. Their effect on final risk estimates is discussed where possible.

7.2 General Methodological Uncertainties

7.2.1 Site Characterization

It is sometimes impossible to completely characterize heterogeneous environmental media from a statistical standpoint. Air contaminant concentrations vary greatly over space and time; soil contaminant concentrations may vary by orders of magnitude over intervals of an inch or less.

In some cases, only a few samples are available to evaluate a particular medium or source area. In these instances, EPA guidance (EPA, 1989a) calls for estimating exposure point concentrations based on the maximum concentrations detected. Although this is a health-protective approach, it probably overestimates true environmental risks. Maximum concentrations may not be representative of actual contamination and may actually be data "outliers."

To address these issues, EPA guidance (EPA, 1989a) calls for using the upper 95 percent confidence limit of the mean concentration when possible. With sufficient numbers of samples for statistical analyses, the upper confidence limit of the mean provides a conservative upper-bound concentration estimate. The potential problem of overestimating true exposure point concentrations is diminished as the number of samples evaluated increases. Increasing sample numbers generally reduces the upper confidence limit to below the maximum concentration detected.

7.2.2 Toxicological Information

Toxicity data used in human health risk assessments can be limited. Much of the data used to generate health criteria are derived from animal studies. Uncertainties result given that:

- Both endpoints of toxicity (effect or target organ) and the dose at which effects are observed are extrapolated from animals to humans;
- Results of short-term exposure studies are used to predict the effects of long-term exposures;
- Results of studies using high doses are used to predict effects from exposures to low doses usually expected at hazardous waste sites; and
- Effects exhibited by homogeneous populations of animals (or humans) are used to predict effects in heterogeneous populations with variable sensitivities (the young, elderly, or infirm).

In addition, thorough toxicity data are not available for all contaminants detected at many Superfund sites. Guidance suggests that individual compounds within groups of similar chemicals (e.g., PAHs) be grouped with respect to structure-activity relationships, toxicity characteristics, and chemical similarities.

EPA and other regulatory agencies attempt to account for these sources of uncertainty by including uncertainty factors in the determination of health criteria such as RfDs. In addition, the level of confidence in RfDs for noncarcinogenic effects and the weight of evidence for carcinogenic effects are specified for each contaminant. These qualifiers have been discussed in the dose-response section of this study.

7.2.3 Exposure Assumptions

Evaluating exposure to environmental contaminants requires a number of different inputs and assumptions. These include: the types of exposed populations, including their ages and health conditions; average lifespans; activity patterns such as time spent indoors versus outdoors and time spent at different locations; time spent working or residing in the area of the site; ingestion rates for soil and drinking water; skin surface area for dermal contact; and absorption rates via the skin and digestive tract.

Current EPA guidance for conducting risk assessments at Superfund sites recommends values to be used for many of these parameters. This serves to reduce unwarranted variability in exposure assumptions used to perform baseline risk assessments across different sites. Because values specified in guidance documents are often conservative, upper-bound figures, they would rarely lead to underestimating risks. However, using standard assumptions may mask site-specific variations.

Baseline risk assessments also estimate current and future exposure scenarios based on contaminant concentrations detected at the site during the RI. In general, no attenuation or degradation of contaminants over space or time is assumed. This also results in a conservative estimate of risk.

7.2.4 Dermal Contact Pathway

EPA (EPA Region II, 1992) has recommended that dermal contact risks should only be evaluated quantitatively for three types of contaminants: cadmium, PCBs, and dioxins. This guidance is based on the high level of uncertainty in data needed to evaluate this pathway (e.g., chemical-specific dermal absorption factors). However, none of the three listed contaminants were detected in site soils such that the dermal pathway was not evaluated quantitatively. This approach leads to an underestimation of total medium-specific risk. This underestimation is not expected to be significant.

7.2.5 Risk Characterization

Contaminant-specific risks are generally assumed to be additive. This oversimplifies the fact that some contaminants are thought to act synergistically ($1 + 1 > 2$) while others act antagonistically ($1 + 1 < 2$). The overall effect of these mechanisms on multi-contaminant, multi-media risk estimates is difficult to determine but the effects are usually assumed to balance.

7.3 Site-Specific Uncertainties

Potential site-specific sources of uncertainty for the NCR site include the following:

- Degree of characterization of contamination in all media;
- Process used to select COCs;
- Availability of toxicity data for certain COCs;
- Future land use and status of local public water supplies;
- Exposure parameter values; and
- Availability of sufficient background data.

The nature and extent of contamination at the NCR site was assessed for separate media and potential migration pathways. For each medium and contaminant either the maximum concentration or the upper 95 percent confidence limit was selected as the reasonable maximum exposure point concentration. The smaller of these two values was selected as a conservative, but realistic, approximation of exposure point concentration.

Certain media (e.g., leachate soils, subsurface soils) were characterized with only two or several samples. There is inherent variability in the environmental sampling results given spatial distribution of contamination and composition of the matrix sampled. Small numbers of samples may not completely characterize levels and numbers of contaminants actually present.

COCs were selected using a conservative methodology. Due to the lengthy list of contaminants identified at the site, a screening procedure based on concentration and toxicity was utilized to identify chemicals in a particular medium most likely to contribute significantly to calculated risks. This focused the risk assessment on the "most significant" chemicals. Because each chemical "screened" was weighted according to its toxicity, as well its detected concentration, it is unlikely that significant contributors to risk were excluded.

Certain COCs lacked health criteria and therefore had to be evaluated qualitatively. For example, the evidence for lead carcinogenicity is under review and no slope factor is available. Lead was evaluated in relation to the target soil clean-up levels recommended by OSWER and the MCL/action level for drinking water. Aluminum, cobalt, phenanthrene, and delta-BHC were also selected as COCs but could not be evaluated due to a lack of appropriate health criteria. Several detected carcinogenic PAHs evaluated for oral exposures were grouped together, as recommended by RAGS. Criteria for the more toxic species of PAH were used to evaluate other species. While this approach may be a source of uncertainty, it can only result in conservative risk estimates.

Future land use and future use of public drinking water supplies are difficult to define. For this risk assessment, local officials and planning boards were consulted for information on these issues. Risk scenarios are based on land use and water supply estimates that would result in "reasonable maximum" exposures. The ground water ingestion scenario may overestimate risk because it assumes that the maximum contaminant concentrations detected will reach private wells installed in place of the public supply. Future use of the site property was assumed to be equivalent to current use. More stringent security, however, may decrease exposures. This risk assessment assumed the site would be accessible to excavation workers, and youth trespassers.

Exposure parameters for the NCR risk assessment were obtained from EPA guidance or the peer-reviewed literature. Most of these assumptions are considered to be average or reasonable worst-case estimates that would not likely under-predict exposure. However, there are situations where parameters may produce underestimates. It is highly unlikely that the cumulative effect of all exposure parameter estimates will lead to underestimates of risk.

Finally, one of the purposes of this risk assessment is to characterize the incremental risk associated with the NCR site, i.e., risk over and above that attributable to anthropogenic or natural chemicals in the vicinity of the site. This determination relies partially on collection of "background samples" to estimate concentrations of chemicals (especially inorganics) in areas not influenced by the site. No background samples were collected at the NCR site; therefore, it is unknown if risk at the NCR site is attributable, to any extent, to naturally occurring chemicals.

7.4 Analysis of Alternative Exposure Parameters

Uncertainties in risk estimates can be evaluated by considering the full range of potential values (i.e., data distribution) for RME risk calculation inputs. As described in this risk assessment, these inputs include chemical concentrations, chemical toxicity values, and exposure parameters. While some of the inputs to the RME risk calculations are average values, others are selected to be more health protective. Input values for specific parameters (e.g., ingestion rate) may be taken from the upper end of statistical distributions of values (e.g., 90th or 95th percentile).

An estimate of "central tendency" risk can be obtained by substituting average or median (50th percentile) values for "upper bound" values. This is most useful for the exposure pathway which results in the highest estimated carcinogenic or noncarcinogenic risk; i.e., ground water ingestion. Table 7-1 lists the exposure parameters used to calculate the RME ground water ingestion risks (from Section 4) as well as central tendency exposure parameters obtained from EPA guidance. Note that for some parameters, there is insufficient information on the statistical distribution of values to justify using a different value in the central tendency estimate. For other parameters, the RME risk calculation already uses an average value.

TABLE 7-1. COMPARISON OF GROUND WATER EXPOSURE PARAMETERS FOR RME VERSUS "CENTRAL TENDENCY" RISK

VARIABLE	RME VALUE	VALUE BASIS	SOURCE	CENTRAL TENDENCY VALUE	VALUE BASIS	SOURCE
Body Weight (kg)						
Adult Resident	70	Average	RAGS Suppl.	70	Average	RAGS Suppl.
Duration of Exposure (years)						
Adult Resident	30	90th Percentile	RAGS Suppl.	9	50th Percentile	EFH
Exposure Frequency (days/year)	350	Average	RAGS Suppl.	350	Average	RAGS Suppl.
Ingestion Rate (l/day)						
Adult Resident	2	90th Percentile Residential	RAGS Suppl.	1.4	Average Residential	RAGS Suppl.
Averaging Time (days)						
noncarcinogenic	10950	30 x 365	N/A	3285	9 x 365	N/A
carcinogenic	25550	70 x 365	N/A	25550	70 x 365	N/A

RAGS Suppl.: U.S. EPA, *Risk Assessment Guidance for Superfund, Vol. I. Supplemental Guidance: Standard Default Exposure Factors*, Interim Final. Office of Emergency and Remedial Response. March 1991.
RAGS: U.S. EPA, *Risk Assessment Guidance for Superfund, Volume I*, EPA 540/1-89/002. Office of Emergency and Remedial Response. December, 1989.
EFH: *Exposure Factors Handbook*, EPA 600/8-89/043. Exposure Assessment Group, Office of Health and Environmental Assessment, 1989.

Table 7-1 indicates that values for three parameters decrease when estimating central tendency risk:

- exposure duration,
- ingestion rate, and
- noncarcinogenic averaging time (based on reduced exposure duration).

Applying these lower values to risk calculations (See Appendix E) results in the following changes in risk values:

- carcinogenic risk decreases by a factor of 4.8, and
- noncarcinogenic risk decreases by a factor of 1.4.

These central tendency risk values are compared with RME risk values in Table 7-2. The table includes chemical-specific carcinogenic risks and noncarcinogenic HQs for all "driver" COCs. The total pathway carcinogenic risk and noncarcinogenic HI for all COCs is also listed.

TABLE 7-2. COMPARISON OF RME RISK AND CENTRAL TENDENCY RISK FOR GROUND WATER INGESTION				
	Perimeter Ground Water		**Northern Landfill Cell Ground Water**	
	RME Risk[a]	**Central Tendency Risk[b]**	**RME Risk[a]**	**Central Tendency Risk[b]**
Total Carcinogenic Risk[c]	2×10^{-4}	4×10^{-5}	1×10^{-4}	2×10^{-5}
Driver Chemicals:				
Arsenic	1×10^{-4}	2×10^{-5}	5×10^{-5}	1×10^{-5}
Beryllium	5×10^{-5}	1×10^{-5}	5×10^{-5}	1×10^{-5}
Styrene	- -	- -	2×10^{-5}	4×10^{-6}
Total Noncarcinogenic HI[c]	$5 \times 10^{+0}$	$4 \times 10^{+0}$	$4 \times 10^{+0}$	$3 \times 10^{+0}$
Driver Chemicals:				
Antimony	$1 \times 10^{+0}$	7×10^{-1}	$3 \times 10^{+0}$	$2 \times 10^{+0}$
Aluminum	5×10^{-1}	4×10^{-1}	- -	
Arsenic	5×10^{-1}	4×10^{-1}	- -	
Iron	$2 \times 10^{+0}$	$1 \times 10^{+0}$	--	
Manganese	5×10^{-1}	4×10^{-1}	- -	

[a]From Appendix E.
[b]See text for derivation of central tendency risk estimates.
[c]Total risk from driver chemicals and other COCs.

8.0 ECOLOGICAL RISK ASSESSMENT

8.1 Introduction

8.1.1 Background

This ecological risk assessment describes the terrestrial and aquatic habitats and species that have been noted or are expected to be present at the NCR site and in the immediate vicinity. This assessment evaluates the potential risks associated with the exposure of biota to surface water/sediments, surface soils, and leachate contaminants detected during the RI. Quantitative measures to evaluate terrestrial ecological risks (e.g., wildlife inventories, biota sampling, bioassays, and modelling) were not within the scope of this risk assessment, as agreed with EPA.

8.1.2 General Methodology

This analysis is divided into five parts. The first part (Habitat and Species Characterization) describes ecological characteristics including habitats and species at the site and in the vicinity. Habitat descriptions and lists of wildlife species occurring at the site are based on information provided in the Remedial Investigation report (CRA, 1991). The second part (Hazard Identification) identifies potential media and contaminants of concern. Potential exposure pathways are identified (Exposure Assessment). Then, the Toxicity Assessment describes the responses of biota to contaminants of concern. Risk Characterization forms a quantitative and qualitative evaluation of potential risks to biota at and near the NCR site based on reported levels of contamination.

8.2 Habitat and Species Characterization

8.2.1 Onsite Habitats

Plant species typical of disturbed areas have become established on the surface of the landfill after the landfill was covered with approximately 20 inches of soil material when it was closed (CRA, 1991). A portion of the surface water present within the drainage swales flows north into an adjacent wetland and eventually into Black Creek although most of the surface water from the site flows south through a series of drainage swales into the Niagara River. A description of these habitats is provided below.

Onsite Terrestrial Habitats

Vegetation on the landfill surface includes a variety of grasses and herbaceous plants typical of successional areas, as well as scattered trees (white oak, white ash, honey locust, quaking aspen and box elder) and shrubs (staghorn sumac, black willow, multiflora rose and honeysuckle) based upon limited information provided in the RI (CRA, 1991). An access road covers a portion of the landfill. Additional characteristics of this habitat were not provided in the RI.

Onsite Aquatic Habitats

The only onsite aquatic habitats are the drainage swales. The drainage swale located along the eastern border of the site (near SW-11), reportedly contains water year-round (CRA, 1991). The remainder of the drainage swales are likely to have water present in response to recent precipitation events. Vegetation characteristic of wetlands are present within and adjacent to the drainage swales. These species include cat-tail, common reed, and purple loosestrife (CRA, 1991). Additional habitat variables of this aquatic cover type were not provided in the RI. Although the drainage swales do not

represent pristine habitats, they do provide habitat for aquatic species and may be used by terrestrial wildlife as a source of drinking water.

8.2.2 Offsite Habitats

A variety of aquatic and terrestrial habitats are present in the vicinity of the site. Immediately adjacent to the former refuse site are deciduous forested areas, a disturbed shrub/grass community, cultivated crop fields, and emergent/forested wetland areas. The Niagara River is present several hundred feet south of the site.

Offsite Terrestrial Habitats

An upland shrub/grassland habitat is present along the western boundary of the NCR site. This area is similar to the landfill cap area in that both are comprised of early successional vegetation species (primarily grasses/forbs with scattered shrubs and trees also present). Secondary growth deciduous forested areas are present adjacent to the northeast and southeastern areas of the NCR site. These forested areas contain white ash, red and silver maple, aspens, black cherry, and American elm in the tree overstory; dogwoods, honeysuckle, and staghorn sumac in the shrub understory; and a variety of herbaceous plant species in the ground layer vegetation (CRA, 1991). Many of the species reported to be present in this habitat cover type (Appendix D of RI) are frequently found in wetland areas.

A wet meadow area containing herbaceous vegetation and scattered shrubs is present along the eastern border of the landfill site between the two forested areas described above. Ash and dogwoods are present, however, this habitat is comprised primarily of herbaceous vegetation such as various grasses, milkweed, wild carrot, goldenrod, and purple loosestrife.

An emergent marsh and associated wooded swamp is present immediately north of the site. The marsh contains cat-tails and purple loosestrife while the forested swamp area contains willow, ash, dogwood, and arrow-wood. An intermittent stream also flows to the northwest through this area.

Cultivated fields are present primarily to the west of the site although a crop field is present east of the site as well. Corn and wheat are reported to be cultivated in the western fields while various vegetables are reported to be grown organically in the eastern field.

Offsite Aquatic Habitats

An intermittent stream is present within the wetland area immediately north of the NCR site. The northern landfill drainage swales flow northward and merge with this intermittent stream. This combined flow discharges into Black Creek. The southern landfill drainage swales flow south through an underground culvert into the Niagara River. No information concerning habitat conditions at Black Creek, the Niagara River, or the intermittent stream within the northern wetland were provided in the RI.

8.2.3 Regionally Significant Habitats

The Niagara River is classified by the U.S. Fish and Wildlife Service (USFWS) as a riverine, lower perennial, open-water wetland. According to the USFWS National Wetland Inventory map of the area, palustrine broad-leaved deciduous forested wetlands are also present immediately southeast and north of the NCR site. A scrub/shrub wetland is also present a short distance northeast of the NCR site. The New York State Department of Environmental Conservation (NYSDEC) has also designated the wetland area located north and adjacent to the NCR site.

Water quality of the Niagara River is classified by NYSDEC as Class A-Special because it is an international boundary water. Black Creek and the drainage swales present on the site are Class C surface waters. Class C water bodies are defined as being suitable for fish propagation and survival and also suitable for primary and secondary contact recreation, although other factors may limit the use for these purposes (NYSDEC, 1991).

Another significant habitat a tern nesting area, is listed in the NYSDEC. Natural Heritage Database. This area is located approximately two miles downstream of the site adjacent to the Niagara River. Rare and endangered species present in the vicinity of the site are discussed in Section 8.2.5.

8.2.4 Regional Species Profile

Information in this section concerning onsite species was obtained from Appendix D of the RI report (CRA, 1991), which provided a brief summary of plant species encountered on the site and wildlife observed or expected to be present onsite. Information concerning rare and endangered species was obtained from the NYSDEC Natural Heritage Database. Both plant and animal species are discussed below. A list of wildlife species noted at or expected to inhabit the vicinity of the site is also presented in Table 8-1.

Plants

The dominant vegetational species noted in the early successional habitats (landfill cap and upland/grassland areas) are grasses and herbaceous plants that are characteristic of disturbed areas. Representative species include red fescue, timothy, panic grass, common mullein, dandelion, clovers, milkweeds, and goldenrods. Scattered trees and shrubs as listed in Section 8.2.1 were also noted in these areas.

TABLE 8-1. WILDLIFE SPECIES OBSERVED OR EXPECTED TO BE PRESENT ON OR ADJACENT TO THE NIAGARA COUNTY REFUSE SITE, WHEATFIELD, NEW YORK (from CRA, 1991)

Common Name	Scientific Name
Mammals	
White-tailed Deer*	*Odocoileus virginianus*
Raccoon*	*Procyon lotor*
Red fox*	*Vulpes fulva*
Woodchuck*	*Marmota monax*
Eastern Cottontail*	*Sylvilagus floridanus*
Gray Squirrel	*Sciurus carolinensis*
Opossum	*Didelphis virginiana*
Muskrat	*Ondatra zibethica*
Striped skunk	*Mephitis mephitis*
Meadow vole*	*Microtus pennsylvanicus*
White-footed mouse*	*Peromyscus leucopus*
It is likely that several other mouse, mole and shrew species exist onsite.	
Birds	
Great blue heron	*Ardea herodias*
Wood duck	*Aix sponsa*
Mallard duck	*Anas platyrhynchos*
Turkey Vulture	*Cathartes aura*
Redtail hawk*	*Buteo jamaicensis*
Harrier*	*Circus cyaneus*
American Kestrel*	*Falco sparverius*
Ring-necked pheasant	*Phasianus colchicus*
Killdeer*	*Charadrius vociferus*
Herring Gull*	*Larus argentatus*
Ring-billed Gull	*Larus delawarensis*
Mourning Dove*	*Zenaidura macroura*
Northern Flicker*	*Colaptes auratus*
Eastern Kingbird*	*Tyrannus tyrannus*
Horned Lark	*Eremophila alpestris*
Blue Jay*	*Cyanocitta cristata*
American Crow*	*Corvus brachyrhynchos*

TABLE 8-1 (CONTINUED)

Common Name	Scientific Name
Birds (cont.)	
Black-capped chickadee*	*Parus atricapillus*
American Robin*	*Turdus migratorius*
Gray Catbird*	*Dumetella carolinensis*
Cedar waxwing*	*Bombycilla cedorum*
Yellow warbler*	*Dendroica petechia*
Common Yellowthroat*	*Geothlypis trichas*
Field Sparrow*	*Spizella pusilla*
Savannah Sparrow*	*Passerculus sandwichensis*
Song Sparrow	*Melospiza melodia*
Redwing Blackbird*	*Agelaius phoeniceus*
Brown-headed Cowbird*	*Molothrus ater*
Common Grackle	*Quiscalus quiscula*
Starling*	*Sturnus vulgaris*
Northern Cardinal*	*Cardinalis cardinalis*
American Goldfinch*	*Carduelistristis*
Purple Finch*	*Carpodacus purpureus*
Reptiles and Amphibians	
American Toad*	*Bufo americanus*
Northern Leopard Frog*	*Rana pipiens*
Common Garter Snake*	*Thanophis sirtalis*
Milk Snake	*Lampropeltis triangulum*

*Actually observed onsite or immediately adjacent to the site (CRA, 1991).

TRC

The wooded areas located in the northeastern and southeastern portions of the study area support various species of large trees, shrubs, and herbaceous plants. Trees recorded as present within these areas include oak, maples, willow, cherry, elm, and aspens. Herbaceous plants observed within the wet meadow area include purple loosestrife, goldenrods, cat-tails, and a variety of grasses. Plant species noted within and adjacent to the drainage swales included staghorn sumac, cat-tails, common reed, and purple loosestrife. Cat-tails, purple loosestrife, and several shrub/tree species were noted in the RI as occurring in the emergent marsh/forested wetland area present in the northern portion of the study area.

Birds

Common species observed at the site included killdeer, sparrows, starling, and brown-headed cowbird (Table 8-1). Many of the species observed onsite utilize wooded areas for nesting and open fields for foraging. The wooded areas adjacent to the site may support breeding species such as American crow and raptors that forage onsite. Although waterfowl were not observed and reported within the RI, several species of ducks may utilize the limited aquatic habitat provided by the drainage swales. It is interesting to note that the northern harrier, a state-listed threatened species, was reportedly observed onsite during mid-summer, 1990 (CRA, 1991). This species is not known to nest in the immediate vicinity of the NCR site based on information provided by the NYSDEC Natural Heritage Database.

Mammals

A variety of mammals are reported or expected to be present onsite and in the vicinity (Table 8-1). White-tailed deer, fox, raccoon, squirrel, and cottontail rabbit have been observed or are expected at and in the vicinity of the site. Smaller mammals, such as voles, shrews, and mice may also inhabit the cover types present. Muskrat may be present within the drainage swale areas.

Reptiles and Amphibians

According to the RI report (CRA, 1991), only four species of amphibians and reptiles were observed/expected to be present in the vicinity of the site (Table 8-1). However, additional species of snakes, salamanders and frogs likely inhabit the onsite fields, woodlands, and drainage swales.

Fish

The RI report did not indicate whether fish are present within the easternmost drainage swale that contains water throughout the year. However, a large diversity of fish species have been reported from the upper Niagara River in the general vicinity of the site. Species that have been reported as abundant in previous studies within the upper Niagara River include golden shiner, alewife, muskellunge, white sucker, goldfish, bluntnose minnow, and rock bass (Mooradian, 1992). No information was obtained regarding fisheries for Black Creek.

8.2.5 Species of Concern

Information on species of concern was obtained from the NYSDEC Natural Heritage Database, lists federal and state endangered, threatened, and rare species known to inhabit the vicinity of the NCR site. These species are listed in Table 8-2. If suitable habitats are present at or in the vicinity of the site, it is possible for these species to be present. No federally-listed species are reported to be present in the vicinity of the site.

According to the RI report (CRA, 1991), one threatened bird species, the northern harrier, has recently been observed at the site. Another state-listed threatened species, the common tern, is known to be present approximately two miles downstream of the site along the Niagara River. It is unlikely that this species utilizes the site because

TABLE 8-2. SPECIES OF SPECIAL CONCERN PRESENT IN THE VICINITY OF THE NIAGARA COUNTY REFUSE SITE, WHEATFIELD, NEW YORK (NYDEC, 1992)

Common Name	Scientific Name	Classification[1]	Habitat Utilized
Stiff-leaf Goldenrod	*Solidago rigida*	T	sandy woodlands
Elk Sedge	*Carex garberi*	R	wet sandy/marly shores
Fringed Gentian	*Gentianopsis procera*	E	moist meadows/low woods
Tall-tick Clover	*Desmodium glabellum*	T	dry woods and borders
Ninebark	*Physocarpus opulifolius var. intermedius*	R	river/stream banks
Common Tern	*Sterna hirundo*	T	river, sand bars
Small Skullcap	*Scutellaria parvula var. leonardii*	R	sandy soils (w/limestone)

[1]E: State Endangered Species
T: State Threatened Species
SC: State Special Concern Species
R: State Rare Species

suitable habitat is not present. Numerous plant species that are listed as either state endangered, state threatened, or rare, are known to inhabit the vicinity of the site. Stiff-leaf goldenrod has recently been documented to occur in close proximity (less than 0.5 miles) of the site.

8.3 Hazard Identification

8.3.1 Media of Concern

For the characterization of ecological risk, the primary media of concern at the NCR site are onsite surface waters and sediments. Surface soils and leachate seep on the site are also potential exposure media. Exposure of biota to subsurface soils, ground water, and airborne contaminants (through volatilization or fugitive dust emissions) is assumed to be negligible.

8.3.2 Contaminants of Concern

The selection of COCs for the ecological risk assessment is based on contaminant concentrations, frequency of detection, chemical persistence and bioaccumulation potential, and toxicity to aquatic/terrestrial biota. Analytical results for the ecological risk assessment are summarized in Appendix B. No background samples were available for any of the exposure pathways. A summary of the COCs selected by medium is presented in Table 8-3.

Surface Water

Surface water sample data were grouped into three areas: the northern wetland, the northern drainage ditches (flow into Black Creek), and the southern drainage ditches (flow into the Niagara River). Inorganics comprised the majority of the COCs for all three surface water groups although low levels of pesticides were also detected and

TABLE 8-3. NIAGARA COUNTY REFUSE SITE ECOLOGICAL RISK ASSESSMENT: CONTAMINANTS OF CONCERN

Contaminant	Northern Wetland		N. Drainage Ditches		S. Drainage Ditches		Leachate	Surface Soils
	Surface Water	Sediment	Surface Water	Sediment	Surface Water	Sediment		
VOLATILES								
Acetone							X	
2-Butanone (MEK)							X	
Chlorobenzene							X	
Ethylbenzene							X	
Total Xylenes							X	
BASE-NEUTRAL/ACID EXTRACTABLES								
Phenol			X					
2-Methlyphenol							X	
4-Methylphenol							X	
2-4-Dimethylphenol							X	
Benzoic Acid							X	
Bis(2-ethylhexyl)phthalate		X		X	X	X		X
PAHs								
Benzo(a)anthracene		X		X		X		
Benzo(b)fluoranthene		X		X		X		
Benzo(k)fluoranthene		X				X		
Benzo(a)pyrene		X				X		
Benzo(g,h,i)perylene						X		
Chrysene		X		X		X		

TABLE 8-3 (CONTINUED)

Contaminant	Northern Wetland		N. Drainage Ditches		S. Drainage Ditches		Leachate	Surface Soils
	Surface Water	Sediment	Surface Water	Sediment	Surface Water	Sediment		
Fluoranthene		X		X		X		
Phenanthrene		X		X		X		
Pyrene		X		X		X		
PESTICIDES								
Delta-BHC			X	X	X	X	X	X
Gamma-BHC	X					X	X	
Dieldrin						X		
Heptachlor							X	
Heptachlor Epoxide					X	X		
Aldrin						X	X	
Endrin						X		
Endosulfan II				X		X		
4,4-DDE						X		
4,4-DDD						X	X	
4,4-DDT		X	X	X		X	X	
INORGANICS								
Aluminum	X	X	X	X	X	X	X	X
Arsenic	X	X		X	X	X	X	X
Barium	X	X	X	X	X	X	X	X
Beryllium		X		X	X	X	X	X

TABLE 8-3 (CONTINUED)

Contaminant	Northern Wetland		N. Drainage Ditches		S. Drainage Ditches		Leachate	Surface Soils
	Surface Water	Sediment	Surface Water	Sediment	Surface Water	Sediment		
Cadmium					X	X	X	
Chromium, Total		X		X	X	X	X	X
Cobalt		X		X	X	X	X	X
Copper		X		X	X	X	X	X
Cyanide			X		X			
Iron	X	X	X	X	X	X	X	X
Lead	X	X	X	X	X	X	X	X
Manganese	X	X	X	X	X	X	X	X
Mercury		X		X		X	X	
Nickel		X		X	X	X	X	X
Thallium					X			
Vanadium		X		X	X	X	X	X
Zinc	X	X		X	X	X	X	X

These compounds were retained as COCs. The southern drainage swale water samples contained a greater frequency of both inorganics and organics.

Leachate

The leachate water sample results included VOCs, BNAs, pesticides, and inorganics. VOCs and BNAs detected in high concentrations (generally greater than 500 µg/l) were retained as COCs. PAHs were detected at low concentrations (less than 200 µg/l) and were not retained as COCs. All pesticides detected in the samples were retained as COCs. All inorganics except essential nutrients were retained as COCs.

Sediments

Sediment samples were grouped into the same three areas as described above for the surface water samples. Contaminants retained as COCs for the northern wetland sediment sample group included PAHs, bis(2-ethylhexyl)phthalate, 4,4-DDT, and 14 inorganics. The northern drainage swale sample group was similar except that several more pesticides and inorganics were detected and retained as COCs. The southern drainage swale sediment sample data group contained the highest number of detected pesticides and inorganics. VOCs were detected at low concentrations (less than 100 µg/l) in all three sediment sample data groups and were not retained as COCs. All BNAs and pesticides detected were retained as COCs except for chemicals detected in at a low frequency (one sample only) in the southern drainage swale sample group.

Surface Soils

VOCs were either detected infrequently (one sample) or at low concentrations (less than 35 µg/kg) and were not retained as COCs. Bis(2-ethylhexyl)phthalate and delta-BHC were the only BNAs detected and both were retained as COCs. All inorganics detected except essential nutrients were retained as COCs because of the absence of inorganic background levels.

8.4 Exposure Assessment

Possible exposure pathways for ecological receptors on or adjacent to the NCR site are:

- ingestion of and/or dermal contact with surface soil contaminants;
- ingestion of contaminated biota in the food chain; and
- contact with and/or ingestion of leachate, surface water, and sediment contaminants.

Exposures to leachate, surface water, and sediment contaminants (geometric mean and maximum concentrations) are evaluated quantitatively using risk indices. Surface soil contamination and biota effects through food chain transport are evaluated qualitatively.

8.5 Toxicity Assessment

Available criteria, guidelines, and toxicity information for each medium of concern were reviewed. NYSDEC Water Quality Regulations for surface waters, EPA Ambient Water Quality Criteria (AWQC), New York State Sediment Criteria, EPA Interim Sediment Criteria, and National Oceanic and Atmospheric Administration (NOAA) sediment guidelines were utilized for comparison to site surface water and sediment contaminant concentrations, as appropriate. Surface water contaminants of concern for which no criteria exist were evaluated by searching the AQUIRE database (1991). Criteria for soil contaminants are not available, however, surface soil COCs were assessed by comparing concentrations to soil cleanup guidelines developed by the Ontario Ministry of the Environment (1991).

8.5.1 Surface Water

Water criteria presented in the New York State Water Quality Regulations (NYSDEC, 1991) for the protection of aquatic organisms were utilized for assessing adverse impact of surface water COC concentrations present in the site drainage swales, northern wetland stream, and leachate samples. Aquatic life criteria (acute and chronic) available from the AWQC were also utilized. If New York State regulations and AWQC documents did not contain criteria for a particular COC, then a review of the literature through the AQUIRE database was conducted to obtain a reference toxicity value. This value was based on results of LC_{50} toxicity tests and was then multiplied by an application factor. New York State Water Quality Regulations identify application factors of 0.01 for LC_{50} values less than or equal to 1.0 mg/l, 0.05 where test results are for sensitive species, and 0.03 for LC_{50} results conducted on species of lower sensitivity. These adjusted literature values are utilized as chronic toxicity values. Acute toxicity values were obtained by applying an application factor of 0.1 to LC_{50} toxicity test results (EPA, 1986). Resulting surface water quality criteria/guidance values are presented in Table 8-4 for each COC identified in leachate or surface water samples.

8.5.2 Sediment

Sediment criteria developed as guidance by NYSDEC (1989), EPA (1988), and NOAA (1991) were utilized for assessing sediment contaminant levels where adverse impacts may potentially occur. The total organic carbon (TOC) content of sediment samples is utilized in determining NYSDEC and EPA sediment criteria/guidance values. However, TOC was not analyzed for sediment samples collected on the site. Therefore, an assumed TOC of 1% was selected for the drainage swale and intermittent wetland stream sediments present on the site. This value is expected to be conservative and will result in lower sediment criteria for organic COCs. Available sediment criteria for each COC are presented in Table 8-5.

TABLE 8-4. SURFACE WATER QUALITY CRITERIA FOR CONTAMINANTS OF CONCERN, NIAGARA COUNTY REFUSE SITE, WHEATFIELD, NEW YORK

Contaminant	Criteria (µg/l) Acute	Chronic	Reference
VOLATILES			
Acetone	610,000.0	305,000.0	Mejewski et al., 1978
2-Butanone	52,000.0	15,600.0	LeBlanc, 1980
Chlorobenzene	250.0	5.0	NYSDEC, 1991
Ethylbenzene	32,000.0	--	EPA, 1991
Xylenes, Total	1,690.0	507.0	Brenniman et al., 1976
SEMIVOLATILES			
Phenol	10,200	5.0	EPA, 1991 & NYSDEC, 1991
2-Methylphenol	500.0	250.0	Parkhurst et al., 1979
4-Methylphenol	140.0	70.0	Parkhurst et al., 1979
2,4-Dimethylphenol	2,120.0	--	EPA, 1991
Benzoic Acid	46,000.0	13,800.0	Juhnke & Luedman, 1978
Bis(2-ethylhexyl)phthalate	940.0	0.6	EPA, 1991 & NYSDEC, 1991
PESTICIDES			
Delta-BHC	100.0	--	EPA, 1991
Gamma-BHC	100.0	--	EPA, 1991
Heptachlor	0.52	0.001	NYSDEC, 1991
Heptachlor Epoxide	0.52	0.001	NYSDEC, 1991
Aldrin	3.0	--	EPA, 1991
4,4-DDD	--	0.001	NYSDEC, 1991
4,4-DDT	1.1	0.001	EPA, 1991 & NYSDEC, 1991
INORGANICS			
Aluminum	750.0	100.0	EPA, 1991 & NYSDEC, 1991
Arsenic	360.0	190.0	NYSDEC, 1991
Barium	50,000.0	--	EPA, 1986
Beryllium	130.0	3.0	EPA, 1991
Cadmium	3.9	1.1	NYSDEC, 1991
Chromium, Hexavalent	16.0	11.0	NYSDEC, 1991
Cobalt	--	5.0	NYSDEC, 1991

TABLE 8-4 (CONTINUED)

Contaminant	Criteria (µg/l) Acute	Chronic	Reference
Copper	18.0	11.8	NYSDEC, 1991
Cyanide	22.0	5.2	NYSDEC, 1991
Iron	--	1,000.0	EPA, 1991
Lead	83.0	3.2	NYSDEC, 1991
Manganese	1,000,000.0	--	McKee and Wolf, 1963
Mercury	2.4	0.012	EPA, 1991
Nickel	1,844.0	95.6	NYSDEC, 1991
Thallium	1,400.0	8.0	NYSDEC, 1991
Vanadium	--	14.0	NYSDEC, 1991
Zinc	321.3	30.0	NYSDEC, 1991

-- No criteria available

TABLE 8-5. SEDIMENT QUALITY CRITERIA/GUIDELINES FOR CONTAMINANTS OF CONCERN, NIAGARA COUNTY REFUSE SITE, WHEATFIELD, NY

Contaminant	Criteria/Guidelines (mg/kg) Aquatic Toxicity	Wildlife Residue	Reference
SEMIVOLATILES			
Bis(2-ethylhexyl)phthalate	1.2	--	NYSDEC, 1989
PAHs			
Benzo(a)anthracene	13.2	--	EPA, 1988
Benzo(b)fluoranthene	--	--	--
Benzo(k)fluoranthene	--	--	--
Benzo(a)pyrene	10.6	--	EPA, 1988
Benzo(g,h,i)perylene	--	--	--
Chrysene	0.4	--	NOAA, 1990
Fluoranthene	18.8	--	EPA, 1988
Phenanthrene	1.4	--	EPA, 1988
Pyrene	13.1	--	EPA, 1988
PESTICIDES			
Delta-BHC	--	--	--
Gamma-BHC	0.00157	--	EPA, 1988
Dieldrin	0.199	0.0013	EPA, 1988
Heptachlor Epoxide	0.0003	0.001	NYSDEC, 1989
Aldrin	0.084	0.0077	NYSDEC, 1989
Endrin	0.0104	0.0005328	EPA, 1988
Endosulfan II	0.0003	--	NYSDEC, 1989
4,4-DDE	0.5	--	NYSDEC, 1989
4,4-DDD	0.5	--	NYSDEC, 1989
4,4-DDT	0.5	0.0828	NYSDEC, 1989
INORGANICS			
Aluminum	--	--	
Arsenic	5.0	--	NYSDEC, 1989
Barium	--	--	--
Beryllium	--	--	--
Cadmium	0.8	--	NYSDEC, 1989
Chromium, Total	26.0	--	NYSDEC, 1989
Cobalt	--	--	--
Copper	19.0	--	NYSDEC, 1989
Iron	24,000.0	--	NYSDEC, 1989
Lead	27.0	--	NYSDEC, 1989
Manganese	428.0	--	NYSDEC, 1989
Mercury	0.11	--	NYSDEC, 1989
Nickel	22.0	--	NYSDEC, 1989
Vanadium	--	--	--
Zinc	85.0	--	NYSDEC, 1989

8.5.3 Surface Soils

Criteria for assessing toxicity of surficial soil contaminants to terrestrial wildlife are unavailable. Neither Federal or state regulatory agencies have developed toxicity values for exposure to contaminants in surface soil by biota. Soil cleanup guidelines have been developed for decommissioning industrial lands, however, by the Ontario Ministry of the Environment (1991). These guidelines are based primarily on phytotoxicity and were developed for future residential, parkland, or agricultural use. These values (for inorganics only) were used as benchmarks for contaminant levels expected to show ecological effects and are presented in Table 8-6.

8.6 Risk Characterization

Potential risks to ecological receptors from contaminants present in surface water (including leachate), sediments, and surficial soils were assessed by comparing medium-specific contaminant concentrations with criteria/guidelines. This comparison (expressed as a risk index) was calculated for each contaminant of concern as follows:

$$RI = Conc_{med}/Crit_{med}$$

where:

RI =	Risk Index for a particular COC;
$Conc_{med}$ =	Concentration of COC in a medium; and
$Crit_{med}$ =	Criteria/guideline for COC in the same medium.

If the calculated Risk Index is greater than one, it indicates that biota may potentially be at risk to an adverse effect from that contaminant within that exposure pathway. A total risk index was also calculated for each exposure medium by summing all the risk indices for COCs within each medium. This assumes that potential risk from various contaminants is additive and does not consider possible synergistic or antagonistic effects among the contaminants evaluated. If the total risk index is greater than one,

TABLE 8-6. SOIL CRITERIA GUIDELINES FOR SURFACE SOIL CONTAMINANTS OF CONCERN, NIAGARA COUNTY REFUSE SITE, WHEATFIELD, NEW YORK

Contaminant	Cleanup Criteria (mg/kg)[1]
SEMIVOLATILES	
Bis(2-ethylhexyl)phthalate	--
PESTICIDES	
Delta-BHC	--
INORGANICS	
Aluminum	--
Arsenic	20.0
Barium	750.0
Beryllium	4.0
Chromium, Total	750.0
Cobalt	40.0
Copper	150.0[2]
Iron	--
Lead	--
Manganese	--
Nickel	150.0
Vanadium	200.0
Zinc	600.0

[1]Coarse-textured soils are assumed to be present on the site.
[2]Based on health of grazing animals.

this indicates that exposure to all COCs within that medium may potentially pose a risk to organisms.

It is important to note that risk indices provide only a general characterization of potential impacts to the local biota at the individual level. Risk indices do not indicate whether population-level impacts are occurring. If effects are shown to be insignificant at the individual level, it is likely that impacts are also insignificant at the population level. However, risks present at the individual level may or may not be significant at the population level.

8.6.1 Surface Water/Leachate

Potential hazards to aquatic organisms present within the surface waters of the northern wetland stream, northern and southern drainage swales, and leachate seeps were assessed by comparing mean and maximum concentrations of contaminants with AWQC or toxicity effect levels (when AWQC were not available). Results of this evaluation are presented in Table 8-7. Values listed as maximum concentrations represent the maximum concentration detected, or the upper 95% confidence limit of the mean, whichever is lower.

Northern wetland risk indices for mean and maximum concentrations of all contaminants detected within the northern wetland stream except aluminum do not exceed one for acute criteria. This indicates that immediate risks to aquatic biota are low. Mean and maximum concentrations of aluminum, iron and lead are above chronic AWQC. The maximum detected level of zinc is also above the chronic AWQC. The AWQC for iron (1000 µg/l) is based on aesthetic values rather than toxic levels, therefore, a risk index greater than one does not necessarily result in a potential adverse impact to aquatic life from this metal. Maximum and average concentrations for lead and zinc are on the same order of magnitude as the chronic AWQC. Therefore, the potential for adverse effects from these metals may be less than the potential risk of adverse chronic effects due to elevated aluminum levels

TABLE 8-7. SURFACE WATER ECOLOGICAL RISK ASSESSMENT SUMMARY.

CONTAMINANT OF CONCERN	SURFACE WATER CONCENTRATION (ug/l)		WATER QUALITY CRITERIA[1] (ug/l)		RISK INDICES[2] FOR ACUTE CRITERIA		RISK INDICES[2] FOR CHRONIC CRITERIA	
	MEAN	MAXIMUM	ACUTE	CHRONIC	MEAN	MAXIMUM	MEAN	MAXIMUM
NORTHERN WETLAND STREAM								
Gamma-BHC	4.90E-03	5.10E-03	1.00E+02	-	4.90E-05	5.10E-05	-	-
Aluminium	1.23E+03	2.57E+03	7.50E+02	1.00E+02	1.64E+00	3.43E+00	1.23E+01	2.57E+01
Arsenic	3.07E+00	4.70E+00	3.60E+02	1.90E+02	8.53E-03	1.31E-02	1.62E-02	2.47E-02
Barium	1.14E+02	1.47E+02	5.00E+04	-	2.29E-03	2.94E-03	-	-
Iron	1.60E+03	3.40E+03	-	1.00E+03	-	-	1.60E+00	3.40E+00
Lead	1.11E+01	2.00E+01	8.30E+01	3.20E+00	1.33E-01	2.41E-01	3.45E+00	6.25E+00
Manganese	2.49E+02	7.83E+02	1.00E+06	-	2.49E-04	7.83E-04	-	-
Zinc	2.76E+01	7.60E+01	3.21E+02	3.00E+01	8.58E-02	2.37E-01	9.19E-01	2.53E+00
			TOTAL RISK INDEX		1.87E+00	3.92E+00	1.83E+01	3.79E+01
NORTHERN DRAINAGE SWALES								
Phenol	7.42E+00	1.10E+01	1.02E+04	5.00E+00	7.27E-04	1.08E-03	1.48E+00	2.20E+00
Delta-BHC	1.01E-02	2.10E-02	1.00E+02	-	1.01E-04	2.10E-04	-	-
4,4-DDT	2.00E-02	4.00E-02	1.10E+00	1.00E-03	1.82E-02	3.64E-02	2.00E+01	4.00E+01
Aluminium	7.57E+02	8.61E+02	7.50E+02	1.00E+02	1.01E+00	1.15E+00	7.57E+00	8.61E+00
Barium	7.96E+01	9.60E+01	5.00E+04	-	1.59E-03	1.92E-03	-	-
Cyanide	9.82E+00	1.93E+01	2.20E+01	5.20E+00	4.46E-01	8.77E-01	1.89E+00	3.71E+00
Iron	8.70E+02	1.55E+03	-	1.00E+03	-	-	8.70E-01	1.55E+00
Lead	9.33E+00	9.90E+00	8.30E+01	3.20E+00	1.12E-01	1.19E-01	2.92E+00	3.09E+00
Manganese	3.60E+01	4.80E+01	1.00E+06	-	3.60E-05	4.80E-05	-	-
			TOTAL RISK INDEX		1.59E+00	2.18E+00	3.47E+01	5.92E+01
SOUTHERN DRAINAGE SWALES								
Bis (2-ethylhexyl) phthalate	1.25E+01	1.00E+03	9.40E+02	6.00E-01	1.33E-02	1.06E+00	2.08E+01	1.67E+03
Delta-BHC	1.12E-02	1.40E-02	1.00E+02	-	1.12E-04	1.40E-04	-	-
Heptachlor epoxide	1.12E-02	1.40E-02	5.20E-01	1.00E-03	2.15E-02	2.69E-02	1.12E+01	1.40E+01
Aluminium	2.06E+03	2.52E+04	7.50E+02	1.00E+02	2.75E+00	3.36E+01	2.06E+01	2.52E+02
Arsenic	4.50E+00	3.06E+01	3.60E+02	1.90E+02	1.25E-02	8.50E-02	2.37E-02	1.61E-01

TABLE 8-7. SURFACE WATER ECOLOGICAL RISK ASSESSMENT SUMMARY. (continued)

CONTAMINANT OF CONCERN	SURFACE WATER CONCENTRATION (ug/l)		WATER QUALITY CRITERIA[1] (ug/l)		RISK INDICES[2] FOR ACUTE CRITERIA		RISK INDICES[2] FOR CHRONIC CRITERIA	
	MEAN	MAXIMUM	ACUTE	CHRONIC	MEAN	MAXIMUM	MEAN	MAXIMUM
Barium	1.59E+02	4.56E+02	5.00E+04	-	3.18E-03	9.12E-03	-	-
Beryllium	1.01E+00	1.62E+00	1.30E+02	3.00E+00	7.77E-03	1.25E-02	3.37E-01	5.40E-01
Cadmium	2.72E+00	3.89E+00	3.90E+00	1.10E+00	6.97E-01	9.97E-01	2.47E+00	3.54E+00
Chromium, Hexavalent	1.08E+01	3.80E+01	1.60E+01	1.10E+01	6.76E-01	2.38E+00	9.83E-01	3.45E+00
Cobalt	7.18E+00	2.45E+01	-	5.00E+00	-	-	1.44E+00	4.91E+00
Copper	2.30E+01	9.40E+01	1.80E+01	1.18E+01	1.28E+00	5.22E+00	1.95E+00	7.97E+00
Cyanide	1.01E+01	4.06E+01	2.20E+01	5.20E+00	4.60E-01	1.85E+00	1.95E+00	7.81E+00
Iron	4.95E+03	3.80E+04	-	1.00E+03	-	-	4.95E+00	3.80E+01
Lead	4.17E+01	3.52E+02	8.30E+01	3.20E+00	5.03E-01	4.24E+00	1.30E+01	1.10E+02
Manganese	1.55E+02	1.69E+03	1.00E+06		1.55E-04	1.69E-03	-	-
Nickel	2.32E+01	6.30E+01	1.84E+03	9.56E+01	1.26E-02	3.42E-02	2.42E-01	6.59E-01
Thallium	2.71E+00	3.30E+00	1.40E+03	8.00E+00	1.94E-03	2.36E-03	3.39E-01	4.12E-01
Vanadium	1.28E+01	3.65E+01	-	1.40E+01	-	-	9.12E-01	2.61E+00
Zinc	7.98E+01	2.36E+03	3.21E+02	3.00E+01	2.48E-01	7.35E+00	2.66E+00	7.87E+01
			TOTAL RISK INDEX		6.68E+00	5.69E+01	8.39E+01	2.19E+03
LEACHATE								
Acetone	4.19E+01	2.20E+03	6.10E+05	3.05E+05	6.87E-05	3.61E-03	1.37E-04	7.21E-03
2-Butanone	7.71E+01	1.40E+03	5.20E+04	1.56E+04	1.48E-03	2.69E-02	4.94E-03	8.97E-02
Chlorobenzene	1.26E+01	5.60E+01	2.50E+02	5.00E+00	5.04E-02	2.24E-01	2.52E+00	1.12E+01
Ethylbenzene	2.23E+01	6.80E+02	3.20E+04	-	6.97E-04	2.13E-02	-	-
Xylenes, Total	5.17E+01	1.40E+03	1.69E+03	5.07E+02	3.06E-02	8.28E-01	1.02E-01	2.76E+00
2-Methylphenol	1.55E+02	9.60E+02	5.00E+02	2.50E+02	3.11E-01	1.92E+00	6.21E-01	3.84E+00
4-Methylphenol	3.40E+02	3.75E+03	1.40E+02	7.00E+01	2.43E+00	2.68E+01	4.86E+00	5.36E+01
2,4-Dimethylphenol	6.12E+01	9.80E+02	2.12E+03	-	2.89E-02	4.62E-01	-	-
Benzoic Acid	1.47E+02	1.20E+04	4.60E+04	1.38E+04	3.20E-03	2.61E-01	1.07E-02	8.70E-01
Delta-BHC	2.75E-02	1.80E-01	1.00E+02	-	2.75E-04	1.80E-03	-	-
Gamma-BHC	2.36E-02	5.20E-02	1.00E+02	-	2.36E-04	5.20E-04	-	-
Heptachlor	2.24E-02	1.10E+00	5.20E-01	1.00E-03	4.32E-02	2.12E+00	2.24E+01	1.10E+03

TABLE 8-7. SURFACE WATER ECOLOGICAL RISK ASSESSMENT SUMMARY. (continued)

CONTAMINANT OF CONCERN	SURFACE WATER CONCENTRATION (ug/l)		WATER QUALITY CRITERIA[1] (ug/l)		RISK INDICES[2] FOR ACUTE CRITERIA		RISK INDICES[2] FOR CHRONIC CRITERIA	
	MEAN	MAXIMUM	ACUTE	CHRONIC	MEAN	MAXIMUM	MEAN	MAXIMUM
Aldrin	1.86E-02	8.20E-02	3.00E+00	-	6.19E-03	2.73E-02	-	-
4,4-DDD	2.19E-02	1.50E-02 *	-	1.00E-03	-	-	2.19E+01	1.50E+01
4,4-DDT	7.14E-02	1.10E-01	1.10E+00	1.00E-03	6.49E-02	1.00E-01	7.14E+01	1.10E+02
Aluminium	7.05E+03	3.25E+05	7.50E+02	1.00E+02	9.40E+00	4.33E+02	7.05E+01	3.25E+03
Arsenic	2.00E+01	4.16E+01	3.60E+02	1.90E+02	5.56E-02	1.15E-01	1.05E-01	2.19E-01
Barium	7.56E+02	7.61E+03	5.00E+04	-	1.51E-02	1.52E-01	-	-
Beryllium	1.35E+00	2.20E+00	1.30E+02	3.00E+00	1.04E-02	1.69E-02	4.50E-01	7.33E-01
Cadmium	4.37E+00	9.00E+00	3.90E+00	1.10E+00	1.12E+00	2.31E+00	3.97E+00	8.18E+00
Chromium, Hexavalent	5.22E+01	1.05E+02	1.60E+01	1.10E+01	3.26E+00	6.56E+00	4.74E+00	9.55E+00
Cobalt	1.70E+01	9.30E+01	-	5.00E+00	-	-	3.41E+00	1.86E+01
Copper	3.23E+01	9.90E+01	1.80E+01	1.18E+01	1.79E+00	5.50E+00	2.73E+00	8.39E+00
Iron	4.38E+04	3.90E+05	-	1.00E+03	-	-	4.38E+01	3.90E+02
Lead	1.16E+02	1.01E+03	8.30E+01	3.20E+00	1.39E+00	1.22E+01	3.62E+01	3.16E+02
Manganese	3.93E+02	2.96E+03	-	1.00E+06	-	-	3.93E-04	2.96E-03
Mercury	1.50E-01	4.40E-01	2.40E+00	1.20E-02	6.25E-02	1.83E-01	1.25E+01	3.67E+01
Nickel	9.07E+01	1.47E+02	1.84E+03	9.56E+01	4.92E-02	7.97E-02	9.49E-01	1.54E+00
Vanadium	1.79E+01	3.19E+01	-	1.40E+01	-	-	1.28E+00	2.28E+00
Zinc	5.65E+02	1.39E+03	3.21E+02	3.00E+01	1.76E+00	4.33E+00	1.88E+01	4.63E+01
			TOTAL RISK INDEX		2.19E+01	4.98E+02	3.23E+02	5.39E+03

* Maximum detected concentration below detection level. One-half detection limit was utilized for non-detects, therefore mean conc. > maximum conc.

[1] Water Quality Criteria from Table 8-4.

[2] Risk Indices = Surface Water Concentration (mean or maximum) divided by Water Quality Criteria.

(Risk Index = 25.7). The mean chronic total risk index is 18.3. Inorganics with mean chronic risk indices greater than 1.0 are aluminum (67% of total index), lead (19% of total) and iron (9% of total). The maximum total risk index for adverse chronic effects is 37.9; aluminum comprises 68%, lead 16%, iron 9%, and zinc 7%.

Northern Drainage Swales

Risk indices based on acute water quality criteria are all below 1.0 except for aluminum which was detected at concentrations approximately equal to the acute criteria (risk indices of 1.0 and 1.2 for mean and maximum concentrations, respectively. Therefore, acute adverse effects are not expected to occur in aquatic biota residing in the surface waters of these drainage swales. All six of the COCs which have chronic criteria, however, have maximum concentrations above the chronic criteria. Average concentrations of phenol, 4,4-DDT, aluminum, cyanide, and lead also resulted in risk indices greater than one. Although, the maximum phenol concentration detected is well below EPA's chronic Lowest Observed Effect Level of 2,560 µg/l (EPA, 1991), the mean chronic total risk index is well above 1.0 (34.7) with 4,4-DDT comprising 58%, aluminum 22%, lead 8%, cyanide 5%, and phenol 4% of the total risk index. 4,4-DDT is the only contaminant that resulted in a risk index greater than 10.0 (20.0 and 40.0 for mean and maximum concentrations, respectively) indicating a more likely potential for adverse chronic effects. However, this contaminant was only detected in one of the two samples. The remaining COCs have risk indices on the same order of magnitude as their respective criteria.

Southern Drainage Swales

Acute risk indices based on mean concentrations for surface water COCs are all below 1.0 except indices for aluminum and copper. Risk indices for these contaminants are of the same order of magnitude as their respective acute criteria. Maximum concentrations of bis(2-ethylhexyl)phthalate, aluminum, chromium, copper, cyanide,

lead and zinc are all above acute criteria although only aluminum has a risk index an order of magnitude above its acute criterion. Maximum acute risk index for aluminum is 33.6 (59% of total risk index).

The chronic total risk indices for mean and maximum COC levels are 83.9 and 2190.0. Chronic adverse impacts are expected to be presently occurring as a result of maximum detected concentration levels. COCs that produce chronic risk indices greater than 10.0 for mean concentrations are bis(2-ethylhexyl)phthalate, heptachlor epoxide, aluminum, and lead. High risk indices for maximum COC concentrations were calculated for bis(2-ethylhexyl)phthalate (1670.0), heptachlor epoxide (14.0), aluminum (252.0), iron (38.0), lead (10.0), and zinc (78.7). Six other metals also have maximum concentrations above chronic criteria, although of the same order of magnitude as their respective criteria.

Leachate

Acute total risk indices for mean and maximum leachate concentrations are 21.9 and 498, respectively. Maximum concentrations of 4-methylphenol (26.8), aluminum (433.0), and lead (12.2), are an order of magnitude above their respective acute criteria/effects level indicating that acute adverse effects may presently occur for aquatic biota present within these maximum concentration areas. COCs with mean concentrations resulting in an acute risk index above 1.0 are 4-methylphenol, aluminum, cadmium, chromium, copper, lead, and zinc. However, the concentrations of these COCs are of the same order of magnitude as their acute criteria (i.e., risk index is less than 10.0).

Total chronic risk indices for mean and maximum COC concentrations are 323.0 and 5390.0, respectively. COCs for which concentrations exceed criteria by an order of magnitude are heptachlor, 4,4-DDD, 4,4-DDT, aluminum, iron, lead, mercury, and zinc. Seven other COCs also have mean concentrations greater than their chronic

criteria. Eighteen COCs have maximum concentrations greater than chronic criteria. Of these, the following COCs had risk indices an order of magnitude above their chronic criteria/effect levels: chlorobenzene (11.2), 4-methylphenol, (53.6), heptachlor (1100.0), 4,4-DDD (15.0), 4,4-DDT (110.0), aluminum (3250.0), cobalt (18.6), iron (390.0), lead (316.0), mercury (36.7), and zinc (46.3). However, heptachlor, 4,4-DDD, and mercury were detected infrequently (one or two of eight samples) while chlorobenzene, 4,4-DDT and cobalt were detected in three or four samples. Iron levels above chronic criteria may not result in adverse effects. The chronic criteria for iron is based on aesthetics values rather than toxicity. Aluminum, lead, and zinc were detected in all eight samples and the lowest concentration for each of these metals was greater than the respective chronic criteria.

8.6.2 Sediments

Risks to aquatic organisms inhabiting sediments within the northern wetland stream, northern drainage swales, and southern drainage swales were also evaluated by comparing mean and maximum COC concentrations to sediment criteria/guidelines. Results are presented in Table 8-8. Maximum concentrations listed represent either the maximum concentration detected, or the upper 95 percent confidence limit, whichever is lower.

Northern Wetland

Mean and maximum concentrations of arsenic, chromium, copper, iron, lead, mercury, nickel, and zinc exceed sediment criteria guidelines. The maximum concentration of manganese also exceeds sediment criteria. Maximum detected arsenic, chromium, and copper levels are below tolerance limits (NYSDEC, 1989) and effect levels observed to impact the lower tenth percentile of test organisms studied (NOAA, 1990). Maximum concentrations of the remaining inorganics are below tolerance limits and slightly exceed effect levels observed to impact the lower tenth percentile of biota inhabiting sediments. Therefore, the potential of adverse impacts resulting from these

TABLE 8-8. SEDIMENT ECOLOGICAL RISK ASSESSMENT SUMMARY.

CONTAMINANT OF CONCERN	SEDIMENT CONC. (mg/kg)		SEDIMENT CRITERIA OR GUIDELINES[1] (mg/kg)		RISK INDICES[2] ER-Ls or AQUATIC TOXICITY		RISK INDICES[2] for WILDLIFE RESIDUE	
	MEAN	MAXIMUM	AQ. TOXIC.	WILD. RES.	MEAN	MAXIMUM	MEAN	MAXIMUM
NORTHERN WETLAND								
Bis (2-ethylhexyl) phthalate	5.10E-01	2.90E-01 *	1.20E+00	-	4.26E-01	2.42E-01	-	-
Benzo(a)anthracene	3.55E-01	1.40E-01 *	1.32E+01	-	2.69E-02	1.06E-02	-	-
Benzo(b)fluoranthene	3.42E-01	1.30E-01 *	-	-	-	-	-	-
Benzo(k)fluoranthene	3.79E-01	1.60E-01 *	-	-	-	-	-	-
Benzo(a)pyrene	3.55E-01	1.40E-01 *	1.06E+01	-	3.35E-02	1.32E-02	-	-
Chrysene	3.67E-01	1.50E-01 *	4.00E-01	-	9.18E-01	3.75E-01	-	-
Fluoranthene	1.19E-01	2.30E-01	1.88E+01	-	6.33E-03	1.22E-02	-	-
Phenanthrene	6.26E-02	9.80E-02	1.39E+00	-	4.50E-02	7.05E-02	-	-
Pyrene	9.75E-02	1.90E-01	1.31E+01	-	7.44E-03	1.45E-02	-	-
4,4-DDT	1.69E-02	7.70E-02	5.00E-01	8.28E-03	3.38E-02	1.54E-01	2.04E+00	9.30E+00
Aluminum	2.19E+04	2.49E+04	-	-	-	-	-	-
Arsenic	2.44E+01	2.70E+01	5.00E+00	-	4.87E+00	5.40E+00	-	-
Barium	1.28E+02	1.48E+02	-	-	-	-	-	-
Beryllium	1.05E+00	1.10E+00	-	-	-	-	-	-
Chromium, Total	2.87E+01	3.08E+01	2.60E+01	-	1.10E+00	1.18E+00	-	-
Cobalt	1.14E+01	1.27E+01	-	-	-	-	-	-
Copper	2.94E+01	3.55E+01	1.90E+01	-	1.55E+00	1.87E+00	-	-
Iron	2.60E+04	3.01E+04	2.40E+04	-	1.08E+00	1.25E+00	-	-
Lead	4.14E+01	4.90E+01	2.70E+01	-	1.53E+00	1.81E+00	-	-
Manganese	3.21E+02	5.51E+02	4.28E+02	-	7.50E-01	1.29E+00	-	-
Mercury	1.49E-01	5.60E-01	1.10E-01	-	1.35E+00	5.09E+00	-	-
Nickel	3.05E+01	3.09E+01	2.20E+01	-	1.38E+00	1.40E+00	-	-
Vanadium	3.67E+01	4.05E+01	-	-	-	-	-	-
Zinc	1.15E+02	1.33E+02	8.50E+01	-	1.35E+00	1.56E+00	-	-
			TOTAL RISK INDEX		1.65E+01	2.18E+01	2.04E+00	9.30E+00
NORTHERN DRAINAGE SWALES								
Bis (2-ethylhexyl) phthalate	3.74E-01	4.40E-01	1.20E+00	-	3.12E-01	3.68E-01	-	-
Benzo(a)anthracene	3.65E-01	8.20E-02 *	1.32E+01	-	2.77E-02	6.21E-03	-	-
Benzo(b)fluoranthene	5.30E-01	2.50E-01 *	-	-	-	-	-	-
Chrysene	4.47E-01	1.50E-01 *	4.00E-01	-	1.12E+00	3.75E-01	-	-
Fluoranthene	4.47E-01	1.50E-01 *	1.88E+01	-	2.38E-02	7.98E-03	-	-
Phenanthrene	3.33E-01	6.20E-02 *	1.39E+00	-	2.39E-01	4.46E-02	-	-
Pyrene	4.37E-01	1.40E-01 *	1.31E+01	-	3.33E-02	1.07E-02	-	-
Delta-BHC	2.45E-03	5.40E-03	-	-	-	-	-	-
Endosulfan II	5.68E-03	7.80E-03	3.00E-04	-	1.89E+01	2.60E+01	-	-
4,4-DDT	5.46E-03	1.50E-02	5.00E-01	8.28E-03	1.09E-02	3.00E-02	6.59E-01	1.81E+00
Aluminum	1.77E+04	2.18E+04	-	-	-	-	-	-
Arsenic	1.71E+01	2.40E+01	5.00E+00	-	3.42E+00	4.80E+00	-	-
Barium	9.06E+01	1.13E+02	-	-	-	-	-	-
Beryllium	7.51E-01	9.10E-01	-	-	-	-	-	-
Chromium, Total	2.27E+01	2.80E+01	2.60E+01	-	8.72E-01	1.08E+00	-	-
Cobalt	8.69E+00	1.11E+01	-	-	-	-	-	-
Copper	1.90E+01	3.19E+01	1.90E+01	-	1.00E+00	1.68E+00	-	-
Iron	2.19E+04	2.73E+04	2.40E+04	-	9.14E-01	1.14E+00	-	-
Lead	3.03E+01	4.40E+01	2.70E+01	-	1.12E+00	1.63E+00	-	-
Manganese	4.32E+02	6.45E+02	4.28E+02	-	1.01E+00	1.51E+00	-	-

TABLE 8-8. SEDIMENT ECOLOGICAL RISK ASSESSMENT SUMMARY. (continued)

CONTAMINANT OF CONCERN	SEDIMENT CONC. (mg/kg)		SEDIMENT CRITERIA OR GUIDELINES[1] (mg/kg)		RISK INDICES[2] ER-Ls or AQUATIC TOXICITY		RISK INDICES[2] for WILDLIFE RESIDUE	
	MEAN	MAXIMUM	AQ. TOXIC.	WILD. RES.	MEAN	MAXIMUM	MEAN	MAXIMUM
Mercury	1.00E-01	3.20E-01	1.10E-01	-	9.09E-01	2.91E+00	-	-
Nickel	1.96E+01	2.76E+01	2.20E+01	-	8.92E-01	1.25E+00	-	-
Vanadium	3.29E+01	3.91E+01	-	-	-	-	-	-
Zinc	8.39E+01	1.24E+02	8.50E+01	-	9.87E-01	1.46E+00	-	-
			TOTAL RISK INDEX		3.18E+01	4.43E+01	6.59E-01	1.81E+00
SOUTHERN DRAINAGE SWALES								
Bis (2-ethylhexyl) phthalate	6.45E-01	1.40E+00	1.20E+00	-	5.39E-01	1.17E+00	-	-
Benzo(a)anthracene	6.60E-01	2.10E-01 *	1.32E+01	-	5.00E-02	1.59E-02	-	-
Benzo(b)fluoranthene	6.90E-01	3.20E-01 *	-	-	-	-	-	-
Benzo(k)fluoranthene	6.70E-01	2.50E-01 *	-	-	-	-	-	-
Benzo(a)pyrene	7.43E-01	2.50E-01 *	1.06E+01	-	7.01E-02	2.36E-02	-	-
Benzo(g,h,i)perylene	1.43E+00	2.30E-01 *	-	-	-	-	-	-
Chrysene	5.99E-01	2.70E-01 *	4.00E-01	-	1.50E+00	6.75E-01	-	-
Fluoranthene	5.89E-01	3.30E-01 *	1.88E+01	-	3.13E-02	1.76E-02	-	-
Phenanthrene	5.51E-01	1.80E-01 *	1.39E+00	-	3.97E-01	1.29E-01	-	-
Pyrene	5.97E-01	3.10E-01 *	1.31E+01	-	4.56E-02	2.37E-02	-	-
Delta-BHC	3.94E-03	4.40E-03	-	-	-	-	-	-
Gamma-BHC	2.79E-03	1.50E-03 *	1.57E-03	-	1.78E+00	9.55E-01	-	-
Aldrin	2.77E-03	2.00E-03 *	8.40E-02	7.70E-03	3.29E-02	2.38E-02	3.59E-01	2.60E-01
Heptachlor epoxide	2.70E-03	3.10E-03	3.00E-04	1.00E-03	8.99E+00	1.03E+01	2.70E+00	3.10E+00
Dieldrin	3.35E-03	2.25E-03 *	1.99E-01	1.30E-03	1.68E-02	1.13E-02	2.58E+00	1.73E+00
Endrin	4.00E-03	1.80E-02	1.00E-02	5.33E-04	4.00E-01	1.80E+00	7.50E+00	3.38E+01
Endosulfan II	7.55E-03	4.25E-03 *	3.00E-04	-	2.52E+01	1.42E+01	-	-
4,4-DDD	3.05E-03	4.70E-03	5.00E-01	-	6.10E-03	9.40E-03	-	-
4,4-DDE	3.77E-03	2.80E-02	5.00E-01	-	7.54E-03	5.60E-02	-	-
4,4-DDT	7.33E-03	1.10E-02	5.00E-01	8.28E-03	1.47E-02	2.20E-02	8.85E-01	1.33E+00
Aluminum	1.60E+04	2.78E+04	-	-	-	-	-	-
Antimony	6.54E+00	7.75E+00	-	-	-	-	-	-
Arsenic	1.34E+01	2.76E+01	5.00E+00	-	2.68E+00	5.52E+00	-	-
Barium	1.10E+02	1.32E+02	-	-	-	-	-	-
Beryllium	7.34E-01	8.64E-01	-	-	-	-	-	-
Cadmium	4.67E-01	9.06E-01	8.00E-01	-	5.84E-01	1.13E+00	-	-
Chromium, Total	2.16E+01	2.58E+01	2.60E+01	-	8.31E-01	9.92E-01	-	-
Cobalt	9.03E+00	1.18E+01	-	-	-	-	-	-
Copper	2.01E+01	2.68E+01	1.90E+01	-	1.06E+00	1.41E+00	-	-
Iron	2.21E+04	6.90E+04	2.40E+04	-	9.19E-01	2.88E+00	-	-
Lead	4.70E+01	6.32E+01	2.70E+01	-	1.74E+00	2.34E+00	-	-
Manganese	3.62E+02	5.64E+02	4.28E+02	-	8.46E-01	1.32E+00	-	-
Mercury	1.24E-01	5.25E-01	1.10E-01	-	1.13E+00	4.77E+00	-	-
Nickel	2.11E+01	2.74E+01	2.20E+01	-	9.59E-01	1.25E+00	-	-
Vanadium	2.83E+01	3.57E+01	-	-	-	-	-	-
Zinc	1.31E+02	1.74E+02	8.50E+01	-	1.54E+00	2.05E+00	-	-
			TOTAL RISK INDEX		5.13E+01	5.31E+01	1.40E+01	4.02E+01

* Maximum detected concentration below detection limit. One-half detection limit was utilized for non-detects, therefore, mean conc. > maximum conc.

[1] Sediment Criteria/Guidance from Table 8-5.

[2] Risk Indices = Contaminant Sediment Concentration (mean or maximum) divided by Sediment Criteria/Guidelines.

metals is low. The average concentrations of 4,4-DDT exceed criteria developed to prevent bioaccumulation of contaminants within aquatic organisms to harmful levels that may impact species that feed on these organisms. The extent of the 4,4-DDT contamination is unknown as it was detected in one of two samples.

Northern Drainage Swales

Average concentrations of the COCs chrysene, endosulfan II, arsenic, copper, lead, and manganese are at or above criteria/guidelines. Maximum concentrations of endosulfan II, arsenic, chromium, copper, iron, lead, manganese, mercury, nickel and zinc are also above their respective criteria/guidelines. However, only arsenic has concentrations above inorganic background concentrations (NYSDEC, 1989). Arsenic concentrations (mean and maximum) are below reported levels resulting in acute adverse effects (NYSDEC, 1989 and NOAA, 1990). The concentrations of inorganic contaminants detected, therefore, do not have a high probability of resulting in adverse effects to aquatic biota residing in these swales. Concentrations of chrysene are of the same order of magnitude at their respective criteria. A high likelihood of acute adverse impacts is only expected from organic contaminants with a risk index greater than 100.0 although chronic impacts are likely if a risk index is greater than 10.0 (NYSDEC, 1989). This is a result of uncertainties involved in establishing sediment criteria using equilibrium partitioning (an order of magnitude relating to the correlation between the organic carbon and octanol/water partition coefficients) and acute: chronic toxicity ratios (generally one order of magnitude). Endosulfan II concentrations exceeded the endosulfan criterion (Risk Index = 18.9 for mean concentration), however, this COC was detected in only one of three samples.

Southern Drainage Swales

Mean concentrations for chrysene, gamma-BHC, heptachlor epoxide, endosulfan II, arsenic, lead, mercury, and zinc are above criteria/guidelines. However, the risk index was below 10.0 for each organic COC except endosulfan II; and maximum

concentrations of copper and mercury were below inorganic background concentrations (NYSDEC, 1989). Although the maximum concentrations of arsenic, lead, and zinc are above background levels, the reported levels are below tolerance limits reported by the NYSDEC (1989). Average and maximum concentrations of lead and zinc, however, are above effect levels reported to adversely impact the lower tenth percentile of test organisms observed (NOAA, 1990). The average concentrations of endosulfan II may result in chronic effects to aquatic organisms; however, this contaminant was detected in only two of fifteen samples; therefore, exposure of benthic organisms within this area to endosulfan II is expected to be limited.

8.6.3 Surficial Soils

Mean and maximum surface soil inorganic COC levels were compared with soil cleanup guidelines (OMOE, 1991) developed to protect agricultural vegetation from adverse effects. Results are presented in Table 8-9. Mean concentrations of all inorganics (for which guidelines have been developed) produce risk indices below 1.0. Risk indices based on maximum concentrations exceeded 1.0 for only arsenic (risk index = 1.1).

8.6.4 Uncertainty Analysis

AWQC for COCs detected in surface water samples do not consider synergistic effects from the interaction of different chemicals with one another. In addition, EPA believes that "acid-soluble" metal concentrations in surface waters is the most scientifically valid method to assess the impact of metals on aquatic organisms (EPA, 1986). An approved methodology for measuring "acid-soluble" metal concentrations does not currently exist, therefore, EPA recommends applying metal AWQC to total recoverable metal concentrations. This comparison, however, may be overly protective of aquatic life.

TABLE 8-9. SURFACE SOIL ECOLOGICAL RISK ASSESSMENT SUMMARY.

CONTAMINANT OF CONCERN	SURFACE SOIL CONCENTRATION (mg/kg)		SOIL CLEAN-UP GUIDELINES[1] (mg/kg)	RISK INDICES[2]	
	MEAN	MAXIMUM		MEAN	MAXIMUM
Bis (2-ethylhexyl) phthalate	3.26E-01	6.52E-01	-	-	-
Delta-BHC	2.47E-03	1.40E-03 *	-	-	-
Aluminum	1.06E+04	2.60E+04	-	-	-
Arsenic	1.06E+01	2.21E+01	2.00E+01	5.29E-01	1.11E+00
Barium	5.08E+01	1.03E+02	7.50E+02	6.77E-02	1.38E-01
Beryllium	4.07E-01	8.08E-01	4.00E+00	1.02E-01	2.02E-01
Chromium, Total	1.42E+01	2.10E+01	7.50E+02	1.89E-02	2.80E-02
Cobalt	5.59E+00	8.04E+00	4.00E+01	1.40E-01	2.01E-01
Copper	1.30E+01	1.99E+01	1.50E+02	8.64E-02	1.33E-01
Iron	1.49E+04	3.10E+04	-	-	-
Lead	1.52E+01	1.43E+02	-	-	-
Manganese	2.60E+02	4.82E+02	-	-	-
Nickel	1.50E+01	1.98E+01	1.50E+02	1.00E-01	1.32E-01
Vanadium	1.99E+01	2.55E+01	2.00E+02	9.95E-02	1.28E-01
Zinc	4.87E+01	6.87E+01	6.00E+02	8.12E-02	1.15E-01
		TOTAL RISK INDEX		1.22E+00	2.10

* Maximum detected concentration below detection limit. One-half detection limit was utilized for non-detects, therefore, mean conc. > maximum conc.

[1] Soil Criteria/Guidance from Table 8-6.

[2] Risk Indices = Contaminant Surface Soil Concentration (mean or maximum) divided by Soil Guidelines.

9.0 SUMMARY AND CONCLUSIONS

Public Health Risk

The public health risk assessment evaluated potential risks associated with contamination in ground water, surface soils, subsurface soils, leachate soils, and sediments at the NCR site. Results of the quantitative risk assessment indicate that, under current site conditions, site media do not pose a risk to public health. Under certain future conditions, however, groundwater at the site potentially poses a significant risk (either a carcinogenic risk greater than 10^{-4} or a noncarcinogenic hazard index greater than one). It is important to note that all risk calculations were based on either the maximum concentrations detected or the upper ninety-five percent confidence limit of the mean, whichever is lower.

Both future ground water scenarios indicate a potential for risk if (1) future residents adjacent to the site install drinking water wells, and (2) ground water contaminants at the NCR site migrate to these future wells.

Ingestion of contaminated ground water from the perimeter wells and the northernmost landfill cell well produced carcinogenic risks of 2E-04 and 1E-04, respectively. Risks in both scenarios are attributed primarily to arsenic and beryllium. Styrene also contributed (increased CA risk = 2E-05), but less significantly, to the total CA risk in the northern landfill cell ground water evaluation.

The highest levels of arsenic in site ground water (including well NCR-12) were detected in samples from wells NCR-1, NCR-2, and NCR-11. All of these wells are located beyond the immediate perimeter of the landfill cells and may reflect background concentrations of inorganics rather than site-related contamination.

Beryllium was detected in 8 out of 45 samples analyzed from the perimeter ground water monitoring wells and in one out of three samples in the northern landfill cell well NCR-12. Increased CA risk due to beryllium in perimeter ground water was based on an upper ninety-five percent confidence limit of the mean concentration equal to 0.77 ug/kg. A significantly elevated concentration of 21.20 ug/kg was detected in well NCR-6. Styrene was detected in 3 out of the 3 samples analyzed from well location NCR-12 and at a maximum concentration of 67 ug/kg. Styrene was not detected in samples from any other well location.

Significant noncarcinogenic hazard indices for both the northern landfill cell and perimeter ground water scenarios were attributable to antimony. Iron, aluminum, arsenic, and manganese also contributed to the total hazard index for perimeter ground water. The highest levels of iron and aluminum detected in site ground water (both perimeter and northern landfill cell ground water) were from NCR-2. The highest level of antimony detected was from NCR-11. The maximum concentrations detected for arsenic and manganese were from NCR-1 and NCR-2, respectively. Outlying well locations NCR-11, NCR-1, and NCR-2 are the source of the highest concentrations of the contaminants primarily responsible for the elevated hazard indices for the groundwater ingestion pathways. Due to their outlying location, it is questionable if elevated levels of inorganics detected in these locations are site-related or, rather, are naturally occurring.

No other future scenarios evaluated indicate a potential for significant risk. However, youth trespassers scenarios involving exposure via incidental ingestion of surface soils and sediments resulted in carcinogenic risk estimates of 4E-06 and 5E-06, respectively. Both risk estimates are attributable to arsenic which was detected in both media, in all samples analyzed, and at maximum concentrations of approximately 28 mg/kg. Site-specific background samples to evaluate inorganics levels in soils are not available.

Exposure via dermal contact as well as exposure to contaminants detected but without numerical toxicity values could pose additional risks not represented in this quantitative risk assessment. For example, exposure via dermal contact with soils is likely in the current and future trespasser scenarios as well as in the future excavation scenarios. However, as mentioned earlier in this document, uncertainty in methods available to evaluate this pathway preclude a quantitative assessment for the contaminants detected in soils. The combined risks for dermal contact and ingestion in both scenarios could potentially result in a nominally greater risk. However, although dermal risks are possible, they are unlikely to be greater than ingestion and may be lower.

Several chemicals selected as COCs could not be evaluated quantitatively because numerical toxicity values are currently unavailable. These chemicals are phenanthrene, delta-BHC, cobalt, and lead. Lead is of concern because it was detected in perimeter ground water at a maximum concentration of 77.90 ug/L (at location NCR-2) which exceeds the EPA's MCL/action level of 15 ug/L. The upper 95 percent confidence limit, however, did not exceed this level. Lead is currently classified by EPA as a B2 carcinogen. Lead was detected frequently in all soil media, although below EPA's target clean-up range of 500 to 1000 mg/kg. Other COCs detected frequently in many site media were cobalt, phenanthrene, and delta-BHC. The potential effect of these chemicals is difficult to evaluate due to a lack of toxicity data. Cobalt concentrations produced dose estimates generally considered to be safe.

Both carcinogenic and noncarcinogenic risks result from contaminants detected in ground water at the NCR site. Except for styrene, all contaminants contributing to risk are inorganics. The historical use of the site as a landfill for a variety of household, commercial, and industrial waste precludes the elimination of chemicals that may be representative of naturally occurring concentrations. However, the collection of site-specific background samples will assist in determining whether detected concentrations of inorganics are anthropengenic or naturally occurring.

Ecological Risk

Surface water and sediment concentrations of metals (primarily aluminum, lead, and zinc) and pesticides (primarily 4,4-DDT) may result in adverse chronic effects in aquatic organisms within the drainage swales and streams present on the site or in close proximity. Acute toxic effects may also occur in aquatic organisms within the southern drainage swale due to elevated metal concentrations detected in the surface water.

Northern wetland stream sediments do not pose significant risk to aquatic benthic organisms although 4,4-DDT concentrations may potentially bioaccumulate within species that forage on the aquatic species present in this area. 4,4-DDT was detected in one of two samples, but, the full extent of the 4,4-DDT contamination is unknown. Aluminum concentrations within the northern wetland surface water may result in adverse chronic effects in aquatic organisms. However, the risk of aquatic organisms suffering acute effects due to the aluminum concentrations present within this stream is low. Maximum detected concentrations of lead and zinc may also result in chronic adverse effects if organisms are frequently exposed to these levels.

Surface water contaminant concentrations within the northern drainage swales are not expected to be acutely toxic to aquatic organisms. The concentration of 4,4-DDT present within these drainage swales may result in adverse chronic effects in aquatic organisms within this area as this contaminant biomagnifies within the food chain. However, 4,4-DDT was only detected in one of the two surface water samples and one of the three sediment samples collected; therefore, it is not known if biota are frequently exposed to this contaminant which would result in elevated tissue concentrations. Sediment contaminant concentrations do not appear to pose a significant risk.

The surface water and sediment in the southern drainage swales contained high concentrations of heavy metals (aluminum, lead, and zinc). The maximum reported surface water concentrations for aluminum may presently result in acute adverse effects in sensitive aquatic organisms. The maximum concentrations of aluminum, zinc, lead, and bis(2-ethylhexyl)phthalate in surface waters may result in chronic adverse impacts if biota are exposed to these levels frequently. Sediments within these swales also contained elevated concentrations of pesticides that may potentially biomagnify within the tissues of organisms located at higher trophic levels of the food chain. Pesticides of concern were detected in 20% or less of samples, therefore, exposure may not be widespread through the southern drainage swales. However, receptor species that forage within the impacted areas may be exposed to pesticide concentrations which result in tissues accumulating elevated levels of pesticides.

Leachate water concentrations of 4-methylphenol, aluminum, lead, and zinc may result in acute toxic effects to aquatic organisms (if present) within these areas. Chronic effects may also occur from these contaminants and elevated levels of heptachlor, 4,4-DDD, 4,4-DDT, and mercury.

Based upon the computed risk indices for the Northern wetland stream, the northern drainage swales and the southern drainage swales, the potential for chronic impacts to occur to resident species has been established (i.e., the indices exceeding unity). In addition, in the southern drainage swale, acute effects are also likely to occur. The stressed vegetation associated with the northern wetland area may be chemically induced.

10.0 REFERENCES

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APPENDIX A

SUMMARY STATISTICS FOR THE NCR SITE PUBLIC HEALTH RISK ASSESSMENT

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE.

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Surface Soils

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Vinyl Chloride	1	12	240.00	240.00	NCR-13(0-0.8')	8.03	38.23	10.00	13.0
Methylene Chloride	6	12	6.50	22.00	HA-3	6.49	19.81	5.20	6.5
Acetone	4	12	6.50	17.00	HA-3	7.42	10.48	10.00	13.0
1,1,1-Trichloroethane	2	12	3.00	6.10	NCR-8(0.0-2.0')	3.16	3.65	5.20	6.5
Trichloroethylene	2	12	20.00	32.00	NCR-13(0-0.8')	4.24	11.82	5.20	6.5
Styrene	1	12	1.40	1.40	NCR-13(0-0.8')	2.79	3.24	5.20	6.5
1,2-Dichloroethylene (total)	1	12	440.00	440.00	NCR-13(0-0.8')	4.50	66.28	5.20	6.5
bis(2-Ethylhexyl)phthalate	2	12	700.00	1025.00	HA-9	326.36	651.98	340.00	1200.0
Delta-BHC	1	12	1400.00	1400.00	HA-8	2465.99	14123.04	1100.00	14000.0
Aluminum	12	12	4650000.00	26000000.00	NCR-8(0.0-2.0')	10575373.34	16326885.74	.	.
Arsenic	12	12	2700.00	28000.00	NCR-11A(0-3.2')	10578.94	22102.73	.	.
Barium	12	12	18600.00	135000.00	NCR-11B(0-3.2')	50755.91	103277.58	.	.
Beryllium	11	12	260.00	1100.00	NCR-8(0.0-2.0')	407.32	808.73	180.00	180.0
Calcium	12	12	3140000.00	68900000.00	HA-14	14630170.16	72547708.80	.	.
Chromium, total	12	12	6400.00	31000.00	NCR-8(0.0-2.0')	14190.55	21035.94	.	.
Cobalt	12	12	3000.00	14000.00	NCR-8(0.0-2.0')	5586.08	8044.25	.	.
Copper	12	12	7400.00	32000.00	NCR-13(0-0.8')	12964.15	19922.59	.	.
Iron	12	12	10395000.00	31000000.00	NCR-8(0.0-2.0')	14895316.02	19370286.45	.	.
Lead	11	11	4900.00	175000.00	NCR-11B(0-3.2')	15213.35	143424.42	.	.
Magnesium	12	12	2850000.00	25500000.00	HA-14	7162948.35	15744803.91	.	.
Manganese	12	12	63000.00	575000.00	HA-8	259798.95	482275.12	.	.
Nickel	12	12	9000.00	26000.00	NCR-11B(0-3.2')	15035.72	19755.66	.	.
Potassium	12	12	727000.00	6300000.00	NCR-8(0.0-2.0')	1404302.59	2521111.61	.	.
Sodium	11	12	89000.00	690000.00	NCR-7(0.0-2.0')	207982.45	455773.89	100000.00	100000.0
Vanadium	12	12	13100.00	37000.00	NCR-8(0.0-2.0')	19905.94	25547.40	.	.
Zinc	12	12	30700.00	105000.00	NCR-11B(0-3.2')	48734.98	68688.54	.	.

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Subsurface Soils

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Vinyl Chloride	1	10	210.00	210.00	NCR-13(2.5-3.5)	8.70	57.50	11.00	15.0
Methylene Chloride	4	10	3.50	17.00	NCR-13(2.5-3.5)	6.91	42.50	5.60	150.0
Acetone	1	10	58.00	58.00	NCR-6(2.0-6.0')	8.80	28.45	11.00	66.0
1,1,1-Trichloroethane	2	10	5.60	5.70	NCR-10(2-4')	3.37	4.22	5.40	7.0
Trichloroethylene	4	10	6.80	15.00	NCR-6(2.0-6.0')	5.23	13.57	5.40	7.0
1,2-Dichloroethylene (total)	2	10	160.00	320.00	NCR-9M(2.0-6.0)	7.14	798.91	5.40	7.0
4-Methylphenol	1	9	68.00	68.00	TESTPIT3R	187.26	301.69	350.00	860.0
Benzoic Acid	1	9	220.00	220.00	TESTPIT3R	882.67	1731.85	1700.00	4200.0
Naphthalene	1	9	43.00	43.00	TESTPIT3R	177.96	356.10	350.00	860.0
2,4,5-Trichlorophenol	1	9	50.00	50.00	TESTPIT3R	748.69	4513.85	1700.00	4200.0
Phenanthrene	1	9	60.00	60.00	TESTPIT3R	184.67	313.74	350.00	860.0
Fluoranthene	1	9	86.00	86.00	TESTPIT3R	192.21	283.84	350.00	860.0
Pyrene	1	9	67.00	67.00	TESTPIT3R	186.95	303.02	350.00	860.0
Chrysene	1	9	46.00	46.00	TESTPIT3R	179.30	346.18	350.00	860.0
bis(2-Ethylhexyl)phthalate	1	9	160.00	160.00	TESTPIT3R	205.93	263.00	350.00	860.0
Di-n-octylphthalate	1	9	52.00	52.00	NCR-6(2.0-6.0')	184.54	336.66	350.00	860.0
Alpha-BHC	1	10	260.00	260.00	NCR-9M(2.0-6.0)	3623.01	34859.69	1000.00	21000.0
Heptachlor epoxide	1	9	580.00	580.00	NCR-5(5.4-7.0')	3744.94	27961.32	1000.00	21000.0
Aluminum	9	9	5000000.00	25000000.00	NCR-3M(4-6')	11278846.01	19395607.03	.	.
Antimony	1	10	13000.00	13000.00	NCR-5(5.4-7.0')	4323.12	7818.92	2800.00	12000.0
Arsenic	10	10	3100.00	26000.00	NCR-3M(4-6')	9392.10	20212.47	.	.
Barium	9	10	12000.00	160000.00	NCR-3M(4-6')	37497.33	1777260.25	1100.00	1100.0
Beryllium	8	10	200.00	960.00	NCR-3M(4-6')	301.52	1115.53	140.00	160.0
Calcium	10	10	2600000.00	90000000.00	NCR-3M(4-6')	32360885.40	208620777.88	.	.
Chromium, total	10	10	4000.00	30000.00	NCR-3M(4-6')	12781.02	25758.69	.	.
Cobalt	10	10	2100.00	15000.00	NCR-3M(4-6')	5910.77	12078.41	.	.
Copper	10	10	4000.00	242000.00	TESTPIT3SP	16741.90	107784.90	.	.

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Subsurface Soils

(continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Iron	9	9	8700000.00	31000000.00	NCR-3M(4-6')	16392629.60	26166527.21	.	.
Lead	10	10	6300.00	20500.00	TESTPIT3R	11809.43	16354.81	.	.
Magnesium	9	9	2200000.00	39000000.00	NCR-5(5.4-7.0')	12372993.45	54082052.04	.	.
Manganese	9	9	190000.00	1300000.00	NCR-3M(4-6')	483370.01	1001137.35	.	.
Mercury	1	10	230.00	230.00	TESTPIT3R	35.03	81.62	50.00	110.0
Nickel	10	10	2600.00	30000.00	NCR-3M(4-6')	12429.08	32719.44	.	.
Potassium	10	10	470000.00	6000000.00	NCR-3M(4-6')	1978699.33	4619697.63	.	.
Selenium	1	10	820.00	820.00	TESTPIT3R	628.09	4667.37	340.00	5000.0
Sodium	10	10	120000.00	920000.00	NCR-3M(4-6')	292499.18	486389.39	.	.
Thallium	1	10	610.00	610.00	NCR-3M(4-6')	216.18	319.11	240.00	500.0
Vanadium	10	10	6300.00	36000.00	NCR-3M(4-6')	18616.95	33381.29	.	.
Zinc	10	10	25000.00	135000.00	TESTPIT3R	64390.05	104279.38	.	.

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE.

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Deep Subsurface Soils

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Vinyl Chloride	1	11	190.00	190.00	NCR-5(44-47')	7.77	38.82	11.00	12.0
Methylene Chloride	2	11	20.00	49.00	NCR-2(24-24.7')	4.37	18.33	5.40	5.9
Acetone	2	11	50.00	98.00	NCR-12(22-26')	9.30	41.02	11.00	15.5
2-Butanone (MEK)	1	10	10.00	10.00	NCR-3M(24-26')	5.99	6.84	11.00	12.0
Trichloroethylene	1	11	14.00	14.00	NCR-10(26-28')	3.21	5.06	5.35	5.9
Toluene	2	11	3.60	51.00	NCR-12(22-26')	3.71	11.64	5.35	5.9
Ethylbenzene	2	11	5.00	65.00	NCR-5(44-47')	3.90	14.66	5.35	5.9
Styrene	2	11	4.90	39.00	NCR-5(44-47')	3.72	9.94	5.35	5.9
Total Xylenes	1	11	26.00	26.00	NCR-12(22-26')	3.42	7.19	5.35	5.9
1,2-Dichloroethylene (total)	1	11	390.00	390.00	NCR-5(44-47')	4.34	87.92	5.35	5.9
Phenol	1	10	4550.00	4550.00	NCR-12(22-26')	251.27	1225.13	340.00	380.0
2-Methylphenol	1	10	230.00	230.00	NCR-12(22-26')	186.43	230.00	340.00	380.0
4-Methylphenol	1	10	290.00	290.00	NCR-12(22-26')	190.80	211.65	340.00	380.0
Benzoic Acid	1	9	2190.00	2190.00	NCR-12(22-26')	1001.99	1291.15	1700.00	1900.0
Di-n-butylphthalate	1	10	430.00	430.00	NCR-12(22-26')	198.47	246.13	340.00	380.0
Benzylbutylphthalate	3	10	1200.00	2800.00	NCR-12(22-26')	349.81	1982.24	340.00	380.0
bis(2-Ethylhexyl)phthalate	3	10	1400.00	2900.00	NCR-12(22-26')	362.58	2464.65	340.00	380.0
Di-n-octylphthalate	1	10	150.00	150.00	NCR-3M(24-26')	186.02	205.93	340.00	540.0
Delta-BHC	1	11	25000.00	25000.00	NCR-12(22-26')	6533.72	9775.73	9800.00	12000.0
Aroclor-1254	1	11	47000.00	47000.00	NCR-12(22-26')	104762.80	128496.72	200000.00	250000.0
Aluminum	11	11	2300000.00	9500000.00	NCR-10(26-28')	5978744.69	8729973.07	.	.
Antimony	3	11	13000.00	20000.00	NCR-7(44-46')	5409.47	12064.03	5050.00	9750.0
Arsenic	11	11	1800.00	13000.00	NCR-2(24-24.7')	6524.34	11376.45	.	.
Barium	11	11	33000.00	280000.00	NCR-1(44-46')	66706.23	115555.75	.	.
Beryll(	8	11	220.00	400.(	CR-10(44-46')	204.82	431.38	130.00	160.0
Cadmium	1	11	3600.00	3600.00	NCR-12(22-26')	239.41	818.05	300.00	490.0
[illegible]	11	11	54000000.00	150000000.00	NCR-7(44-46')	74096851.83	92956132.73	.	.

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Deep Subsurface Soils (continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Chromium, total	11	11	3500.00	12000.00	NCR-10(44-46')	7988.36	11288.53	.	.
Cobalt	11	11	1500.00	6050.00	NCR-3M(24-26')	3523.03	5436.62	.	.
Copper	11	11	4300.00	16700.00	NCR-12(22-26')	9631.73	14840.23	.	.
Iron	11	11	5200000.00	14100000.00	NCR-3M(24-26')	9389897.40	12228271.78	.	.
Lead	11	11	4900.00	14750.00	NCR-12(22-26')	9153.51	11871.59	.	.
Magnesium	11	11	16000000.00	93000000.00	NCR-7(44-46')	29700088.98	47402410.33	.	.
Manganese	11	11	220000.00	530000.00	NCR-2(24-24.7')	407740.34	496264.54	.	.
Mercury	1	11	150.00	150.00	NCR-12(22-26')	30.71	52.35	50.00	80.0
Nickel	11	11	3500.00	12850.00	NCR-3M(24-26')	7945.08	10998.85	.	.
Potassium	11	11	660000.00	3600000.00	NCR-7(44-46')	1864177.97	2991397.71	.	.
Sodium	11	11	210000.00	390000.00	NCR-12(22-26')	285831.32	331992.15	.	.
Vanadium	11	11	7400.00	17000.00	NCR-10(44-46')	11948.53	15207.72	.	.
Zinc	10	10	5400.00	120000.00	NCR-2(24-24.7')	42721.41	130247.99	.	.

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Landfill Leachate - Soil

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Methylene Chloride	1	2	67.00	67.00	SEEP-1-R	22.42	67.00	15.00	15.0
1,4-Dichlorobenzene (para)	1	2	1200.00	1200.00	SEEP-3-R	848.53	1200.00	1200.00	1200.0
4-Methylphenol	1	2	440.00	440.00	SEEP-3-R	513.81	440.00	1200.00	1200.0
Naphthalene	1	2	240.00	240.00	SEEP-3-R	379.47	240.00	1200.00	1200.0
2-Methylnaphthalene	1	2	120.00	120.00	SEEP-3-R	268.33	120.00	1200.00	1200.0
Acenaphthene	1	2	100.00	100.00	SEEP-3-R	244.95	100.00	1200.00	1200.0
Phenanthrene	1	2	470.00	470.00	SEEP-3-R	531.04	470.00	1200.00	1200.0
Anthracene	1	2	190.00	190.00	SEEP-3-R	337.64	190.00	1200.00	1200.0
Di-n-butylphthalate	2	2	81.00	350.00	SEEP-3-R	168.37	350.00	.	.
Fluoranthene	1	2	480.00	480.00	SEEP-3-R	536.66	480.00	1200.00	1200.0
Pyrene	1	2	840.00	840.00	SEEP-3-R	709.93	840.00	1200.00	1200.0
Benzylbutylphthalate	1	2	1200.00	1200.00	SEEP-3-R	848.53	1200.00	1200.00	1200.0
Benzo(a)anthracene	1	2	210.00	210.00	SEEP-3-R	354.96	210.00	1200.00	1200.0
Chrysene	1	2	200.00	200.00	SEEP-3-R	346.41	200.00	1200.00	1200.0
bis(2-Ethylhexyl)phthalate	1	2	750.00	750.00	SEEP-3-R	670.82	750.00	1200.00	1200.0
Delta-BHC	1	2	2100.00	2100.00	SEEP-1-R	3694.59	2100.00	13000.00	13000.0
Aldrin	1	2	2100.00	2100.00	SEEP-1-R	3694.59	2100.00	13000.00	13000.0
Dieldrin	1	2	3200.00	3200.00	SEEP-1-R	4560.70	3200.00	13000.00	13000.0
4,4-DDE	1	2	690.00	690.00	SEEP-1-R	2117.78	690.00	13000.00	13000.0
gamma-chlordane	1	2	1200.00	1200.00	SEEP-1-R	2792.85	1200.00	13000.00	13000.0
Aluminum	2	2	7810000.00	11700000.00	SEEP-3-R	9559131.76	11700000.00	.	.
Arsenic	2	2	7000.00	12000.00	SEEP-3-R	9165.15	12000.00	.	.
Barium	2	2	110000.00	110000.00	SEEP-1-R	110000.00	110000.00	.	.
Beryllium	2	2	290.00	600.00	SEEP-3-R	417.13	600.00	.	.
Cadmium	1	2	710.00	710.(	EEP-3-R	386.13	710.00	420.00	420.0
Calcium	2	2	78600000.00	81400000.00	SEEP-1-R	79987749.06	81400000.00	.	.

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Landfill Leachate - Soil

(continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Cobalt	2	2	4700.00	6800.00	SEEP-3-R	5653.32	6800.00	.	.
Copper	2	2	29400.00	47800.00	SEEP-3-R	37487.60	47800.00	.	.
Iron	2	2	23400000.00	25700000.00	SEEP-3-R	24523050.38	25700000.00	.	.
Lead	2	2	40000.00	110000.00	SEEP-1-R	66332.50	110000.00	.	.
Magnesium	2	2	29700000.00	31300000.00	SEEP-1-R	30489506.39	31300000.00	.	.
Manganese	2	2	441000.00	511000.00	SEEP-3-R	474711.49	511000.00	.	.
Mercury	2	2	390.00	1200.00	SEEP-3-R	684.11	1200.00	.	.
Nickel	2	2	16100.00	18700.00	SEEP-3-R	17351.37	18700.00	.	.
Potassium	2	2	1390000.00	2890000.00	SEEP-3-R	2004270.44	2890000.00	.	.
Sodium	2	2	365000.00	394000.00	SEEP-1-R	379222.89	394000.00	.	.
Vanadium	2	2	16300.00	23100.00	SEEP-3-R	19404.38	23100.00	.	.
Zinc	2	2	102000.00	119000.00	SEEP-3-R	110172.59	119000.00	.	.

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Drainage Swale Sediments

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Methylene Chloride	18	20	13.00	73.00	SED-13-R	27.55	40.44	26.50	28.0
Acetone	11	20	12.00	89.00	SED-11-R	17.25	37.77	15.00	61.0
1,1-Dichloroethane	1	20	19.00	19.00	SED-14-R	4.61	5.77	7.00	11.0
1,1,1-Trichloroethane	2	20	2.00	3.00	SED-7-R	4.09	4.63	7.00	11.0
Benzene	1	20	3.00	3.00	SED-14-R	4.21	4.56	7.00	11.0
Phenanthrene	6	20	40.00	180.00	SED-10-R	411.25	1418.18	980.00	2100.0
Di-n-butylphthalate	1	20	140.00	140.00	SED-12-R	743.56	997.61	980.00	2100.0
Fluoranthene	7	20	62.00	330.00	SED-10-R	481.56	1045.63	1100.00	2100.0
Pyrene	7	20	50.00	310.00	SED-10-R	475.21	1077.79	1100.00	2100.0
Benzo(a)anthracene	4	20	82.00	210.00	SED-10-R	567.52	1077.44	980.00	2100.0
Chrysene	5	20	120.00	270.00	SED-8-R	545.96	1007.92	980.00	2100.0
bis(2-Ethylhexyl)phthalate	11	20	110.00	3900.00	SED-18-R	581.04	1127.78	760.00	2100.0
Di-n-octylphthalate	1	20	290.00	290.00	SED-15-R	773.34	922.25	980.00	2100.0
Benzo(b)fluoranthene	4	20	130.00	320.00	SED-8-R	618.28	944.83	980.00	2100.0
Benzo(k)fluoranthene	3	20	160.00	250.00	SED-8-R	651.18	964.55	980.00	2100.0
Benzo(a)pyrene	2	20	140.00	250.00	SED-8-R	699.42	998.79	980.00	2100.0
Benzo(g,h,i)perylene	1	20	230.00	230.00	SED-8-R	1661.52	3362.68	980.00	6300.0
Delta-BHC	7	20	1700.00	5400.00	SED-4-R	3690.39	8760.88	2500.00	36000.0
Gamma-BHC	2	20	920.00	1500.00	SED-17-R	2702.18	8714.11	1700.00	36000.0
Aldrin	2	20	1100.00	2000.00	SED-10-R	2683.05	8973.99	1500.00	36000.0
Heptachlor epoxide	3	20	300.00	3100.00	SED-17-R	2633.35	10156.62	1700.00	36000.0
Dieldrin	3	20	1900.00	2250.00	SED-11-R	3099.56	11981.79	1600.00	54000.0
4,4-DDE	3	20	1100.00	28000.00	SED-17-R	3385.88	17101.00	1700.00	54000.0
Endrin	2	20	7600.00	18000.00	SED-10-R	3535.21	17269.26	1500.00	54000.0
Endosulfan II	3	20	2700.00	7800.00	SED-4-R	7602.86	22425.58	4700.00	94000.0
4,4-DD[illegible]	1	20	4700.00	4700.00	SED-13-R	2886.90	13285.69	1500.00	54000.0
4,4-DD[illegible]	5	20	6900.00	77000.(	SED-2-R	7627.34	28532.83	3500.00	71000.(

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Drainage Swale Sediments

(continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Methoxyclor	1	20	16000.00	16000.00	SED-7-R	14474.54	62847.59	7300.00	270000.0
Aluminum	20	20	6480000.00	27800000.00	SED-16-R	16790176.31	20683775.34	.	.
Antimony	1	20	15200.00	15200.00	SED-12SP	6388.67	7263.19	9600.00	15000.0
Arsenic	20	20	710.00	27600.00	SED-10-R	14770.00	34174.30	.	.
Barium	20	20	64500.00	218000.00	SED-9-R	108553.56	126267.53	.	.
Beryllium	20	20	480.00	1300.00	SED-16-R	763.27	877.54	.	.
Cadmium	3	20	840.00	2100.00	SED-18-R	416.41	690.26	480.00	1500.0
Calcium	20	20	5170000.00	115000000.00	SED-18-R	31275490.02	71761993.64	.	.
Chromium, total	20	20	13400.00	34800.00	SED-16-R	22382.94	25924.49	.	.
Cobalt	20	20	4800.00	17700.00	SED-18-R	9190.23	11282.33	.	.
Copper	20	20	10400.00	41950.00	SED-4SP	20678.88	26689.57	.	.
Iron	20	20	8590000.00	69000000.00	SED-9-R	22410606.62	29146279.32	.	.
Lead	20	20	21000.00	100000.00	SED-8-R	43413.93	56342.47	.	.
Magnesium	20	20	4210000.00	48700000.00	SED-18-R	11974238.01	19965754.78	.	.
Manganese	20	20	117000.00	695000.00	SED-8-R	367373.04	529966.90	.	.
Mercury	13	20	80.00	1650.00	SED-4SP	122.26	438.81	60.00	100.0
Nickel	20	20	7400.00	35400.00	SED-16-R	21627.57	26940.52	.	.
Potassium	20	20	1320000.00	6340000.00	SED-16-R	3226462.71	4321208.48	.	.
Selenium	1	19	710.00	710.00	SED-12SP	333.22	384.77	500.00	920.0
Sodium	20	20	240000.00	2260000.00	SED-12SP	550368.81	915804.80	.	.
Vanadium	20	20	12000.00	49000.00	SED-16-R	29698.60	35624.23	.	.
Zinc	20	20	66800.00	293000.00	SED-11-R	120834.76	155387.89	.	.

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Northern Landfill Cell Ground Water

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Methylene Chloride	1	2	12.50	12.50	NCR-12DSP	5.00	12.50	4.00	4.0
Acetone	2	3	46.00	320.00	NCR-12DSP	54.09	98782535923601	21.50	21.5
Carbon Disulfide	3	3	0.45	1.00	NCR-12D-II	0.63	3.36	.	.
1,1-Dichloroethane	1	3	1.00	1.00	NCR-12DSP	0.63	2.99	1.00	1.0
cis-1,2-Dichloroethylene	1	3	0.35	0.35	NCR-12DSP	0.44	0.74	1.00	1.0
Chloroform	1	3	5.50	5.50	NCR-12DSP	1.11	142059072.90	1.00	1.0
2-Butanone (MEK)	3	3	15.00	60.50	NCR-12DSP	25.83	5788.95	.	.
Trichloroethylene	1	3	9.50	9.50	NCR-12DSP	1.33	2241049355649.3	1.00	1.0
Benzene	2	3	0.70	5.00	NCR-12DSP	1.21	4190856.40	1.00	1.0
4-Methyl-2-Pentanone	1	3	6.50	6.50	NCR-12DSP	3.65	44.18	5.00	6.0
2-Hexanone (MBK)	1	3	4.00	4.00	NCR-12DSP	3.11	5.80	5.00	6.0
Tetrachloroethylene	1	3	1.00	1.00	NCR-12DSP	0.63	2.99	1.00	1.0
Toluene	3	3	1.00	49.50	NCR-12DSP	5.30	7.127841117E17	.	.
Ethylbenzene	1	3	9.50	9.50	NCR-12DSP	1.33	2241049355649.3	1.00	1.0
Styrene	3	3	2.00	67.00	NCR-12DSP	7.38	3.0285093115E16	.	.
1,4-Dichlorobenzene (para)	2	3	0.50	8.00	NCR-12DSP	2.15	340290871.49	5.00	5.0
Isopropylbenzene	1	1	0.30	0.30	NCR-12DSP	0.30	0.30	.	.
Naphthalene	1	1	0.80	0.80	NCR-12DSP	0.80	0.80	.	.
1,2,4-Trimethylbenzene	1	1	0.95	0.95	NCR-12DSP	0.95	0.95	.	.
1,3,5-Trimethylbenzene	1	1	0.30	0.30	NCR-12DSP	0.30	0.30	.	.
Total Xylenes	3	3	0.95	26.00	NCR-12DSP	3.67	20692517313047	.	.
Phenol	3	3	775.00	2650.00	NCR-12DSP	1350.75	58539.74	.	.
2-Methylphenol	3	3	16.50	175.00	NCR-12DSP	48.70	50726087.94	.	.
4-Methylphenol	3	3	21.50	245.00	NCR-12DSP	60.96	269616243.69	.	.
2,4-Di( )ylphenol	3	3	2.00	27.(	)CR-12DSP	5.74	497721907.72	.	.
Di-n-butylphthalate	1	3	2.00	2.0(	NCR-12D-I	5.16	4406981982.06	5.00	55.0
bis(2-Ethylhexyl)phthalate	1	3	3.50	3.50	NCR-12D-I	6.22	83517866.96	5.00	55.0

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Northern Landfill Cell Ground Water (continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Gamma-BHC	1	3	0.41	0.41	NCR-12D-II	3.71	5.2875826357E18	10.00	50.0
Heptachlor	2	3	0.59	6.70	NCR-12D-I	4.62	8.8525121067E15	50.00	50.0
Endosulfan sulfate	1	3	0.69	0.69	NCR-12D-II	7.01	4.4996957382E20	20.00	100.0
Aluminum	2	3	99.20	223.50	NCR-12DSP	130.40	1075.46	200.00	200.0
Antimony	2	3	26.60	44.75	NCR-12DSP	25.54	682.79	28.00	28.0
Arsenic	1	3	2.50	2.50	NCR-12DSP	1.78	4.33	3.00	3.0
Barium	3	3	9.45	97.60	NCR-12DSP	22.56	173036746.27	.	.
Beryllium	1	3	1.00	1.00	NCR-12DSP	0.63	2.99	1.00	1.0
Calcium	3	3	379000.00	511000.00	NCR-12D-II	458748.96	667440.07	.	.
Chromium, total	1	3	32.85	32.85	NCR-12DSP	5.90	13314286863.38	5.00	5.0
Cobalt	1	3	8.40	8.40	NCR-12DSP	4.19	186.96	5.00	7.0
Copper	1	3	31.00	31.00	NCR-12DSP	4.53	3003140543796.9	3.00	4.0
Iron	3	3	631.50	655.50	NCR-12DSP	641.42	655.50	.	.
Magnesium	3	3	61500.00	97150.00	NCR-12D-I	83172.82	172258.50	.	.
Manganese	3	3	21.30	24.25	NCR-12D-II	23.13	24.25	.	.
Nickel	1	3	22.80	22.80	NCR-12DSP	7.60	83333.04	7.00	11.0
Potassium	3	3	9990.00	117500.00	NCR-12DSP	25957.52	653736088476.73	.	.
Selenium	1	3	1.00	1.00	NCR-12DSP	4.83	1132078340.20	15.00	30.0
Silver	1	3	4.25	4.25	NCR-12DSP	2.77	11.95	4.00	5.0
Sodium	3	3	66050.00	83050.00	NCR-12DSP	72631.71	92942.70	.	.
Vanadium	1	3	15.25	15.25	NCR-12DSP	3.94	2578741.55	4.00	4.0
Zinc	2	3	12.25	15.70	NCR-12D-II	12.44	22.21	20.00	20.0

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Landfill Perimeter Ground Water

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Methylene Chloride	1	23	4.00	4.00	NCR-2I-II	1.50	1.97	1.00	5.0
Acetone	2	45	5.50	27.00	NCR-11DSP	2.86	3.90	1.00	27.0
Chloroform	1	45	1.00	1.00	NCR-11DSP	0.51	0.52	1.00	1.0
Benzene	1	45	1.00	1.00	NCR-2M-I	0.51	0.52	1.00	1.0
Toluene	1	45	5.00	5.00	NCR-2M-I	0.53	0.61	1.00	1.0
Ethylbenzene	1	45	1.00	1.00	NCR-2M-I	0.51	0.52	1.00	1.0
Total Xylenes	1	45	8.00	8.00	NCR-2M-I	0.53	0.65	1.00	1.0
Phenol	4	45	1.00	4.00	NCR-4S-I	2.49	2.67	5.00	10.0
Diethylphthalate	2	45	1.00	2.00	NCR-9M-II	2.45	2.68	2.00	10.0
Pentachlorophenol	1	45	3.00	3.00	NCR-13S-I	9.94	10.82	20.00	50.0
Di-n-butylphthalate	1	45	1.00	1.00	NCR-4M-I	2.49	2.64	5.00	10.0
Benzylbutylphthalate	3	45	2.00	4.00	NCR-5M-I	2.49	2.69	2.00	10.0
bis(2-Ethylhexyl)phthalate	9	45	0.80	23.00	NCR-5S-I	2.33	3.46	1.00	10.0
Alpha-BHC	2	45	0.54	0.65	NCR-3M-I	4.32	6.85	2.00	50.0
Beta-BHC	2	45	10.00	49.00	NCR-3S-I	5.08	7.70	2.00	50.0
Delta-BHC	3	45	0.51	1.20	NCR-2S-I	4.01	6.96	0.86	50.0
Gamma-BHC	6	45	0.85	3.00	NCR-11D-II	3.83	5.86	2.00	50.0
Heptachlor	18	45	0.70	90.00	NCR-13S-II	3.67	8.00	1.80	50.0
Aldrin	1	45	0.89	0.89	NCR-9M-I	4.57	6.56	2.00	50.0
Heptachlor epoxide	1	45	0.86	0.86	NCR-11D-I	4.57	6.57	2.00	50.0
Dieldrin	1	45	0.58	0.58	NCR-10M-I	8.92	13.98	4.00	100.0
4,4-DDE	10	45	0.81	570.00	NCR-11D-II	5.50	23.27	0.78	100.0
4,4-DDD	1	45	240.00	240.00	NCR-11D-II	9.61	15.34	4.00	100.0
Endosulfan sulfate	3	45	0.89	56.00	NCR-11D-II	8.56	14.22	4.00	100.0
4,4-DI	2	45	140.00	670.	CR-11D-II	10.67	21.52	4.00	100.0
Methoxyclor	2	45	2.50	7.8	NCR-4S-I	37.94	83.47	2.90	500.0
[illegible]	2	45	2.40	22.00	NCR-11D-II	9.04	12.27	4.00	100.0

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Landfill Perimeter Ground Water

(continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Endrin aldehyde	3	45	2.50	7.60	NCR-11D-II	6.94	12.83	1.70	100.0
alpha-chlordane	4	45	0.62	2.20	NCR-4S-II	4.56	8.73	2.00	500.0
gamma-chlordane	2	45	1.80	15.00	NCR-11D-II	3.88	9.24	0.74	500.0
Aluminum	32	44	73.80	80800.00	NCR-2I-I	543.84	19125.76	35.00	228.0
Antimony	4	41	23.10	69.80	NCR-11DSP	13.96	16.30	22.00	28.0
Arsenic	20	44	3.00	16.40	NCR-1M-II	2.95	5.73	2.00	3.0
Barium	44	45	3.40	431.00	NCR-2I-I	36.54	139.76	200.00	200.0
Beryllium	8	45	1.00	3.10	NCR-2I-I	0.61	0.77	1.00	1.0
Cadmium	3	43	4.40	5.00	NCR-11M-I	2.12	2.30	4.00	4.0
Calcium	44	44	35600.00	577000.00	NCR-2I-II	216649.56	498585.03	.	.
Chromium	17	45	7.50	134.00	NCR-3S-I	7.33	46.16	5.00	5.0
Cobalt	9	45	6.60	43.90	NCR-2I-I	4.15	6.80	5.00	7.0
Copper	22	45	3.10	127.00	NCR-2I-II	5.51	25.99	3.00	4.0
Iron	42	45	59.50	108000.00	NCR-2I-I	1247.78	30423.70	42.00	287.0
Lead	11	43	2.60	77.90	NCR-2I-I	3.97	13.01	1.00	27.0
Magnesium	44	44	28500.00	340000.00	NCR-11M-I	86075.61	117865.09	.	.
Manganese	43	45	17.25	3930.00	NCR-2I-II	135.41	1871.25	33.70	57.7
Mercury	2	44	1.20	1.80	NCR-2I-I	0.11	0.16	0.20	0.2
Nickel	18	44	9.50	155.00	NCR-3S-I	12.21	53.64	7.00	11.0
Potassium	44	44	1370.00	24300.00	NCR-2I-I	6714.22	10034.81	.	.
Silver	3	42	5.00	6.50	NCR-11DSP	2.36	2.63	4.00	5.0
Sodium	43	43	15100.00	3610000.00	NCR-3S-II	93015.46	193169.53	.	.
Vanadium	13	45	4.70	150.00	NCR-2I-I	4.02	14.76	4.00	4.0
Zinc	31	44	3.70	508.00	NCR-2I-II	23.84	101.35	9.30	96.8

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Drainage Swale Surface Water

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Carbon Disulfide	3	11	0.65	8.00	SW-18-R	2.26	4.37	5.00	5.0
1,1,1-Trichloroethane	1	11	2.00	2.00	SW-4-R	2.45	2.00	5.00	5.0
4-Methyl-2-Pentanone	1	11	2.00	2.00	SW-11-R	4.60	5.66	10.00	10.0
Tetrachloroethylene	1	11	4.00	4.00	SW-18-R	2.61	2.86	5.00	5.0
Toluene	2	11	2.00	2.00	SW-11-R	2.40	2.00	5.00	5.0
Ethylbenzene	1	11	1.00	1.00	SW-13	2.30	2.83	5.00	5.0
Total Xylenes	1	11	3.00	3.00	SW-11SP	2.54	3.00	5.00	5.0
Phenol	1	11	11.00	11.00	SW-4-R	5.32	6.34	9.00	10.0
2,4-Dimethylphenol	2	11	3.00	6.50	SW-11-R	4.84	5.47	9.00	10.0
Benzoic Acid	1	11	5.00	5.00	SW-13	21.00	32.51	47.00	51.0
Diethylphthalate	1	11	0.55	0.55	SW-11-R	4.05	8.36	9.00	10.0
Di-n-butylphthalate	2	11	0.30	0.40	SW-11-R	3.05	15.93	9.00	10.0
bis(2-Ethylhexyl)phthalate	8	11	0.70	1000.00	SW-18-R	9.10	1087.71	10.00	10.0
Delta-BHC	2	11	14.00	21.00	SW-4-R	9.39	24.55	9.40	51.0
Gamma-BHC	1	11	5.10	5.10	SW-2-R	7.47	18.26	9.40	51.0
Heptachlor epoxide	1	11	14.00	14.00	SW-8-R	8.19	20.43	9.40	51.0
4,4-DDT	1	11	40.00	40.00	SW-4-R	17.00	44.51	19.00	100.0
Aluminum	11	11	426.00	25200.00	SW-13	1563.81	28889.58	.	.
Arsenic	4	11	3.10	30.60	SW-13	3.62	17.03	4.00	4.0
Barium	11	11	55.00	456.00	SW-13	132.07	294.03	.	.
Beryllium	1	11	2.10	2.10	SW-8-R	1.00	1.29	1.00	2.0
Cadmium	1	11	5.70	5.70	SW-13	2.64	3.21	4.00	5.0
Calcium	11	11	46900.00	286000.00	SW-13	120300.45	227421.36	.	.
Chromium, total	3	11	26.90	38.00	SW-8-R	8.17	24.08	10.00	10.0
Cobalt	2	11	18.00	25.00	SW-13	6.30	11.97	7.00	10.0
Copper	4	11	21.90	94.00	SW-13	16.98	47.97	20.00	20.0
Iron	11	11	488.00	38000.	SW-13	2938.94	82958.16	.	.

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Drainage Swale Surface Water

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Lead	11	11	6.10	352.00	SW-8-R	24.95	587.36	.	.
Magnesium	11	11	28500.00	211000.00	SW-11SP	59387.65	113425.60	.	.
Manganese	11	11	27.00	1690.00	SW-8-R	129.55	3226.35	.	.
Nickel	4	11	27.00	63.00	SW-8-R	17.07	43.30	20.00	20.0
Potassium	11	11	5850.00	211000.00	SW-11SP	21577.36	135455.43	.	.
Sodium	11	11	29800.00	393500.00	SW-11-R	92516.52	262707.26	.	.
Thallium	1	11	4.40	4.40	SW-11SP	2.63	2.95	5.00	5.0
Vanadium	2	11	9.10	61.00	SW-13	11.69	20.05	20.00	20.0
Zinc	6	11	24.00	2360.00	SW-13	45.10	3687.53	20.00	20.0
Cyanide	4	11	15.60	40.60	SW-7-R	8.86	25.08	10.00	10.0

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Landfill Leachate - Water

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Methylene Chloride	1	8	470.00	470.00	SEEP-13-R	14.09	5561.49	5.00	200.0
Acetone	2	8	490.00	2200.00	SEEP-13-R	41.90	147284.93	10.00	200.0
2-Butanone (MEK)	4	8	96.00	1400.00	SEEP-13-R	77.11	36989.82	10.00	200.0
Benzene	3	8	36.00	50.00	SEEP-16-R	10.68	635.31	5.00	100.0
4-Methyl-2-Pentanone	3	8	3.00	21.00	SEEP-14-R	15.14	422.50	10.00	200.0
2-Hexanone (MBK)	2	8	11.00	270.00	SEEP-5SP	21.54	667.06	10.00	200.0
Toluene	6	8	3.00	410.00	SEEP-5SP	21.46	98816.98	5.00	5.0
Chlorobenzene	3	8	26.00	56.00	SEEP-16-R	12.61	392.01	5.00	100.0
Ethylbenzene	6	8	2.00	680.00	SEEP-16-R	22.32	149234.44	5.00	25.0
Total Xylenes	5	8	12.00	1400.00	SEEP-5SP	51.72	3142928.48	5.00	25.0
Phenol	6	8	45.00	1800.00	SEEP-7-R	138.89	1300392.06	10.00	11.0
1,3-Dichlorobenzene	1	8	6.00	6.00	SEEP-16-R	7.32	39.87	10.00	160.0
1,2-Dichlorobenzene	1	8	16.00	16.00	SEEP-16-R	8.28	50.02	10.00	160.0
2-Methylphenol	7	8	12.00	960.00	SEEP-7-R	155.33	81552.74	10.00	10.0
4-Methylphenol	6	8	400.00	3750.00	SEEP-5-R	340.17	39568104.28	10.00	11.0
2,4-Dimethylphenol	7	8	18.00	980.00	SEEP-16-R	61.24	3256.22	10.00	10.0
Benzoic Acid	2	8	4300.00	12000.00	SEEP-7-R	147.21	9364706.78	48.00	800.0
Naphthalene	4	8	0.60	200.00	SEEP-5-R	11.51	2970.92	10.00	160.0
4-Chloroaniline	1	8	160.00	160.00	SEEP-5SP	7.80	103.75	10.00	11.0
2-Methylnaphthalene	1	8	5.00	5.00	SEEP-21-R	7.07	39.96	10.00	160.0
2,6-Dinitrotoluene	1	8	51.00	51.00	SEEP-7-R	9.57	107.91	10.00	160.0
Acenaphthene	3	8	0.80	1.00	SEEP-21-R	3.76	163.63	10.00	160.0
Diethylphthalate	7	8	1.00	55.00	SEEP-7-R	13.98	332.92	10.00	10.0
Fluorene	2	8	0.60	1.00	SEEP-21-R	4.44	156.19	10.00	160.0
N-Nitrosodiphenylamine	2	8	2.00	7.00	SEEP-16-R	6.66	50.40	10.00	160.0
Phenanthrene	3	8	1.00	2.00	SEEP-16-R	4.60	85.73	10.00	160.0
Di-n-B iphthalate	3	8	0.40	4.	EEP-16-R	4.15	220.23	10.00	160.

SUMMARY STATISTICS FOR THE NIAGARA COUNTY REFUSE SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Landfill Leachate - Water

(continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Benzylbutylphthalate	2	8	0.90	4.00	SEEP-5-R	5.62	77.18	10.00	160.0
bis(2-Ethylhexyl)phthalate	7	8	0.70	10.00	SEEP-5-R	4.11	154.01	160.00	160.0
Delta-BHC	3	8	19.00	180.00	SEEP-5-R	27.49	433.53	9.40	100.0
Gamma-BHC	1	8	52.00	52.00	SEEP-14-R	23.57	929.91	9.40	535.0
Heptachlor	2	8	91.00	1100.00	SEEP-5-R	22.44	14360.69	9.40	97.0
Aldrin	1	8	82.00	82.00	SEEP-16-R	18.56	1305.50	9.40	535.0
4,4-DDD	1	8	15.00	15.00	SEEP-13-R	21.94	899.77	9.40	535.0
4,4-DDT	3	8	44.00	110.00	SEEP-10-R	71.41	655.50	19.00	1050.0
Aluminum	8	8	1140.00	325000.00	SEEP-14-R	7047.34	1204135.71	.	.
Arsenic	8	8	9.50	58.60	SEEP-10-R	20.02	41.56	.	.
Barium	8	8	147.00	7610.00	SEEP-10-R	756.29	9253.53	.	.
Beryllium	1	8	2.20	2.20	SEEP-14-R	1.35	2.85	2.00	10.0
Cadmium	2	8	6.10	9.00	SEEP-16-R	4.37	17.52	5.00	50.0
Calcium	8	8	93000.00	396000.00	SEEP-14-R	153062.89	279944.47	.	.
Chromium, total	8	8	26.00	118.00	SEEP-5SP	52.19	105.03	.	.
Cobalt	4	8	12.50	93.00	SEEP-21-R	17.04	178.88	10.00	100.0
Copper	5	8	28.00	99.00	SEEP-16-R	32.26	130.24	20.00	50.0
Iron	8	8	12555.00	390000.00	SEEP-10-R	43788.58	1010524.33	.	.
Lead	8	8	17.40	1010.00	SEEP-16-R	115.74	3668.17	.	.
Magnesium	8	8	107000.00	419000.00	SEEP-7-R	254841.73	432266.14	.	.
Manganese	8	8	76.00	2960.00	SEEP-14-R	392.71	8077.09	.	.
Mercury	2	8	0.50	0.50	SEEP-16-R	0.15	0.44	0.20	0.2
Nickel	8	8	34.00	157.00	SEEP-5SP	90.73	146.97	.	.
Potassium	8	8	26200.00	445000.00	SEEP-21-R	202462.04	1095864.25	.	.
Sodium	8	8	84800.00	1660000.00	SEEP-21-R	524801.63	4053790.68	.	.
Vanadium	4	8	23.00	33.00	SEEP-5-R	17.91	31.93	20.00	40.0
Zinc	8	8	204.00	1410.00	SEEP-7-R	564.89	1389.99	.	.

APPENDIX B

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSMENT AT THE NCR SITE

TRC

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE.

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

units = ppb, except pesticides/PCBs: units = ppt

TYPE=Northern Drainage Swale Sediments

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Methylene Chloride	3	3	23.00	32.00	SED-4-R	28.05	41.99	.	.
Acetone	2	3	13.00	18.00	SED-5-R	12.06	80.54	15.0	15.0
Phenanthrene	1	3	62.00	62.00	SED-4-R	332.89	333339828837.55	1400.0	1700.0
Fluoranthene	1	3	150.00	150.00	SED-4-R	446.89	2919833.82	1400.0	1700.0
Pyrene	1	3	140.00	140.00	SED-4-R	436.73	6038141.46	1400.0	1700.0
Benzo(a)anthracene	1	3	82.00	82.00	SED-4-R	365.41	4843713729.05	1400.0	1700.0
Chrysene	1	3	150.00	150.00	SED-4-R	446.89	2919833.82	1400.0	1700.0
bis(2-Ethylhexyl)phthalate	2	3	170.00	440.00	SED-4-R	374.11	58727.85	1400.0	1400.0
Benzo(b)fluoranthene	1	3	250.00	250.00	SED-4-R	529.85	35321.77	1400.0	1700.0
Delta-BHC	1	3	5400.00	5400.00	SED-4-R	2446.74	243517.22	3100.0	3500.0
Endosulfan II	1	3	7800.00	7800.00	SED-4-R	5680.51	12607.37	9400.0	10000.0
4,4-DDT	1	3	15000.00	15000.00	SED-4-R	5462.70	9492798.26	6300.0	6900.0
Aluminum	3	3	13800000.00	21800000.00	SED-4-R	17657486.79	32124467.27	.	.
Arsenic	3	3	11000.00	24000.00	SED-4-R	17117.98	81421.06	.	.
Barium	3	3	72300.00	113000.00	SED-4-R	90624.38	160241.55	.	.
Beryllium	3	3	630.00	910.00	SED-4-R	751.40	1158.72	.	.
Calcium	3	3	35600000.00	48200000.00	SED-4-R	39929173.00	57828567.31	.	.
Chromium, total	3	3	17400.00	28000.00	SED-4-R	22665.66	43202.53	.	.
Cobalt	3	3	7400.00	11100.00	SED-4-R	8693.91	14911.11	.	.
Copper	3	3	12500.00	31900.00	SED-4-R	18999.54	171052.45	.	.
Iron	3	3	18600000.00	27300000.00	SED-4-R	21940489.30	35344605.23	.	.
Lead	3	3	21000.00	44000.00	SED-4-R	30264.33	115619.32	.	.
Magnesium	3	3	10200000.00	15400000.00	SED-4-R	12178848.96	20642754.73	.	.
Manganese	3	3	313000.00	645000.00	SED-4-R	431875.91	1628191.38	.	.
Mercury	2	3	80.00	320.00	SED-3-R	100.79	5512793.36	80.0	80.0
Nickel	3	3	13700.00	27600.00	SED-4-R	19628.47	65979.81	.	.

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

units = ppb, except pesticides/PCBs: units = ppt

TYPE=Northern Drainage Swale Sediments

(continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Potassium	3	3	3060000.00	5980000.00	SED-4-R	3899322.96	15052443.66	.	.
Sodium	3	3	265000.00	669000.00	SED-4-R	403659.61	3392632.69	.	.
Vanadium	3	3	27400.00	39100.00	SED-4-R	32853.76	49623.53	.	.
Zinc	3	3	66800.00	124000.00	SED-4-R	83860.87	264104.38	.	.

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

units = ppb, except pesticides/PCBs: units = ppt

TYPE=Northern Drainage Swale Surface Water

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
1,1,1-Trichloroethane	1	2	2.00	2.00	SW-4-R	2.24	2.00	5.0	5.0
Toluene	1	2	2.00	2.00	SW-4-R	2.24	2.00	5.0	5.0
Phenol	1	2	11.00	11.00	SW-4-R	7.42	11.00	10.0	10.0
bis(2-Ethylhexyl)phthalate	1	2	3.00	3.00	SW-5-R	3.87	3.00	10.0	10.0
Delta-BHC	1	2	21.00	21.00	SW-4-R	10.14	21.00	9.8	9.8
4,4-DDT	1	2	40.00	40.00	SW-4-R	20.00	40.00	20.0	20.0
Aluminum	2	2	666.00	861.00	SW-4-R	757.25	861.00	.	.
Barium	2	2	66.00	96.00	SW-4-R	79.60	96.00	.	.
Calcium	2	2	81800.00	94600.00	SW-4-R	87967.49	94600.00	.	.
Iron	2	2	488.00	1550.00	SW-4-R	869.71	1550.00	.	.
Lead	2	2	8.80	9.90	SW-4-R	9.33	9.90	.	.
Magnesium	2	2	34300.00	50100.00	SW-4-R	41453.95	50100.00	.	.
Manganese	2	2	27.00	48.00	SW-4-R	36.00	48.00	.	.
Potassium	2	2	12700.00	29100.00	SW-4-R	19224.20	29100.00	.	.
Sodium	2	2	47700.00	150000.00	SW-4-R	84587.23	150000.00	.	.
Cyanide	1	2	19.30	19.30	SW-5-R	9.82	19.30	10.0	10.0

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

units = ppb, except pesticides/PCBs: units = ppt

TYPE=Southern Drainage Swale Sediments

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Methylene Chloride	13	15	15.00	73.00	SED-13-R	27.93	45.06	26.5	28.0
Acetone	8	15	12.00	89.00	SED-11-R	18.75	53.03	15.0	61.0
1,1-Dichloroethane	1	15	19.00	19.00	SED-14-R	4.73	6.43	7.0	11.0
1,1,1-Trichloroethane	2	15	2.00	3.00	SED-7-R	4.03	4.75	7.0	11.0
Benzene	1	15	3.00	3.00	SED-14-R	4.18	4.65	7.0	11.0
Phenanthrene	3	15	83.00	180.00	SED-10-R	551.39	1255.65	980.0	2100.0
Di-n-butylphthalate	1	15	140.00	140.00	SED-12-R	712.20	1066.89	980.0	2100.0
Fluoranthene	4	15	150.00	330.00	SED-10-R	588.68	1042.05	1100.0	2100.0
Pyrene	4	15	170.00	310.00	SED-10-R	596.98	1002.74	1100.0	2100.0
Benzo(a)anthracene	2	15	210.00	210.00	SED-10-R	659.78	998.66	980.0	2100.0
Chrysene	3	15	120.00	270.00	SED-8-R	599.08	1065.20	980.0	2100.0
bis(2-Ethylhexyl)phthalate	8	15	110.00	3900.00	SED-18-R	645.50	1398.63	760.0	2100.0
Di-n-octylphthalate	1	15	290.00	290.00	SED-15-R	750.48	951.57	980.0	2100.0
Benzo(b)fluoranthene	2	15	270.00	320.00	SED-8-R	690.03	929.74	980.0	2100.0
Benzo(k)fluoranthene	2	15	220.00	250.00	SED-8-R	669.57	972.94	980.0	2100.0
Benzo(a)pyrene	1	15	250.00	250.00	SED-8-R	743.09	969.76	980.0	2100.0
Benzo(g,h,i)perylene	1	15	230.00	230.00	SED-8-R	1433.25	3608.62	980.0	6300.0
Delta-BHC	6	15	1700.00	4400.00	SED-10-R	3939.00	11219.79	2500.0	36000.0
Gamma-BHC	2	15	920.00	1500.00	SED-17-R	2792.05	13201.16	1700.0	36000.0
Aldrin	2	15	1100.00	2000.00	SED-10-R	2765.73	13836.36	1500.0	36000.0
Heptachlor epoxide	3	15	300.00	3100.00	SED-17-R	2697.63	16808.65	1700.0	36000.0
Dieldrin	3	15	1900.00	2250.00	SED-11-R	3352.52	20432.06	1600.0	54000.0
4,4-DDE	3	15	1100.00	28000.00	SED-17-R	3771.67	33767.45	1700.0	54000.0
Endrin	2	15	7600.00	18000.00	SED-10-R	3995.08	33472.08	1500.0	54000.0
Endosulfan II	2	15	2700.00	4250.00	SED-7-R	7549.60	31553.76	4700.0	94000.0
4,4-DDD	1	15	4700.00	4700.00	SED-13-R	3049.38	24630.02	1500.0	54000.0

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

units = ppb, except pesticides/PCBs: units = ppt

TYPE=Southern Drainage Swale Sediments (continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
4,4-DDT	3	15	6900.00	11000.00	SED-17-R	7334.50	28802.44	3500.0	71000.0
Methoxyclor	1	15	16000.00	16000.00	SED-7-R	15323.02	113627.24	7300.0	270000.0
Aluminum	15	15	6480000.00	27800000.00	SED-16-R	16046778.55	20902142.32	.	.
Antimony	1	15	15200.00	15200.00	SED-12SP	6542.23	7752.04	9600.0	15000.0
Arsenic	15	15	710.00	27600.00	SED-10-R	13414.18	42370.39	.	.
Barium	15	15	64500.00	218000.00	SED-9-R	110078.90	131910.16	.	.
Beryllium	15	15	480.00	1300.00	SED-16-R	733.90	863.97	.	.
Cadmium	3	15	840.00	2100.00	SED-18-R	466.82	906.15	480.0	1500.0
Calcium	15	15	5170000.00	115000000.00	SED-18-R	31536878.81	91271289.11	.	.
Chromium, total	15	15	13400.00	34800.00	SED-16-R	21601.23	25794.37	.	.
Cobalt	15	15	4800.00	17700.00	SED-18-R	9025.74	11800.09	.	.
Copper	15	15	10400.00	41950.00	SED-4SP	20065.32	26791.64	.	.
Iron	15	15	8590000.00	69000000.00	SED-9-R	22061698.94	31542978.06	.	.
Lead	15	15	27100.00	100000.00	SED-8-R	46957.13	63174.18	.	.
Magnesium	15	15	4210000.00	48700000.00	SED-18-R	12238364.28	24363077.43	.	.
Manganese	15	15	117000.00	695000.00	SED-8-R	362136.00	564149.72	.	.
Mercury	10	15	80.00	1650.00	SED-4SP	123.69	525.11	60.0	100.0
Nickel	15	15	7400.00	35400.00	SED-16-R	21068.21	27381.10	.	.
Potassium	15	15	1320000.00	6340000.00	SED-16-R	3122718.82	4510312.15	.	.
Selenium	1	14	710.00	710.00	SED-12SP	345.45	419.84	500.0	920.0
Sodium	15	15	240000.00	2260000.00	SED-12SP	600584.46	1155045.72	.	.
Vanadium	15	15	12000.00	49000.00	SED-16-R	28298.15	35712.04	.	.
Zinc	15	15	76600.00	293000.00	SED-11-R	130847.56	174288.46	.	.

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

units = ppb, except pesticides/PCBs: units = ppt

TYPE=Southern Drainage Swale Surface Water

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Carbon Disulfide	3	7	0.65	8.00	SW-18-R	2.14	8.23	5.0	5.0
4-Methyl-2-Pentanone	1	7	2.00	2.00	SW-11-R	4.39	6.35	10.0	10.0
Tetrachloroethylene	1	7	4.00	4.00	SW-18-R	2.67	3.13	5.0	5.0
Toluene	1	7	2.00	2.00	SW-11-R	2.42	2.00	5.0	5.0
Ethylbenzene	1	7	1.00	1.00	SW-13	2.19	3.18	5.0	5.0
Total Xylenes	1	7	3.00	3.00	SW-11SP	2.57	3.00	5.0	5.0
2,4-Dimethylphenol	2	7	3.00	6.50	SW-11-R	4.75	5.92	9.0	10.0
Benzoic Acid	1	7	5.00	5.00	SW-13	19.35	44.31	47.0	51.0
Diethylphthalate	1	7	0.55	0.55	SW-11-R	3.59	15.10	9.0	10.0
Di-n-butylphthalate	2	7	0.30	0.40	SW-11-R	2.30	59.39	9.0	10.0
bis(2-Ethylhexyl)phthalate	6	7	0.70	1000.00	SW-18-R	12.48	427960.85	10.0	10.0
Delta-BHC	1	7	14.00	14.00	SW-13	11.18	45.02	9.5	51.0
Heptachlor epoxide	1	7	14.00	14.00	SW-8-R	11.16	45.20	9.4	51.0
Aluminum	7	7	426.00	25200.00	SW-13	2060.29	749219.12	.	.
Arsenic	3	7	3.10	30.60	SW-13	4.50	92.75	4.0	4.0
Barium	7	7	55.00	456.00	SW-13	159.03	640.62	.	.
Beryllium	1	7	2.10	2.10	SW-8-R	1.01	1.62	1.0	2.0
Cadmium	1	7	5.70	5.70	SW-13	2.72	3.89	4.0	5.0
Calcium	7	7	46900.00	286000.00	SW-13	115911.63	335365.76	.	.
Chromium, total	3	7	26.90	38.00	SW-8-R	10.81	71.98	10.0	10.0
Cobalt	2	7	18.00	25.00	SW-13	7.18	24.53	7.0	10.0
Copper	4	7	21.90	94.00	SW-13	22.99	128.03	20.0	20.0
Iron	7	7	663.00	38000.00	SW-13	4952.30	1224034.92	.	.
Lead	7	7	6.30	352.00	SW-8-R	41.72	10842.95	.	.
Magnesium	7	7	28500.00	211000.00	SW-11SP	69646.35	212291.97	.	.
Manganese	7	7	30.00	1690.00	SW-8-R	155.02	30752.05	.	.

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

units = ppb, except pesticides/PCBs: units = ppt

TYPE=Southern Drainage Swale Surface Water

(continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Nickel	4	7	27.00	63.00	SW-8-R	23.18	99.05	20.0	20.0
Potassium	7	7	7380.00	211000.00	SW-11SP	30310.11	649399.96	.	.
Sodium	7	7	29800.00	393500.00	SW-11-R	107392.85	719145.13	.	.
Thallium	1	7	4.40	4.40	SW-11SP	2.71	3.30	5.0	5.0
Vanadium	2	7	9.10	61.00	SW-13	12.77	36.51	20.0	20.0
Zinc	5	7	24.00	2360.00	SW-13	79.83	141043.34	20.0	20.0
Cyanide	3	7	15.60	40.60	SW-7-R	10.13	57.62	10.0	10.0

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

units = ppb, except pesticides/PCBs: units = ppt

TYPE=Northern Wetland Sediments

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Methylene Chloride	2	2	13.00	45.00	SED-2-R	24.19	45.00	.	.
Acetone	1	2	26.00	26.00	SED-2-R	15.72	26.00	19.0	19.0
Phenanthrene	2	2	40.00	98.00	SED-2-R	62.61	98.00	.	.
Fluoranthene	2	2	62.00	230.00	SED-2-R	119.42	230.00	.	.
Pyrene	2	2	50.00	190.00	SED-2-R	97.47	190.00	.	.
Benzo(a)anthracene	1	2	140.00	140.00	SED-2-R	354.96	140.00	1800.0	1800.0
Chrysene	1	2	150.00	150.00	SED-2-R	367.42	150.00	1800.0	1800.0
bis(2-Ethylhexyl)phthalate	1	2	290.00	290.00	SED-1-R	510.88	290.00	1800.0	1800.0
Benzo(b)fluoranthene	1	2	130.00	130.00	SED-2-R	342.05	130.00	1800.0	1800.0
Benzo(k)fluoranthene	1	2	160.00	160.00	SED-2-R	379.47	160.00	1800.0	1800.0
Benzo(a)pyrene	1	2	140.00	140.00	SED-2-R	354.96	140.00	1800.0	1800.0
4,4-DDT	1	2	77000.00	77000.00	SED-2-R	16878.98	77000.00	7400.0	7400.0
Aluminum	2	2	19200000.00	24900000.00	SED-2-R	21865040.59	24900000.00	.	.
Arsenic	2	2	22000.00	27000.00	SED-2-R	24372.12	27000.00	.	.
Barium	2	2	111000.00	148000.00	SED-2-R	128171.76	148000.00	.	.
Beryllium	2	2	1000.00	1100.00	SED-2-R	1048.81	1100.00	.	.
Calcium	2	2	8790000.00	47200000.00	SED-1-R	20368799.67	47200000.00	.	.
Chromium, total	2	2	26700.00	30800.00	SED-2-R	28676.82	30800.00	.	.
Cobalt	2	2	10300.00	12700.00	SED-1-R	11437.22	12700.00	.	.
Copper	2	2	24400.00	35500.00	SED-1-R	29431.28	35500.00	.	.
Iron	2	2	22500000.00	30100000.00	SED-1-R	26024027.36	30100000.00	.	.
Lead	2	2	35000.00	49000.00	SED-1-R	41412.56	49000.00	.	.
Magnesium	2	2	6140000.00	16000000.00	SED-1-R	9911609.35	16000000.00	.	.
Manganese	2	2	187000.00	551000.00	SED-1-R	320993.77	551000.00	.	.
Merc(	1	2	560.00	[illegible] 00	SED-1-R	149.67	560.00	80.0	80.0
Nickel	2	2	30000.00	30900.00	SED-2-R	30446.67	30900.00	.	.

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

units = ppb, except pesticides/PCBs: units = ppt

TYPE=Northern Wetland Sediments

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Potassium	2	2	2360000.00	4080000.00	SED-1-R	3103030.78	4080000.00	.	.
Sodium	2	2	361000.00	574000.00	SED-1-R	455207.64	574000.00	.	.
Vanadium	2	2	33200.00	40500.00	SED-2-R	36668.79	40500.00	.	.
Zinc	2	2	99500.00	133000.00	SED-1-R	115036.95	133000.00	.	.

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

units = ppb, except pesticides/PCBs: units = ppt

TYPE=Northern Wetland Surface Water

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
bis(2-Ethylhexyl)phthalate	1	2	10.00	10.00	SW-1-R	7.07	10.00	10.0	10.0
Gamma-BHC	1	2	5.10	5.10	SW-2-R	4.90	5.10	9.4	9.4
Aluminum	2	2	589.00	2570.00	SW-2-R	1230.34	2570.00	.	.
Arsenic	1	2	4.70	4.70	SW-2-R	3.07	4.70	4.0	4.0
Barium	2	2	89.00	147.00	SW-2-R	114.38	147.00	.	.
Calcium	2	2	134000.00	262000.00	SW-2-R	187371.29	262000.00	.	.
Iron	2	2	752.00	3400.00	SW-2-R	1599.00	3400.00	.	.
Lead	2	2	6.10	20.00	SW-2-R	11.05	20.00	.	.
Magnesium	2	2	36900.00	64300.00	SW-2-R	48710.06	64300.00	.	.
Manganese	2	2	79.00	783.00	SW-2-R	248.71	783.00	.	.
Potassium	2	2	5850.00	9290.00	SW-1-R	7372.01	9290.00	.	.
Sodium	2	2	59400.00	60700.00	SW-2-R	60046.48	60700.00	.	.
Zinc	1	2	76.00	76.00	SW-2-R	27.57	76.00	20.0	20.0

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Landfill Leachate - Soil

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Methylene Chloride	1	2	67.00	67.00	SEEP-1-R	22.42	67.00	15.00	15.0
1,4-Dichlorobenzene (para)	1	2	1200.00	1200.00	SEEP-3-R	848.53	1200.00	1200.00	1200.0
4-Methylphenol	1	2	440.00	440.00	SEEP-3-R	513.81	440.00	1200.00	1200.0
Naphthalene	1	2	240.00	240.00	SEEP-3-R	379.47	240.00	1200.00	1200.0
2-Methylnaphthalene	1	2	120.00	120.00	SEEP-3-R	268.33	120.00	1200.00	1200.0
Acenaphthene	1	2	100.00	100.00	SEEP-3-R	244.95	100.00	1200.00	1200.0
Phenanthrene	1	2	470.00	470.00	SEEP-3-R	531.04	470.00	1200.00	1200.0
Anthracene	1	2	190.00	190.00	SEEP-3-R	337.64	190.00	1200.00	1200.0
Di-n-butylphthalate	2	2	81.00	350.00	SEEP-3-R	168.37	350.00	.	.
Fluoranthene	1	2	480.00	480.00	SEEP-3-R	536.66	480.00	1200.00	1200.0
Pyrene	1	2	840.00	840.00	SEEP-3-R	709.93	840.00	1200.00	1200.0
Benzylbutylphthalate	1	2	1200.00	1200.00	SEEP-3-R	848.53	1200.00	1200.00	1200.0
Benzo(a)anthracene	1	2	210.00	210.00	SEEP-3-R	354.96	210.00	1200.00	1200.0
Chrysene	1	2	200.00	200.00	SEEP-3-R	346.41	200.00	1200.00	1200.0
bis(2-Ethylhexyl)phthalate	1	2	750.00	750.00	SEEP-3-R	670.82	750.00	1200.00	1200.0
Delta-BHC	1	2	2100.00	2100.00	SEEP-1-R	3694.59	2100.00	13000.00	13000.0
Aldrin	1	2	2100.00	2100.00	SEEP-1-R	3694.59	2100.00	13000.00	13000.0
Dieldrin	1	2	3200.00	3200.00	SEEP-1-R	4560.70	3200.00	13000.00	13000.0
4,4-DDE	1	2	690.00	690.00	SEEP-1-R	2117.78	690.00	13000.00	13000.0
gamma-chlordane	1	2	1200.00	1200.00	SEEP-1-R	2792.85	1200.00	13000.00	13000.0
Aluminum	2	2	7810000.00	11700000.00	SEEP-3-R	9559131.76	11700000.00	.	.
Arsenic	2	2	7000.00	12000.00	SEEP-3-R	9165.15	12000.00	.	.
Barium	2	2	110000.00	110000.00	SEEP-1-R	110000.00	110000.00	.	.
Beryllium	2	2	290.00	600.00	SEEP-3-R	417.13	600.00	.	.
Cadmium	1	2	710.00	710.00	SEEP-3-R	386.13	710.00	420.00	420.0
Calcium	2	2	78600000.00	81400000.00	SEEP-1-R	79987749.06	81400000.00	.	.
Chromium, total	2	2	17500.00	18100.00	SEEP-1-R	17797.47	18100.00	.	.

SUMMARY STATISTICS FOR THE ECOLOGICAL RISK ASSESSEMENT AT THE NCR SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA

all in units of parts per billion, except pesticides/PCBs which are in units of parts per trillion

TYPE=Landfill Leachate - Soil

(continued)

Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Geom. Mean Conc.	95 Pct. Upp. Conf. Limit	Min. Detect. Limit	Max. Detect. Limit
Cobalt	2	2	4700.00	6800.00	SEEP-3-R	5653.32	6800.00	.	.
Copper	2	2	29400.00	47800.00	SEEP-3-R	37487.60	47800.00	.	.
Iron	2	2	23400000.00	25700000.00	SEEP-3-R	24523050.38	25700000.00	.	.
Lead	2	2	40000.00	110000.00	SEEP-1-R	66332.50	110000.00	.	.
Magnesium	2	2	29700000.00	31300000.00	SEEP-1-R	30489506.39	31300000.00	.	.
Manganese	2	2	441000.00	511000.00	SEEP-3-R	474711.49	511000.00	.	.
Mercury	2	2	390.00	1200.00	SEEP-3-R	684.11	1200.00	.	.
Nickel	2	2	16100.00	18700.00	SEEP-3-R	17351.37	18700.00	.	.
Potassium	2	2	1390000.00	2890000.00	SEEP-3-R	2004270.44	2890000.00	.	.
Sodium	2	2	365000.00	394000.00	SEEP-1-R	379222.89	394000.00	.	.
Vanadium	2	2	16300.00	23100.00	SEEP-3-R	19404.38	23100.00	.	.
Zinc	2	2	102000.00	119000.00	SEEP-3-R	110172.59	119000.00	.	.

APPENDIX C

TOXICITY VALUES FOR ALL THE CONTAMINANTS DETECTED AT THE NCR SITE

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TOXICITY VALUES FOR ALL THE CONTAMINANTS DETECTED AT THE NCR SITE.

CHEMICAL	CARCINOGENIC				CHRONIC		SUBCHRONIC	
	Weight of Evidence Classification		Oral Slope Factor (mg/kg/day)-1		Chronic Oral RfD (mg/kg/day)		Subchronic Oral RfD (mg/kg/day)	
Volatiles								
Acetone	D	a			1.00E-01	a	1.00E+00	b
Benzene	A	a	2.90E-02	a				
2-Butanone (MEK)	D	a			5.00E-02	b	5.00E-01	b
Carbon disulfide	--	a			1.00E-01	a	1.00E-01	b
Chlorobenzene	D	a			2.00E-02	a	2.00E-01	b
Chloroform	B2	a	6.10E-03	a	1.00E-02	a	1.00E-02	b
1,2 Dichlorobenzene (ortho)	D	a			9.00E-02	a	9.00E-01	b
1,3 Dichlorobenzene (meta)	D	a			3.00E-02	d	9.00E-02	o
1,4 Dichlorobenzene (para)	C	b	2.40E-02	b	1.00E-01	d	1.00E-01	j
1,1-Dichloroethane	C	a			1.00E-01	b	1.00E+00	b
1,2-Dichloroethylene (total)	--				1.00E-02	k	1.00E-01	k
cis-1,2-Dichloroethylene	D	a			1.00E-02	b	1.00E-01	b
Ethylbenzene	D	a			1.00E-01	a	1.00E+00	b
2-Hexanone (MBK)	--	a			4.00E-02	d		
Isopropylbenzene	--							
Methylene chloride	B2	a	7.50E-03	a	6.00E-02	a	6.00E-02	b
4-Methyl-2-pentanone	--				5.00E-02	b	5.00E-01	b
Styrene	B2	b	3.00E-02	b	2.00E-01	a	2.00E+00	b
Tetrachloroethylene	B2	b	5.10E-02	b	1.00E-02	a	1.00E-01	b
Toluene	D	a			2.00E-01	b	2.00E+00	b
1,1,1-Trichloroethane	D	a			9.00E-02	b	9.00E-01	b
Trichloroethylene	B2	b	1.10E-02	b	6.00E-03	d	6.00E-03	j
1,2,4 Trimethylbenzene	D	o			6.00E-04	d	6.00E-04	j
1,3,5 Trimethylbenzene	D	o			4.00E-04	d	4.00E-04	j
Vinyl chloride (chloroethylene)	A	b	1.90E+00	b				
Xylenes	D	a			2.00E+00	a	4.00E+00	b
BNAs								
Acenaphthene	--	a			6.00E-02	a	6.00E-01	b
Anthracene	D	a			3.00E-01	a	3.00E+00	b
Benzo(a)anthracene	B2	a	5.79E-01	e				
Benzo(a)pyrene	B2	a	5.79E+00	a				
Benzo(b)fluoranthene	B2	a	5.79E-01	e				
Benzo(g,h,i)perylene	D	a						
Benzoic acid	D	a			4.00E+00	a	4.00E+00	b
Benzo(k)fluoranthene	B2	a	5.79E-01	e				
Benzylbutylphthalate	C	a			2.00E-01	a	2.00E+00	b
Bis(2-ethylhexyl)phthalate	B2	a	1.40E-02	a	2.00E-02	a	2.00E-02	b
4-Chloroaniline	--				4.00E-03	a	4.00E-03	b
Chrysene	B2	a	5.79E-02	e				
Diethylphthalate	D	a			8.00E-01	a	8.00E+00	b
2,4-Dimethylphenol					2.00E-02	b	2.00E-01	b
Di-n-butyl phthalate	D	a			1.00E-01	a	1.00E+00	b

TOXICITY VALUES FOR ALL THE CONTAMINANTS DETECTED AT THE NCR SITE. (cont.).

CHEMICAL	CARCINOGENIC		CHRONIC	SUBCHRONIC
	Weight of Evidence Classification	Oral Slope Factor (mg/kg/day)-1	Chronic Oral RfD (mg/kg/day)	Subchronic Oral RfD (mg/kg/day)
Di-n-octyl phthalate	--		2.00E-02 b	2.00E-02 j
2,6-Dinitrotoluene	B2 b	6.80E-01 b, m		
Fluoranthene	D a		4.00E-02 a	4.00E-01 b
Fluorene	D a		4.00E-02 a	4.00E-01 b
2-Methylnaphthalene	--			
2-Methylphenol (o-cresol)	-- b		5.00E-02 a	5.00E-01 b
4-Methylphenol (p-cresol)	C a		5.00E-02 b	5.00E-01 b
Naphthalene	D a		4.00E-03 b	4.00E-02 b
N-Nitrosodiphenylamine	B2 a	4.90E-03 a		
Pentachlorophenol	B2 a	1.20E-01 a	3.00E-02 a	3.00E-02 b
Phenanthrene	D a			
Phenol	D a		6.00E-01 a	6.00E-01 b
Pyrene	D a		3.00E-02 a	3.00E-01 b
2,4,5-Trichlorophenol	--		1.00E-01 a	1.00E+00 b
Pesticides				
Aldrin	B2 a	1.70E+01 a	3.00E-05 a	3.00E-05 b
Aroclor - 1254	B2 a	7.70E+00 a		
alpha-BHC	B2 a	6.30E+00 a		
beta-BHC	C a	1.80E+00 a		
delta-BHC	--			
gamma-BHC	B2-C b	1.30E+00 b	3.00E-04 a	3.00E-03 b
alpha-Chlordane (Chlordane)	B2 a	1.30E+00 a	6.00E-05 a	6.00E-06 b
gamma-Chlordane (Chlordane)	B2 a	1.30E+00 a	6.00E-05 a	6.00E-06 b
4,4' DDD	B2 a	2.40E-01 a		
4,4' DDE	B2 a	3.40E-01 a		
4,4' DDT	B2 a	3.40E-01 a	5.00E-04 a	5.00E-04 b
Dieldrin	B2 a	1.60E+01 a	5.00E-05 a	5.00E-05 b
Endosulfan II (n)	--		5.00E-05 a	
Endosulfan Sulfate	--			
Endrin	D a		3.00E-04 a	5.00E-04 b
Endrin aldehyde	--			
Endrin ketone	--			
Heptachlor	B2 a	4.50E+00 a	5.00E-04 a	5.00E-04 b
Heptachlor epoxide	B2 a	9.10E+00 a	1.30E-05 a	1.30E-05 j
Methoxyclor	--		5.00E-03 a	5.00E-03 b
Inorganics				
Aluminum	D d		1.00E+00 d	1.00E+00 j
Antimony	-- a		4.00E-04 a	4.00E-04 b
Arsenic	A a	1.75E+00 f	3.00E-04 a	1.00E-03 b
Barium	-- a		5.00E-02 b	5.00E-02 b
Beryllium	B2 a	4.30E+00 a	5.00E-03 a	5.00E-03 b
Cadmium (l)	B1 a		5.00E-04 a, g	5.00E-04 j
Calcium	--			

TOXICITY VALUES FOR ALL THE CONTAMINANTS DETECTED AT THE NCR SITE. (cont.).

CHEMICAL	CARCINOGENIC			CHRONIC		SUBCHRONIC	
	Weight of Evidence Classification		Oral Slope Factor (mg/kg/day)-1	Chronic Oral RfD (mg/kg/day)		Subchronic Oral RfD (mg/kg/day)	
Chromium, total	--			8.76E-01	i	8.75E+00	i
Chromium, III	--	a		1.00E+00	a	1.00E+01	b
Chromium, VI	A	a		5.00E-03	a	2.00E-02	b
Cobalt	--				d		
Copper	D	c		4.00E-02	d	4.00E-02	j
Cyanide	D	a		2.00E-02	a	2.00E-02	b
Iron	D	d		5.00E-01	d	5.00E-01	j
Lead	B2	a					
Magnesium	--						
Manganese	D	a		1.00E-01	a	1.00E-01	b
Mercury	D	a		3.00E-04	b	3.00E-04	b
Nickel	A	a		2.00E-02	a, h	2.00E-02	b
Potassium	--						
Selenium	D	a		5.00E-03	a	5.00E-03	j
Silver	D	a		5.00E-03	a	3.00E-03	b
Sodium	--						
Thallium	--			7.00E-05	b	7.00E-04	b
Vanadium	D	c		7.00E-03	b	7.00E-03	b
Zinc	D	a		2.00E-01	b	2.00E-01	b

a. From Integrated Risk Information System (IRIS) 5/1/92.
b. From Health Effects Assessment Summary Tables (HEAST) FY 1991.
c. From Drinking Water Regulations and Health Advisories, April 1992.
d. Interim value from ECAO. See text for specific reference.
e. Oral slope factor for B(a)P used for PAHs classified as B2 carcinogens with the following TEFs applied:

Benzo(a)Anthracene	0.1
Benzo(b)Fluoranthene	0.1
Benzo(k)Fluoranthene	0.1
Chrysene	0.01

f. Arsenic oral slope factor derived from unit risk in IRIS.
g. Cadmium RfD is for water; 1.0E-03 mg/kg/day is RfD for food.
h. Value is for nickel soluble salts.
i. Per EPA guidance, value is weighted-average value of the trivalent chromium and hexavalent chromium RfDs, assuming 7 parts tri to 1 part hex.
j. Chronic RfD used as Subchronic RfD if no Subchronic value is available per RAGS.
k. Toxicity values are for the cis isomer.
l. Dermal toxicity values for cadmium have been derived from oral toxicity values applying an absorption factor of 0.01 (10%) per EPA guidance (see text for specific reference).
The RfD for both chronic and subchronic dermal exposure is 5.00E-05 mg/kg/day.
m. Value used applies to mixture of 2,4- and 2,6-dinitrotoluene.
n. Value used is for Endosulfan.
o. Carcinogenic Weight of Evidence Classification obtained from Health Effects Assessment document, not IRIS or HEAST.

APPENDIX D

RESULTS OF THE TOXICITY SCREEN FOR THE NCR SITE

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE.

Ranked Noncarcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Surface Soils

Analyte Detected	Highest Detected Conc.	Oral RfD	Noncarc. Tox. Factor	Noncarcinogenic Ratio
Arsenic	2.80E+01	3.00E-04	9.33E+04	0.47126
Iron	3.10E+04	5.00E-01	6.20E+04	0.31305
Aluminum	2.60E+04	1.00E+00	2.60E+04	0.13128
Manganese	5.75E+02	1.00E-01	5.75E+03	0.02903
Vanadium	3.70E+01	7.00E-03	5.29E+03	0.02669
Barium	1.35E+02	5.00E-02	2.70E+03	0.01363
Nickel	2.60E+01	2.00E-02	1.30E+03	0.00656
Copper	3.20E+01	4.00E-02	8.00E+02	0.00404
Zinc	1.05E+02	2.00E-01	5.25E+02	0.00265
Beryllium	1.10E+00	5.00E-03	2.20E+02	0.00111
bis(2-Ethylhexyl)phthalate	1.02E+00	2.00E-02	5.12E+01	0.00026
1,2-Dichloroethylene (total)	4.40E-01	1.00E-02	4.40E+01	0.00022
Chromium, total	3.10E+01	8.76E-01	3.54E+01	0.00018
Trichloroethylene	3.20E-02	6.00E-03	5.33E+00	0.00003
Methylene Chloride	2.20E-02	6.00E-02	3.67E-01	0.00000
Acetone	1.70E-02	1.00E-01	1.70E-01	0.00000
1,1,1-Trichloroethane	6.10E-03	9.00E-02	6.78E-02	0.00000
Styrene	1.40E-03	2.00E-01	7.00E-03	0.00000
Vinyl Chloride	2.40E-01	.	.	.
Lead	1.75E+02	.	.	.
Magnesium	2.55E+04	.	.	.
Potassium	6.30E+03	.	.	.
Sodium	6.90E+02	.	.	.
Calcium	6.89E+04	.	.	.
Delta-BHC	1.40E-03	.	.	.
				1.00000

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RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Noncarcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Subsurface Soils

Analyte Detected	Highest Detected Conc.	Oral RfD	Noncarc. Tox. Factor	Noncarcinogenic Ratio
Arsenic	2.60E+01	3.00E-04	8.67E+04	0.35271
Iron	3.10E+04	5.00E-01	6.20E+04	0.25233
Antimony	1.30E+01	4.00E-04	3.25E+04	0.13227
Aluminum	2.50E+04	1.00E+00	2.50E+04	0.10174
Manganese	1.30E+03	1.00E-01	1.30E+04	0.05291
Thallium	6.10E-01	7.00E-05	8.71E+03	0.03547
Copper	2.42E+02	4.00E-02	6.05E+03	0.02462
Vanadium	3.60E+01	7.00E-03	5.14E+03	0.02093
Barium	1.60E+02	5.00E-02	3.20E+03	0.01302
Nickel	3.00E+01	2.00E-02	1.50E+03	0.00610
Mercury	2.30E-01	3.00E-04	7.67E+02	0.00312
Zinc	1.35E+02	2.00E-01	6.75E+02	0.00275
Beryllium	9.60E-01	5.00E-03	1.92E+02	0.00078
Selenium	8.20E-01	5.00E-03	1.64E+02	0.00067
Heptachlor epoxide	5.80E-04	1.30E-05	4.46E+01	0.00018
Chromium, total	3.00E+01	8.76E-01	3.43E+01	0.00014
1,2-Dichloroethylene (total)	3.20E-01	1.00E-02	3.20E+01	0.00013
Naphthalene	4.30E-02	4.00E-03	1.07E+01	0.00004
bis(2-Ethylhexyl)phthalate	1.60E-01	2.00E-02	8.00E+00	0.00003
Di-n-octylphthalate	5.20E-02	2.00E-02	2.60E+00	0.00001
Trichloroethylene	1.50E-02	6.00E-03	2.50E+00	0.00001
Pyrene	6.70E-02	3.00E-02	2.23E+00	0.00001
Fluoranthene	8.60E-02	4.00E-02	2.15E+00	0.00001
4-Methylphenol	6.80E-02	5.00E-02	1.36E+00	0.00001
Acetone	5.80E-02	1.00E-01	5.80E-01	0.00000
2,4,5-Trichlorophenol	5.00E-02	1.00E-01	5.00E-01	0.00000
Methylene Chloride	1.70E-02	6.00E-02	2.83E-01	0.00000
1,1,1-Trichloroethane	5.70E-03	9.00E-02	6.33E-02	0.00000
Benzoic Acid	2.20E-01	4.00E+00	5.50E-02	0.00000
Vinyl Chloride	2.10E-01	.	.	.
Chrysene	4.60E-02	.	.	.
Alpha-BHC	2.60E-04	.	.	.
Phenanthrene	6.00E-02	.	.	.
Lead	2.05E+01	.	.	.
Magnesium	3.90E+04	.	.	.
Potassium	6.00E+03	.	.	.
Sodium	9.20E+02	.	.	.
Calcium	9.00E+04	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Noncarcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Northern Landfill Cell Ground Water

Analyte Detected	Highest Detected Conc.	Oral RfD	Noncarc. Tox. Factor	Noncarcinogenic Ratio
Antimony	4.48E-02	4.00E-04	1.12E+02	0.72545
Arsenic	2.50E-03	3.00E-04	8.33E+00	0.05404
4-Methylphenol	2.45E-01	5.00E-02	4.90E+00	0.03177
Phenol	2.65E+00	6.00E-01	4.42E+00	0.02864
2-Methylphenol	1.75E-01	5.00E-02	3.50E+00	0.02270
Acetone	3.20E-01	1.00E-01	3.20E+00	0.02075
Vanadium	1.52E-02	7.00E-03	2.18E+00	0.01413
Barium	9.76E-02	5.00E-02	1.95E+00	0.01266
1,2,4-Trimethylbenzene	9.50E-04	6.00E-04	1.58E+00	0.01027
Trichloroethylene	9.50E-03	6.00E-03	1.58E+00	0.01027
2,4-Dimethylphenol	2.70E-02	2.00E-02	1.35E+00	0.00875
Iron	6.55E-01	5.00E-01	1.31E+00	0.00850
2-Butanone (MEK)	6.05E-02	5.00E-02	1.21E+00	0.00785
Nickel	2.28E-02	2.00E-02	1.14E+00	0.00739
Silver	4.25E-03	5.00E-03	8.50E-01	0.00551
Copper	3.10E-02	4.00E-02	7.75E-01	0.00503
1,3,5-Trimethylbenzene	3.00E-04	4.00E-04	7.50E-01	0.00486
Chloroform	5.50E-03	1.00E-02	5.50E-01	0.00357
Styrene	6.70E-02	2.00E-01	3.35E-01	0.00217
Toluene	4.95E-02	2.00E-01	2.48E-01	0.00160
Manganese	2.43E-02	1.00E-01	2.42E-01	0.00157
Aluminum	2.24E-01	1.00E+00	2.24E-01	0.00145
Methylene Chloride	1.25E-02	6.00E-02	2.08E-01	0.00135
Beryllium	1.00E-03	5.00E-03	2.00E-01	0.00130
Naphthalene	8.00E-04	4.00E-03	2.00E-01	0.00130
Selenium	1.00E-03	5.00E-03	2.00E-01	0.00130
bis(2-Ethylhexyl)phthalate	3.50E-03	2.00E-02	1.75E-01	0.00113
4-Methyl-2-Pentanone	6.50E-03	5.00E-02	1.30E-01	0.00084
Tetrachloroethylene	1.00E-03	1.00E-02	1.00E-01	0.00065
2-Hexanone (MBK)	4.00E-03	4.00E-02	1.00E-01	0.00065
Ethylbenzene	9.50E-03	1.00E-01	9.50E-02	0.00062
1,4-Dichlorobenzene (para)	8.00E-03	1.00E-01	8.00E-02	0.00052
Zinc	1.57E-02	2.00E-01	7.85E-02	0.00051
Chromium, total	3.29E-02	8.76E-01	3.75E-02	0.00024
cis-1,2-Dichloroethylene	3.50E-04	1.00E-02	3.50E-02	0.00023
Di-n-butylphthalate	2.00E-03	1.00E-01	2.00E-02	0.00013
Heptachlor	6.70E-06	5.00E-04	1.34E-02	0.00009
Total Xylenes	2.60E-02	2.00E+00	1.30E-02	0.00008
Carbon Disulfide	1.00E-03	1.00E-01	1.00E-02	0.00006
1,1-Dichloroethane	1.00E-03	1.00E-01	1.00E-02	0.00006
Gamma-BHC	4.10E-07	3.00E-04	1.37E-03	0.00001
Benzene	5.00E-03	.	.	.
Magnesium	9.71E+01	.	.	.
Potassium	1.17E+02	.	.	.
Sodium	8.30E+01	.	.	.
Calcium	5.11E+02	.	.	.
Endosulfan sulfate	6.90E-07	.	.	.
Isopropylbenzene	3.00E-04	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Noncarcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Landfill Perimeter Ground Water

Analyte Detected	Highest Detected Conc.	Oral RfD	Noncarc. Tox. Factor	Noncarcinogenic Ratio
Iron	1.08E+02	5.00E-01	2.16E+02	0.34283
Antimony	6.98E-02	4.00E-04	1.74E+02	0.27617
Aluminum	8.08E+01	1.00E+00	8.08E+01	0.12824
Arsenic	1.64E-02	3.00E-04	5.47E+01	0.08682
Manganese	3.93E+00	1.00E-01	3.93E+01	0.06238
Vanadium	1.50E-01	7.00E-03	2.14E+01	0.03397
Cadmium	5.00E-03	5.00E-04	1.00E+01	0.01587
Barium	4.31E-01	5.00E-02	8.62E+00	0.01368
Nickel	1.55E-01	2.00E-02	7.75E+00	0.01230
Mercury	1.80E-03	3.00E-04	6.00E+00	0.00952
Copper	1.27E-01	4.00E-02	3.18E+00	0.00505
Zinc	5.08E-01	2.00E-01	2.54E+00	0.00403
4,4-DDT	6.70E-04	5.00E-04	1.34E+00	0.00213
Silver	6.50E-03	5.00E-03	1.30E+00	0.00206
bis(2-Ethylhexyl)phthalate	2.30E-02	2.00E-02	1.15E+00	0.00183
Beryllium	3.10E-03	5.00E-03	6.20E-01	0.00098
Acetone	2.70E-02	1.00E-01	2.70E-01	0.00043
gamma-chlordane	1.50E-05	6.00E-05	2.50E-01	0.00040
Heptachlor	9.00E-05	5.00E-04	1.80E-01	0.00029
Chromium, total	1.34E-01	8.76E-01	1.53E-01	0.00024
Pentachlorophenol	3.00E-03	3.00E-02	1.00E-01	0.00016
Chloroform	1.00E-03	1.00E-02	1.00E-01	0.00016
Methylene Chloride	4.00E-03	6.00E-02	6.67E-02	0.00011
Heptachlor epoxide	8.60E-07	1.30E-05	6.62E-02	0.00011
alpha-chlordane	2.20E-06	6.00E-05	3.67E-02	0.00006
Aldrin	8.90E-07	3.00E-05	2.97E-02	0.00005
Toluene	5.00E-03	2.00E-01	2.50E-02	0.00004
Benzylbutylphthalate	4.00E-03	2.00E-01	2.00E-02	0.00003
Dieldrin	5.80E-07	5.00E-05	1.16E-02	0.00002
Gamma-BHC	3.00E-06	3.00E-04	1.00E-02	0.00002
Di-n-butylphthalate	1.00E-03	1.00E-01	1.00E-02	0.00002
Ethylbenzene	1.00E-03	1.00E-01	1.00E-02	0.00002
Phenol	4.00E-03	6.00E-01	6.67E-03	0.00001
Total Xylenes	8.00E-03	2.00E+00	4.00E-03	0.00001
Diethylphthalate	2.00E-03	8.00E-01	2.50E-03	0.00000
Methoxyclor	7.80E-06	5.00E-03	1.56E-03	0.00000
4,4-DDE	5.70E-04	.	.	.
Beta-BHC	4.90E-05	.	.	.
4,4-DDD	2.40E-04	.	.	.
Benzene	1.00E-03	.	.	.
Alpha-BHC	6.50E-07	.	.	.
Lead	7.79E-02	.	.	.
Magnesium	3.40E+02	.	.	.
Potassium	2.43E+01	.	.	.
Sodium	3.61E+03	.	.	.
Calcium	5.77E+02	.	.	.
Endosulfan sulfate	5.60E-05	.	.	.
Endrin ketone	2.20E-05	.	.	.
Endrin aldehyde	7.60E-06	.	.	.
Delta-BHC	1.20E-06	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Noncarcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Surface Water

Analyte Detected	Highest Detected Conc.	Oral RfD	Noncarc. Tox. Factor	Noncarcinogenic Ratio
Arsenic	3.06E-02	3.00E-04	1.02E+02	0.26558
Iron	3.80E+01	5.00E-01	7.60E+01	0.19788
Thallium	4.40E-03	7.00E-05	6.29E+01	0.16366
bis(2-Ethylhexyl)phthalate	1.00E+00	2.00E-02	5.00E+01	0.13018
Aluminum	2.52E+01	1.00E+00	2.52E+01	0.06561
Manganese	1.69E+00	1.00E-01	1.69E+01	0.04400
Zinc	2.36E+00	2.00E-01	1.18E+01	0.03072
Cadmium	5.70E-03	5.00E-04	1.14E+01	0.02968
Barium	4.56E-01	5.00E-02	9.12E+00	0.02375
Vanadium	6.10E-02	7.00E-03	8.71E+00	0.02269
Nickel	6.30E-02	2.00E-02	3.15E+00	0.00820
Copper	9.40E-02	4.00E-02	2.35E+00	0.00612
Cyanide	4.06E-02	2.00E-02	2.03E+00	0.00529
Heptachlor epoxide	1.40E-05	1.30E-05	1.08E+00	0.00280
Beryllium	2.10E-03	5.00E-03	4.20E-01	0.00109
Tetrachloroethylene	4.00E-03	1.00E-02	4.00E-01	0.00104
2,4-Dimethylphenol	6.50E-03	2.00E-02	3.25E-01	0.00085
4,4-DDT	4.00E-05	5.00E-04	8.00E-02	0.00021
Carbon Disulfide	8.00E-03	1.00E-01	8.00E-02	0.00021
Chromium, total	3.80E-02	8.76E-01	4.34E-02	0.00011
4-Methyl-2-Pentanone	2.00E-03	5.00E-02	4.00E-02	0.00010
1,1,1-Trichloroethane	2.00E-03	9.00E-02	2.22E-02	0.00006
Phenol	1.10E-02	6.00E-01	1.83E-02	0.00005
Gamma-BHC	5.10E-06	3.00E-04	1.70E-02	0.00004
Toluene	2.00E-03	2.00E-01	1.00E-02	0.00003
Ethylbenzene	1.00E-03	1.00E-01	1.00E-02	0.00003
Di-n-butylphthalate	4.00E-04	1.00E-01	4.00E-03	0.00001
Total Xylenes	3.00E-03	2.00E+00	1.50E-03	0.00000
Benzoic Acid	5.00E-03	4.00E+00	1.25E-03	0.00000
Diethylphthalate	5.50E-04	8.00E-01	6.87E-04	0.00000
Lead	3.52E-01	.	.	.
Magnesium	2.11E+02	.	.	.
Potassium	2.11E+02	.	.	.
Sodium	3.94E+02	.	.	.
Calcium	2.86E+02	.	.	.
Delta-BHC	2.10E-05	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Noncarcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Sediments

Analyte Detected	Highest Detected Conc.	Oral RfD	Noncarc. Tox. Factor	Noncarcinogenic Ratio
Iron	6.90E+04	5.00E-01	1.38E+05	0.41882
Arsenic	2.76E+01	3.00E-04	9.20E+04	0.27921
Antimony	1.52E+01	4.00E-04	3.80E+04	0.11533
Aluminum	2.78E+04	1.00E+00	2.78E+04	0.08437
Vanadium	4.90E+01	7.00E-03	7.00E+03	0.02124
Manganese	6.95E+02	1.00E-01	6.95E+03	0.02109
Mercury	1.65E+00	3.00E-04	5.50E+03	0.01669
Barium	2.18E+02	5.00E-02	4.36E+03	0.01323
Cadmium	2.10E+00	5.00E-04	4.20E+03	0.01275
Nickel	3.54E+01	2.00E-02	1.77E+03	0.00537
Zinc	2.93E+02	2.00E-01	1.46E+03	0.00445
Copper	4.20E+01	4.00E-02	1.05E+03	0.00318
Beryllium	1.30E+00	5.00E-03	2.60E+02	0.00079
Heptachlor epoxide	3.10E-03	1.30E-05	2.38E+02	0.00072
bis(2-Ethylhexyl)phthalate	3.90E+00	2.00E-02	1.95E+02	0.00059
Endosulfan II	7.80E-03	5.00E-05	1.56E+02	0.00047
4,4-DDT	7.70E-02	5.00E-04	1.54E+02	0.00047
Selenium	7.10E-01	5.00E-03	1.42E+02	0.00043
Aldrin	2.00E-03	3.00E-05	6.67E+01	0.00020
Endrin	1.80E-02	3.00E-04	6.00E+01	0.00018
Dieldrin	2.25E-03	5.00E-05	4.50E+01	0.00014
Chromium, total	3.48E+01	8.76E-01	3.97E+01	0.00012
Di-n-octylphthalate	2.90E-01	2.00E-02	1.45E+01	0.00004
Pyrene	3.10E-01	3.00E-02	1.03E+01	0.00003
Fluoranthene	3.30E-01	4.00E-02	8.25E+00	0.00003
Gamma-BHC	1.50E-03	3.00E-04	5.00E+00	0.00002
Methoxyclor	1.60E-02	5.00E-03	3.20E+00	0.00001
Di-n-butylphthalate	1.40E-01	1.00E-01	1.40E+00	0.00000
Methylene Chloride	7.30E-02	6.00E-02	1.22E+00	0.00000
Acetone	8.90E-02	1.00E-01	8.90E-01	0.00000
1,1-Dichloroethane	1.90E-02	1.00E-01	1.90E-01	0.00000
1,1,1-Trichloroethane	3.00E-03	9.00E-02	3.33E-02	0.00000
Benzo(a)pyrene	2.50E-01	.	.	.
Benzo(b)fluoranthene	3.20E-01	.	.	.
Benzo(k)fluoranthene	2.50E-01	.	.	.
Benzo(a)anthracene	2.10E-01	.	.	.
Chrysene	2.70E-01	.	.	.
4,4-DDE	2.80E-02	.	.	.
4,4-DDD	4.70E-03	.	.	.
Benzene	3.00E-03	.	.	.
Phenanthrene	1.80E-01	.	.	.
Benzo(g,h,i)perylene	2.30E-01	.	.	.
Lead	1.00E+02	.	.	.
Magnesium	4.87E+04	.	.	.
Potassium	6.34E+03	.	.	.
Sodium	2.26E+03	.	.	.
Calcium	1.15E+05	.	.	.
Delta-BHC	5.40E-03	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Noncarcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Landfill Leachate - Soil

Analyte Detected	Highest Detected Conc.	Oral RfD	Noncarc. Tox. Factor	Noncarcinogenic Ratio
Iron	2.57E+04	5.00E-01	5.14E+04	0.42021
Arsenic	1.20E+01	3.00E-04	4.00E+04	0.32701
Aluminum	1.17E+04	1.00E+00	1.17E+04	0.09565
Manganese	5.11E+02	1.00E-01	5.11E+03	0.04178
Mercury	1.20E+00	3.00E-04	4.00E+03	0.03270
Vanadium	2.31E+01	7.00E-03	3.30E+03	0.02698
Barium	1.10E+02	5.00E-02	2.20E+03	0.01799
Cadmium	7.10E-01	5.00E-04	1.42E+03	0.01161
Copper	4.78E+01	4.00E-02	1.19E+03	0.00977
Nickel	1.87E+01	2.00E-02	9.35E+02	0.00764
Zinc	1.19E+02	2.00E-01	5.95E+02	0.00486
Beryllium	6.00E-01	5.00E-03	1.20E+02	0.00098
Aldrin	2.10E-03	3.00E-05	7.00E+01	0.00057
Dieldrin	3.20E-03	5.00E-05	6.40E+01	0.00052
Naphthalene	2.40E-01	4.00E-03	6.00E+01	0.00049
bis(2-Ethylhexyl)phthalate	7.50E-01	2.00E-02	3.75E+01	0.00031
Pyrene	8.40E-01	3.00E-02	2.80E+01	0.00023
Chromium, total	1.81E+01	8.76E-01	2.07E+01	0.00017
gamma-chlordane	1.20E-03	6.00E-05	2.00E+01	0.00016
Fluoranthene	4.80E-01	4.00E-02	1.20E+01	0.00010
1,4-Dichlorobenzene (para)	1.20E+00	1.00E-01	1.20E+01	0.00010
4-Methylphenol	4.40E-01	5.00E-02	8.80E+00	0.00007
Benzylbutylphthalate	1.20E+00	2.00E-01	6.00E+00	0.00005
Di-n-butylphthalate	3.50E-01	1.00E-01	3.50E+00	0.00003
Acenaphthene	1.00E-01	6.00E-02	1.67E+00	0.00001
Methylene Chloride	6.70E-02	6.00E-02	1.12E+00	0.00001
Anthracene	1.90E-01	3.00E-01	6.33E-01	0.00001
Benzo(a)anthracene	2.10E-01	.	.	.
Chrysene	2.00E-01	.	.	.
4,4-DDE	6.90E-04	.	.	.
2-Methylnaphthalene	1.20E-01	.	.	.
Phenanthrene	4.70E-01	.	.	.
Lead	1.10E+02	.	.	.
Magnesium	3.13E+04	.	.	.
Potassium	2.89E+03	.	.	.
Sodium	3.94E+02	.	.	.
Calcium	8.14E+04	.	.	.
Delta-BHC	2.10E-03	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Noncarcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Landfill Leachate - Water

Analyte Detected	Highest Detected Conc.	Oral RfD	Noncarc. Tox. Factor	Noncarcinogenic Ratio
Iron	3.90E+02	5.00E-01	7.80E+02	0.42222
Aluminum	3.25E+02	1.00E+00	3.25E+02	0.17592
Arsenic	5.86E-02	3.00E-04	1.95E+02	0.10573
Barium	7.61E+00	5.00E-02	1.52E+02	0.08239
4-Methylphenol	3.75E+00	5.00E-02	7.50E+01	0.04060
Naphthalene	2.00E-01	4.00E-03	5.00E+01	0.02707
2,4-Dimethylphenol	9.80E-01	2.00E-02	4.90E+01	0.02652
4-Chloroaniline	1.60E-01	4.00E-03	4.00E+01	0.02165
Manganese	2.96E+00	1.00E-01	2.96E+01	0.01602
2-Butanone (MEK)	1.40E+00	5.00E-02	2.80E+01	0.01516
Acetone	2.20E+00	1.00E-01	2.20E+01	0.01191
2-Methylphenol	9.60E-01	5.00E-02	1.92E+01	0.01039
Cadmium	9.00E-03	5.00E-04	1.80E+01	0.00974
Nickel	1.57E-01	2.00E-02	7.85E+00	0.00425
Methylene Chloride	4.70E-01	6.00E-02	7.83E+00	0.00424
Zinc	1.41E+00	2.00E-01	7.05E+00	0.00382
Ethylbenzene	6.80E-01	1.00E-01	6.80E+00	0.00368
2-Hexanone (MBK)	2.70E-01	4.00E-02	6.75E+00	0.00365
Vanadium	3.30E-02	7.00E-03	4.71E+00	0.00255
Phenol	1.80E+00	6.00E-01	3.00E+00	0.00162
Benzoic Acid	1.20E+01	4.00E+00	3.00E+00	0.00162
Chlorobenzene	5.60E-02	2.00E-02	2.80E+00	0.00152
Aldrin	8.20E-05	3.00E-05	2.73E+00	0.00148
Copper	9.90E-02	4.00E-02	2.47E+00	0.00134
Heptachlor	1.10E-03	5.00E-04	2.20E+00	0.00119
Toluene	4.10E-01	2.00E-01	2.05E+00	0.00111
Mercury	5.00E-04	3.00E-04	1.67E+00	0.00090
Total Xylenes	1.40E+00	2.00E+00	7.00E-01	0.00038
bis(2-Ethylhexyl)phthalate	1.00E-02	2.00E-02	5.00E-01	0.00027
Beryllium	2.20E-03	5.00E-03	4.40E-01	0.00024
4-Methyl-2-Pentanone	2.10E-02	5.00E-02	4.20E-01	0.00023
4,4-DDT	1.10E-04	5.00E-04	2.20E-01	0.00012
1,3-Dichlorobenzene	6.00E-03	3.00E-02	2.00E-01	0.00011
1,2-Dichlorobenzene	1.60E-02	9.00E-02	1.78E-01	0.00010
Gamma-BHC	5.20E-05	3.00E-04	1.73E-01	0.00009
Chromium, total	1.18E-01	8.76E-01	1.35E-01	0.00007
Diethylphthalate	5.50E-02	8.00E-01	6.87E-02	0.00004
Di-n-butylphthalate	4.00E-03	1.00E-01	4.00E-02	0.00002
Fluorene	1.00E-03	4.00E-02	2.50E-02	0.00001
Benzylbutylphthalate	4.00E-03	2.00E-01	2.00E-02	0.00001
Acenaphthene	1.00E-03	6.00E-02	1.67E-02	0.00001
2,6-Dinitrotoluene	5.10E-02	.	.	.
Benzene	5.00E-02	.	.	.
N-Nitrosodiphenylamine	7.00E-03	.	.	.
4,4-DDD	1.50E-05	.	.	.
2-Methylnaphthalene	5.00E-03	.	.	.
Phenanthrene	2.00E-03	.	.	.
Lead	1.01E+00	.	.	.
Magnesium	4.19E+02	.	.	.
Potassium	4.45E+02	.	.	.
Sodium	1.66E+03	.	.	.
Calcium	3.96E+02	.	.	.
Delta-BHC	1.80E-04	.	.	.
				1.00000

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RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Carcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Surface Soils

Analyte Detected	Highest Detected Conc.	Oral Slope Factor	Carc. Tox. Factor	Carcinogenic Ratio
Arsenic	2.80E+01	1.75E+00	4.90E+01	0.90404
Beryllium	1.10E+00	4.30E+00	4.73E+00	0.08727
Vinyl Chloride	2.40E-01	1.90E+00	4.56E-01	0.00841
bis(2-Ethylhexyl)phthalate	1.02E+00	1.40E-02	1.44E-02	0.00026
Trichloroethylene	3.20E-02	1.10E-02	3.52E-04	0.00001
Methylene Chloride	2.20E-02	7.50E-03	1.65E-04	0.00000
Styrene	1.40E-03	3.00E-02	4.20E-05	0.00000
Aluminum	2.60E+04	.	.	.
Copper	3.20E+01	.	.	.
Iron	3.10E+04	.	.	.
Lead	1.75E+02	.	.	.
Magnesium	2.55E+04	.	.	.
Manganese	5.75E+02	.	.	.
Nickel	2.60E+01	.	.	.
Potassium	6.30E+03	.	.	.
Sodium	6.90E+02	.	.	.
Vanadium	3.70E+01	.	.	.
Zinc	1.05E+02	.	.	.
Barium	1.35E+02	.	.	.
Calcium	6.89E+04	.	.	.
Chromium, total	3.10E+01	.	.	.
Delta-BHC	1.40E-03	.	.	.
1,1,1-Trichloroethane	6.10E-03	.	.	.
Acetone	1.70E-02	.	.	.
1,2-Dichloroethylene (total)	4.40E-01	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Carcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Subsurface Soils

Analyte Detected	Highest Detected Conc.	Oral Slope Factor	Carc. Tox. Factor	Carcinogenic Ratio
Arsenic	2.60E+01	1.75E+00	4.55E+01	0.90929
Beryllium	9.60E-01	4.30E+00	4.13E+00	0.08250
Vinyl Chloride	2.10E-01	1.90E+00	3.99E-01	0.00797
Heptachlor epoxide	5.80E-04	9.10E+00	5.28E-03	0.00011
Chrysene	4.60E-02	5.79E-02	2.66E-03	0.00005
bis(2-Ethylhexyl)phthalate	1.60E-01	1.40E-02	2.24E-03	0.00004
Alpha-BHC	2.60E-04	6.30E+00	1.64E-03	0.00003
Trichloroethylene	1.50E-02	1.10E-02	1.65E-04	0.00000
Methylene Chloride	1.70E-02	7.50E-03	1.28E-04	0.00000
4-Methylphenol	6.80E-02	.	.	.
Benzoic Acid	2.20E-01	.	.	.
Naphthalene	4.30E-02	.	.	.
2,4,5-Trichlorophenol	5.00E-02	.	.	.
Phenanthrene	6.00E-02	.	.	.
Fluoranthene	8.60E-02	.	.	.
Pyrene	6.70E-02	.	.	.
Di-n-octylphthalate	5.20E-02	.	.	.
Aluminum	2.50E+04	.	.	.
Copper	2.42E+02	.	.	.
Iron	3.10E+04	.	.	.
Lead	2.05E+01	.	.	.
Magnesium	3.90E+04	.	.	.
Manganese	1.30E+03	.	.	.
Mercury	2.30E-01	.	.	.
Nickel	3.00E+01	.	.	.
Potassium	6.00E+03	.	.	.
Selenium	8.20E-01	.	.	.
Antimony	1.30E+01	.	.	.
Sodium	9.20E+02	.	.	.
Thallium	6.10E-01	.	.	.
Vanadium	3.60E+01	.	.	.
Zinc	1.35E+02	.	.	.
Barium	1.60E+02	.	.	.
Calcium	9.00E+04	.	.	.
Chromium, total	3.00E+01	.	.	.
1,1,1-Trichloroethane	5.70E-03	.	.	.
Acetone	5.80E-02	.	.	.
1,2-Dichloroethylene (total)	3.20E-01	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Carcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Northern Landfill Cell Ground Water

Analyte Detected	Highest Detected Conc.	Oral Slope Factor	Carc. Tox. Factor	Carcinogenic Ratio
Arsenic	2.50E-03	1.75E+00	4.38E-03	0.38426
Beryllium	1.00E-03	4.30E+00	4.30E-03	0.37767
Styrene	6.70E-02	3.00E-02	2.01E-03	0.17654
1,4-Dichlorobenzene (para)	8.00E-03	2.40E-02	1.92E-04	0.01686
Benzene	5.00E-03	2.90E-02	1.45E-04	0.01274
Trichloroethylene	9.50E-03	1.10E-02	1.04E-04	0.00918
Methylene Chloride	1.25E-02	7.50E-03	9.37E-05	0.00823
Tetrachloroethylene	1.00E-03	5.20E-02	5.20E-05	0.00457
bis(2-Ethylhexyl)phthalate	3.50E-03	1.40E-02	4.90E-05	0.00430
Chloroform	5.50E-03	6.10E-03	3.36E-05	0.00295
Heptachlor	6.70E-06	4.50E+00	3.02E-05	0.00265
Gamma-BHC	4.10E-07	1.30E+00	5.33E-07	0.00005
Phenol	2.65E+00	.	.	.
4-Methylphenol	2.45E-01	.	.	.
2,4-Dimethylphenol	2.70E-02	.	.	.
Naphthalene	8.00E-04	.	.	.
Di-n-butylphthalate	2.00E-03	.	.	.
2-Methylphenol	1.75E-01	.	.	.
Aluminum	2.24E-01	.	.	.
Copper	3.10E-02	.	.	.
Iron	6.55E-01	.	.	.
Magnesium	9.71E+01	.	.	.
Manganese	2.43E-02	.	.	.
Nickel	2.28E-02	.	.	.
Potassium	1.17E+02	.	.	.
Selenium	1.00E-03	.	.	.
Silver	4.25E-03	.	.	.
Antimony	4.48E-02	.	.	.
Sodium	8.30E+01	.	.	.
Vanadium	1.52E-02	.	.	.
Zinc	1.57E-02	.	.	.
Barium	9.76E-02	.	.	.
Calcium	5.11E+02	.	.	.
Chromium, total	3.29E-02	.	.	.
Endosulfan sulfate	6.90E-07	.	.	.
cis-1,2-Dichloroethylene	3.50E-04	.	.	.
2-Butanone (MEK)	6.05E-02	.	.	.
4-Methyl-2-Pentanone	6.50E-03	.	.	.
2-Hexanone (MBK)	4.00E-03	.	.	.
Toluene	4.95E-02	.	.	.
Ethylbenzene	9.50E-03	.	.	.
Isopropylbenzene	3.00E-04	.	.	.
Acetone	3.20E-01	.	.	.
1,2,4-Trimethylbenzene	9.50E-04	.	.	.
1,3,5-Trimethylbenzene	3.00E-04	.	.	.
Total Xylenes	2.60E-02	.	.	.
Carbon Disulfide	1.00E-03	.	.	.
1,1-Dichloroethane	1.00E-03	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Carcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Landfill Perimeter Ground Water

Analyte Detected	Highest Detected Conc.	Oral Slope Factor	Carc. Tox. Factor	Carcinogenic Ratio
Arsenic	1.64E-02	1.75E+00	2.87E-02	0.65551
Beryllium	3.10E-03	4.30E+00	1.33E-02	0.30377
Heptachlor	9.00E-05	4.50E+00	4.05E-04	0.00925
Pentachlorophenol	3.00E-03	1.20E-01	3.60E-04	0.00822
bis(2-Ethylhexyl)phthalate	2.30E-02	1.40E-02	3.22E-04	0.00735
4,4'-DDT	6.70E-04	3.40E-01	2.28E-04	0.00521
4,4'-DDE	5.70E-04	3.40E-01	1.94E-04	0.00443
beta-BHC	4.90E-05	1.80E+00	8.82E-05	0.00201
4,4'-DDD	2.40E-04	2.40E-01	5.76E-05	0.00132
Methylene Chloride	4.00E-03	7.50E-03	3.00E-05	0.00069
Benzene	1.00E-03	2.90E-02	2.90E-05	0.00066
gamma-chlordane	1.50E-05	1.30E+00	1.95E-05	0.00045
Aldrin	8.90E-07	1.70E+01	1.51E-05	0.00034
Dieldrin	5.80E-07	1.60E+01	9.28E-06	0.00021
Heptachlor epoxide	8.60E-07	9.10E+00	7.83E-06	0.00018
Chloroform	1.00E-03	6.10E-03	6.10E-06	0.00014
alpha-BHC	6.50E-07	6.30E+00	4.09E-06	0.00009
gamma-BHC	3.00E-06	1.30E+00	3.90E-06	0.00009
alpha-chlordane	2.20E-06	1.30E+00	2.86E-06	0.00007
Phenol	4.00E-03	.	.	.
Diethylphthalate	2.00E-03	.	.	.
Di-n-butylphthalate	1.00E-03	.	.	.
Butylbenzylphthalate	4.00E-03	.	.	.
Aluminum	8.08E+01	.	.	.
Copper	1.27E-01	.	.	.
Iron	1.08E+02	.	.	.
Lead	7.79E-02	.	.	.
Magnesium	3.40E+02	.	.	.
Manganese	3.93E+00	.	.	.
Mercury	1.80E-03	.	.	.
Nickel	1.55E-01	.	.	.
Potassium	2.43E+01	.	.	.
Silver	6.50E-03	.	.	.
Antimony	6.98E-02	.	.	.
Sodium	3.61E+03	.	.	.
Vanadium	1.50E-01	.	.	.
Zinc	5.08E-01	.	.	.
Barium	4.31E-01	.	.	.
Cadmium	5.00E-03	.	.	.
Calcium	5.77E+02	.	.	.
Chromium	1.34E-01	.	.	.
Endosulfan sulfate	5.60E-05	.	.	.
Methoxychlor	7.80E-06	.	.	.
Endrin ketone	2.20E-05	.	.	.
Endrin aldehyde	7.60E-06	.	.	.
delta-BHC	1.20E-06	.	.	.
Toluene	5.00E-03	.	.	.
Ethylbenzene	1.00E-03	.	.	.
Acetone	2.70E-02	.	.	.
Total Xylenes	8.00E-03	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Carcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Surface Water

Analyte Detected	Highest Detected Conc.	Oral Slope Factor	Carc. Tox. Factor	Carcinogenic Ratio
Arsenic	3.06E-02	1.75E+00	5.36E-02	0.69604
bis(2-Ethylhexyl)phthalate	1.00E+00	1.40E-02	1.40E-02	0.18197
Beryllium	2.10E-03	4.30E+00	9.03E-03	0.11737
Tetrachloroethylene	4.00E-03	5.20E-02	2.08E-04	0.00270
Heptachlor epoxide	1.40E-05	9.10E+00	1.27E-04	0.00166
4,4-DDT	4.00E-05	3.40E-01	1.36E-05	0.00018
Gamma-BHC	5.10E-06	1.30E+00	6.63E-06	0.00009
Phenol	1.10E-02	.	.	.
2,4-Dimethylphenol	6.50E-03	.	.	.
Benzoic Acid	5.00E-03	.	.	.
Diethylphthalate	5.50E-04	.	.	.
Di-n-butylphthalate	4.00E-04	.	.	.
Aluminum	2.52E+01	.	.	.
Copper	9.40E-02	.	.	.
Iron	3.80E+01	.	.	.
Lead	3.52E-01	.	.	.
Magnesium	2.11E+02	.	.	.
Manganese	1.69E+00	.	.	.
Nickel	6.30E-02	.	.	.
Potassium	2.11E+02	.	.	.
Sodium	3.94E+02	.	.	.
Thallium	4.40E-03	.	.	.
Vanadium	6.10E-02	.	.	.
Zinc	2.36E+00	.	.	.
Cyanide	4.06E-02	.	.	.
Barium	4.56E-01	.	.	.
Cadmium	5.70E-03	.	.	.
Calcium	2.86E+02	.	.	.
Chromium, total	3.80E-02	.	.	.
Delta-BHC	2.10E-05	.	.	.
1,1,1-Trichloroethane	2.00E-03	.	.	.
4-Methyl-2-Pentanone	2.00E-03	.	.	.
Toluene	2.00E-03	.	.	.
Ethylbenzene	1.00E-03	.	.	.
Total Xylenes	3.00E-03	.	.	.
Carbon Disulfide	8.00E-03	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Carcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Sediments

Analyte Detected	Highest Detected Conc.	Oral Slope Factor	Carc. Tox. Factor	Carcinogenic Ratio
Arsenic	2.76E+01	1.75E+00	4.83E+01	0.86255
Beryllium	1.30E+00	4.30E+00	5.59E+00	0.09983
Benzo(a)pyrene	2.50E-01	5.79E+00	1.45E+00	0.02585
Benzo(b)fluoranthene	3.20E-01	5.79E-01	1.85E-01	0.00331
Benzo(k)fluoranthene	2.50E-01	5.79E-01	1.45E-01	0.00258
Benzo(a)anthracene	2.10E-01	5.79E-01	1.22E-01	0.00217
bis(2-Ethylhexyl)phthalate	3.90E+00	1.40E-02	5.46E-02	0.00098
Dieldrin	2.25E-03	1.60E+01	3.60E-02	0.00064
Aldrin	2.00E-03	1.70E+01	3.40E-02	0.00061
Heptachlor epoxide	3.10E-03	9.10E+00	2.82E-02	0.00050
4,4-DDT	7.70E-02	3.40E-01	2.62E-02	0.00047
Chrysene	2.70E-01	5.79E-02	1.56E-02	0.00028
4,4-DDE	2.80E-02	3.40E-01	9.52E-03	0.00017
Gamma-BHC	1.50E-03	1.30E+00	1.95E-03	0.00003
4,4-DDD	4.70E-03	2.40E-01	1.13E-03	0.00002
Methylene Chloride	7.30E-02	7.50E-03	5.47E-04	0.00001
Benzene	3.00E-03	2.90E-02	8.70E-05	0.00000
Phenanthrene	1.80E-01	.	.	.
Di-n-butylphthalate	1.40E-01	.	.	.
Fluoranthene	3.30E-01	.	.	.
Pyrene	3.10E-01	.	.	.
Di-n-octylphthalate	2.90E-01	.	.	.
Benzo(g,h,i)perylene	2.30E-01	.	.	.
Aluminum	2.78E+04	.	.	.
Copper	4.20E+01	.	.	.
Iron	6.90E+04	.	.	.
Lead	1.00E+02	.	.	.
Magnesium	4.87E+04	.	.	.
Manganese	6.95E+02	.	.	.
Mercury	1.65E+00	.	.	.
Nickel	3.54E+01	.	.	.
Potassium	6.34E+03	.	.	.
Selenium	7.10E-01	.	.	.
Antimony	1.52E+01	.	.	.
Sodium	2.26E+03	.	.	.
Vanadium	4.90E+01	.	.	.
Zinc	2.93E+02	.	.	.
Barium	2.18E+02	.	.	.
Cadmium	2.10E+00	.	.	.
Calcium	1.15E+05	.	.	.
Chromium, total	3.48E+01	.	.	.
Endrin	1.80E-02	.	.	.
Endosulfan II	7.80E-03	.	.	.
Methoxyclor	1.60E-02	.	.	.
Delta-BHC	5.40E-03	.	.	.
1,1,1-Trichloroethane	3.00E-03	.	.	.
Acetone	8.90E-02	.	.	.
1,1-Dichloroethane	1.90E-02	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Carcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Landfill Leachate - Soil

Analyte Detected	Highest Detected Conc.	Oral Slope Factor	Carc. Tox. Factor	Carcinogenic Ratio
Arsenic	1.20E+01	1.75E+00	2.10E+01	0.88081
Beryllium	6.00E-01	4.30E+00	2.58E+00	0.10821
Benzo(a)anthracene	2.10E-01	5.79E-01	1.22E-01	0.00510
Dieldrin	3.20E-03	1.60E+01	5.12E-02	0.00215
Aldrin	2.10E-03	1.70E+01	3.57E-02	0.00150
1,4-Dichlorobenzene (para)	1.20E+00	2.40E-02	2.88E-02	0.00121
Chrysene	2.00E-01	5.79E-02	1.16E-02	0.00049
bis(2-Ethylhexyl)phthalate	7.50E-01	1.40E-02	1.05E-02	0.00044
gamma-chlordane	1.20E-03	1.30E+00	1.56E-03	0.00007
Methylene Chloride	6.70E-02	7.50E-03	5.03E-04	0.00002
4,4-DDE	6.90E-04	3.40E-01	2.35E-04	0.00001
4-Methylphenol	4.40E-01	.	.	.
Naphthalene	2.40E-01	.	.	.
2-Methylnaphthalene	1.20E-01	.	.	.
Acenaphthene	1.00E-01	.	.	.
Phenanthrene	4.70E-01	.	.	.
Anthracene	1.90E-01	.	.	.
Di-n-butylphthalate	3.50E-01	.	.	.
Fluoranthene	4.80E-01	.	.	.
Pyrene	8.40E-01	.	.	.
Benzylbutylphthalate	1.20E+00	.	.	.
Aluminum	1.17E+04	.	.	.
Copper	4.78E+01	.	.	.
Iron	2.57E+04	.	.	.
Lead	1.10E+02	.	.	.
Magnesium	3.13E+04	.	.	.
Manganese	5.11E+02	.	.	.
Mercury	1.20E+00	.	.	.
Nickel	1.87E+01	.	.	.
Potassium	2.89E+03	.	.	.
Sodium	3.94E+02	.	.	.
Vanadium	2.31E+01	.	.	.
Zinc	1.19E+02	.	.	.
Barium	1.10E+02	.	.	.
Cadmium	7.10E-01	.	.	.
Calcium	8.14E+04	.	.	.
Chromium, total	1.81E+01	.	.	.
Delta-BHC	2.10E-03	.	.	.
				1.00000

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Carcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Landfill Leachate - Water

Analyte Detected	Highest Detected Conc.	Oral Slope Factor	Carc. Tox. Factor	Carcinogenic Ratio
Arsenic	5.86E-02	1.75E+00	1.03E-01	0.64785
2,6-Dinitrotoluene	5.10E-02	6.80E-01	3.47E-02	0.21909
Beryllium	2.20E-03	4.30E+00	9.46E-03	0.05976
Heptachlor	1.10E-03	4.50E+00	4.95E-03	0.03127
Methylene Chloride	4.70E-01	7.50E-03	3.52E-03	0.02227
Benzene	5.00E-02	2.90E-02	1.45E-03	0.00916
Aldrin	8.20E-05	1.70E+01	1.39E-03	0.00881
bis(2-Ethylhexyl)phthalate	1.00E-02	1.40E-02	1.40E-04	0.00088
Gamma-BHC	5.20E-05	1.30E+00	6.76E-05	0.00043
4,4-DDT	1.10E-04	3.40E-01	3.74E-05	0.00024
N-Nitrosodiphenylamine	7.00E-03	4.90E-03	3.43E-05	0.00022
4,4-DDD	1.50E-05	2.40E-01	3.60E-06	0.00002
Phenol	1.80E+00	.	.	.
4-Methylphenol	3.75E+00	.	.	.
2,4-Dimethylphenol	9.80E-01	.	.	.
Benzoic Acid	1.20E+01	.	.	.
Naphthalene	2.00E-01	.	.	.
4-Chloroaniline	1.60E-01	.	.	.
2-Methylnaphthalene	5.00E-03	.	.	.
Acenaphthene	1.00E-03	.	.	.
Diethylphthalate	5.50E-02	.	.	.
Fluorene	1.00E-03	.	.	.
Phenanthrene	2.00E-03	.	.	.
Di-n-butylphthalate	4.00E-03	.	.	.
Benzylbutylphthalate	4.00E-03	.	.	.
2-Methylphenol	9.60E-01	.	.	.
Aluminum	3.25E+02	.	.	.
Copper	9.90E-02	.	.	.
Iron	3.90E+02	.	.	.
Lead	1.01E+00	.	.	.
Magnesium	4.19E+02	.	.	.
Manganese	2.96E+00	.	.	.
Mercury	5.00E-04	.	.	.
Nickel	1.57E-01	.	.	.
Potassium	4.45E+02	.	.	.
Sodium	1.66E+03	.	.	.
Vanadium	3.30E-02	.	.	.
Zinc	1.41E+00	.	.	.
Barium	7.61E+00	.	.	.
Cadmium	9.00E-03	.	.	.
Calcium	3.96E+02	.	.	.
Chromium, total	1.18E-01	.	.	.
Delta-BHC	1.80E-04	.	.	.
2-Butanone (MEK)	1.40E+00	.	.	.
4-Methyl-2-Pentanone	2.10E-02	.	.	.
2-Hexanone (MBK)	2.70E-01	.	.	.
Toluene	4.10E-01	.	.	.
Chlorobenzene	5.60E-02	.	.	.
Ethylbenzene	6.80E-01	.	.	.
1,2-Dichlorobenzene	1.60E-02	.	.	.
1,3-Dichlorobenzene	6.00E-03	.	.	.

RESULTS OF TOXICITY SCREEN FOR THE NCR SITE. (continued).

Ranked Carcinogenic Toxicity Scores - By Medium
(Concentrations in ppm)

TYPE=Landfill Leachate - Water
(continued)

Analyte Detected	Highest Detected Conc.	Oral Slope Factor	Carc. Tox. Factor	Carcinogenic Ratio
Acetone	2.20E+00	.	.	.
Total Xylenes	1.40E+00	.	.	.
				1.00000

APPENDIX E

RISK SPREADSHEETS

TABLE : 1
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : PERIMETER GROUND WATER
EXPOSURE TYPE : INGESTION/FUTURE/RESIDENT
RISK TYPE : CARCINOGENIC

Contaminant of Concern	*Concentration (mg/L)*	*Ingestion Rate (L/day)*	*Exposure Frequency (days/year)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Intake (mg/kg/day)*	*Oral Slope Factor (mg/kg/day)-1*	*Increased CA Risk*
Acetone	3.90E-03	2	350	30	70	25550	4.58E-05		
Benzene	5.24E-04	2	350	30	70	25550	6.15E-06	2.90E-02	1.78E-07
Methylene Chloride	1.97E-03	2	350	30	70	25550	2.31E-05	7.50E-03	1.73E-07
Bis(2-ethylhexyl)phthalate	3.46E-03	2	350	30	70	25550	4.06E-05	1.40E-02	5.68E-07
Phenol	2.67E-03	2	350	30	70	25550	3.13E-05		
4,4-DDE	2.32E-05	2	350	30	70	25550	2.72E-07	3.40E-01	9.26E-08
4,4-DDT	2.15E-05	2	350	30	70	25550	2.53E-07	3.40E-01	8.59E-08
Aldrin	8.90E-07	2	350	30	70	25550	1.05E-08	1.70E+01	1.78E-07
Dieldrin	5.80E-07	2	350	30	70	25550	6.81E-09	1.60E+01	1.09E-07
Heptachlor	8.00E-06	2	350	30	70	25550	9.39E-08	4.50E+00	4.23E-07
Heptachlor epoxide	8.60E-07	2	350	30	70	25550	1.01E-08	9.10E+00	9.19E-08
Delta-BHC	1.20E-06	2	350	30	70	25550	1.41E-08		
Aluminum	1.91E+01	2	350	30	70	25550	2.25E-01		
Antimony	1.63E-02	2	350	30	70	25550	1.91E-04		
Arsenic	5.73E-03	2	350	30	70	25550	6.72E-05	1.75E+00	1.18E-04
Barium	1.40E-01	2	350	30	70	25550	1.64E-03		
Beryllium	7.70E-04	2	350	30	70	25550	9.04E-06	4.30E+00	3.89E-05
Cadmium	2.30E-03	2	350	30	70	25550	2.70E-05		
Cobalt	6.80E-03	2	350	30	70	25550	7.99E-05		
Copper	2.60E-02	2	350	30	70	25550	3.05E-04		
Iron	3.04E+01	2	350	30	70	25550	3.57E-01		
Lead	1.30E-02	2	350	30	70	25550	1.53E-04		
Manganese	1.87E+00	2	350	30	70	25550	2.20E-02		
Mercury	1.57E-04	2	350	30	70	25550	1.84E-06		
Nickel	5.36E-02	2	350	30	70	25550	6.30E-04		
Silver	2.63E-03	2	350	30	70	25550	3.08E-05		
Vanadium	1.48E-02	2	350	30	70	25550	1.73E-04		
Zinc	1.01E-01	2	350	30	70	25550	1.19E-03		
							TOTAL RISK		1.58E-04

TABLE : 2
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : PERIMETER GROUND WATER
EXPOSURE TYPE : INGESTION/FUTURE/RESIDENT
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/L)*	*Ingestion Rate (L/day)*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Chronic Body Dose (mg/kg/day)*	*Chronic Protective Body Dose (mg/kg/day)*	*Chronic Hazard Quotient*
Acetone	3.90E-03	2	350	30	70	10950	1.07E-04	1.00E-01	1.07E-03
Benzene	5.24E-04	2	350	30	70	10950	1.44E-05		
Methylene Chloride	1.97E-03	2	350	30	70	10950	5.39E-05	6.00E-02	8.98E-04
bis(2-Ethylhexyl)phthalate	3.46E-03	2	350	30	70	10950	9.47E-05	2.00E-02	4.74E-03
Phenol	2.67E-03	2	350	30	70	10950	7.30E-05	6.00E-01	1.22E-04
4,4-DDE	2.32E-05	2	350	30	70	10950	6.36E-07		
4,4-DDT	2.15E-05	2	350	30	70	10950	5.90E-07	5.00E-04	1.18E-03
Aldrin	8.90E-07	2	350	30	70	10950	2.44E-08	3.00E-05	8.13E-04
Dieldrin	5.80E-07	2	350	30	70	10950	1.59E-08	5.00E-05	3.18E-04
Heptachlor	8.00E-06	2	350	30	70	10950	2.19E-07	5.00E-04	4.38E-04
Heptachlor epoxide	8.60E-07	2	350	30	70	10950	2.36E-08	1.30E-05	1.81E-03
Delta-BHC	1.20E-06	2	350	30	70	10950	3.29E-08		
Aluminum	1.91E+01	2	350	30	70	10950	5.24E-01	1.00E+00	5.24E-01
Antimony	1.63E-02	2	350	30	70	10950	4.47E-04	4.00E-04	1.12E+00
Arsenic	5.73E-03	2	350	30	70	10950	1.57E-04	3.00E-04	5.23E-01
Barium	1.40E-01	2	350	30	70	10950	3.83E-03	5.00E-02	7.66E-02
Beryllium	7.70E-04	2	350	30	70	10950	2.11E-05	5.00E-03	4.22E-03
Cadmium	2.30E-03	2	350	30	70	10950	6.31E-05	5.00E-04	1.26E-01
Cobalt	6.80E-03	2	350	30	70	10950	1.86E-04		
Copper	2.60E-02	2	350	30	70	10950	7.12E-04	4.00E-02	1.78E-02
Iron	3.04E+01	2	350	30	70	10950	8.34E-01	5.00E-01	1.67E+00
Lead	1.30E-02	2	350	30	70	10950	3.56E-04		
Manganese	1.87E+00	2	350	30	70	10950	5.13E-02	1.00E-01	5.13E-01
Mercury	1.57E-04	2	350	30	70	10950	4.30E-06	3.00E-04	1.43E-02
Nickel	5.36E-02	2	350	30	70	10950	1.47E-03	2.00E-02	7.35E-02
Silver	2.63E-03	2	350	30	70	10950	7.20E-05	5.00E-03	1.44E-02
Vanadium	1.48E-02	2	350	30	70	10950	4.04E-04	7.00E-03	5.78E-02
Zinc	1.01E-01	2	350	30	70	10950	2.77E-03	2.00E-01	1.38E-02
								HAZARD INDEX	4.75E+00

TABLE : 3
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : NORTHERN LANDFILL CELL GROUND WATER
EXPOSURE TYPE : INGESTION/FUTURE/RESIDENT
RISK TYPE : CARCINOGENIC

Contaminant of Concern	*Concentration (mg/L)*	*Ingestion Rate (L/day)*	*Exposure Frequency (days/year)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Intake (mg/kg/day)*	*Oral Slope Factor (mg/kg/day)-1*	*Increased CA Risk*
Acetone	3.20E-01	2	350	30	70	25550	3.76E-03		
Benzene	5.00E-03	2	350	30	70	25550	5.87E-05	2.90E-02	1.70E-06
2-Butanone (MEK)	6.05E-02	2	350	30	70	25550	7.10E-04		
1,4-Dichlorobenzene (para)	8.00E-03	2	350	30	70	25550	9.39E-05	2.40E-02	2.25E-06
Methylene Chloride	1.25E-02	2	350	30	70	25550	1.47E-04	7.50E-03	1.10E-06
Styrene	6.70E-02	2	350	30	70	25550	7.87E-04	3.00E-02	2.36E-05
Trichloroethylene	9.50E-03	2	350	30	70	25550	1.12E-04	1.10E-02	1.23E-06
1,2,4-Trimethylbenzene	9.50E-04	2	350	30	70	25550	1.12E-05		
bis(2-Ethylhexyl)phthalate	3.50E-03	2	350	30	70	25550	4.11E-05	1.40E-02	5.75E-07
2,4-Dimethylphenol	2.70E-02	2	350	30	70	25550	3.17E-04		
2-Methylphenol	1.75E-01	2	350	30	70	25550	2.05E-03		
4-Methylphenol	2.45E-01	2	350	30	70	25550	2.88E-03		
Naphthalene	8.00E-04	2	350	30	70	25550	9.39E-06		
Phenol	2.65E+00	2	350	30	70	25550	3.11E-02		
Heptachlor	6.70E-06	2	350	30	70	25550	7.87E-08	4.50E+00	3.54E-07
Aluminum	2.23E-01	2	350	30	70	25550	2.62E-03		
Antimony	4.47E-02	2	350	30	70	25550	5.25E-04		
Arsenic	2.50E-03	2	350	30	70	25550	2.94E-05	1.75E+00	5.14E-05
Barium	9.76E-02	2	350	30	70	25550	1.15E-03		
Beryllium	1.00E-03	2	350	30	70	25550	1.17E-05	4.30E+00	5.05E-05
Cobalt	8.40E-03	2	350	30	70	25550	9.86E-05		
Copper	3.10E-02	2	350	30	70	25550	3.64E-04		
Iron	6.55E-01	2	350	30	70	25550	7.70E-03		
Manganese	2.42E-02	2	350	30	70	25550	2.85E-04		
Nickel	2.28E-02	2	350	30	70	25550	2.68E-04		
Silver	4.25E-03	2	350	30	70	25550	4.99E-05		
Vanadium	1.52E-02	2	350	30	70	25550	1.79E-04		
Zinc	1.57E-02	2	350	30	70	25550	1.84E-04		
							TOTAL RISK		1.33E-04

TABLE : 4
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : NORTHERN LANDFILL CELL GROUND WATER
EXPOSURE TYPE : INGESTION/FUTURE/RESIDENT
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/L)*	*Ingestion Rate (L/day)*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Chronic Body Dose (mg/kg/day)*	*Chronic Protective Body Dose (mg/kg/day)*	*Chronic Hazard Quotient*
Acetone	3.20E-01	2	350	30	70	10950	8.77E-03	1.00E-01	8.77E-02
Benzene	5.00E-03	2	350	30	70	10950	1.37E-04		
2-Butanone (MEK)	6.05E-02	2	350	30	70	10950	1.66E-03	5.00E-02	3.32E-02
1,4-Dichlorobenzene (para)	8.00E-03	2	350	30	70	10950	2.19E-04	1.00E-01	2.19E-03
Methylene Chloride	1.25E-02	2	350	30	70	10950	3.42E-04	6.00E-02	5.71E-03
Styrene	6.70E-02	2	350	30	70	10950	1.84E-03	2.00E-01	9.18E-03
Trichloroethylene	9.50E-03	2	350	30	70	10950	2.60E-04	6.00E-03	4.34E-02
1,2,4-Trimethylbenzene	9.50E-04	2	350	30	70	10950	2.60E-05	6.00E-04	4.34E-02
bis(2-Ethylhexyl)phthalate	3.50E-03	2	350	30	70	10950	9.59E-05	2.00E-02	4.79E-03
2,4-Dimethylphenol	2.70E-02	2	350	30	70	10950	7.40E-04	2.00E-02	3.70E-02
2-Methylphenol	1.75E-01	2	350	30	70	10950	4.79E-03	5.00E-02	9.59E-02
4-Methylphenol	2.45E-01	2	350	30	70	10950	6.71E-03	5.00E-02	1.34E-01
Naphthalene	8.00E-04	2	350	30	70	10950	2.19E-05	4.00E-03	5.48E-03
Phenol	2.65E+00	2	350	30	70	10950	7.26E-02	6.00E-01	1.21E-01
Heptachlor	6.70E-06	2	350	30	70	10950	1.84E-07	5.00E-04	3.67E-04
Aluminum	2.23E-01	2	350	30	70	10950	6.12E-03	1.00E+00	6.12E-03
Antimony	4.47E-02	2	350	30	70	10950	1.23E-03	4.00E-04	3.07E+00
Arsenic	2.50E-03	2	350	30	70	10950	6.85E-05	3.00E-04	2.28E-01
Barium	9.76E-02	2	350	30	70	10950	2.67E-03	5.00E-02	5.35E-02
Beryllium	1.00E-03	2	350	30	70	10950	2.74E-05	5.00E-03	5.48E-03
Cobalt	8.40E-03	2	350	30	70	10950	2.30E-04		
Copper	3.10E-02	2	350	30	70	10950	8.49E-04	4.00E-02	2.12E-02
Iron	6.55E-01	2	350	30	70	10950	1.80E-02	5.00E-01	3.59E-02
Manganese	2.42E-02	2	350	30	70	10950	6.64E-04	1.00E-01	6.64E-03
Nickel	2.28E-02	2	350	30	70	10950	6.25E-04	2.00E-02	3.12E-02
Silver	4.25E-03	2	350	30	70	10950	1.16E-04	5.00E-03	2.33E-02
Vanadium	1.52E-02	2	350	30	70	10950	4.18E-04	7.00E-03	5.97E-02
Zinc	1.57E-02	2	350	30	70	10950	4.30E-04	2.00E-01	2.15E-03
							HAZARD INDEX		4.16E+00

TABLE : 5
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : SURFACE SOIL
EXPOSURE TYPE : INGESTION/PRESENT AND FUTURE/YOUTH TRESPASSER
RISK TYPE : CARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Ingestion Rate (mg/day)*	*kg/mg*	*Fraction Ingested*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Intake (mg/kg/day)*	*Oral Slope Factor (mg/kg/day)-1*	*Increased CA Risk*
Acetone	1.05E-02	100	1.0E-06	1	117	10	50	25550	9.60E-10		
Methylene Chloride	1.98E-02	100	1.0E-06	1	117	10	50	25550	1.81E-09	7.50E-03	1.36E-11
Styrene	1.40E-03	100	1.0E-06	1	117	10	50	25550	1.28E-10	3.00E-02	3.85E-12
Trichloroethylene	1.18E-02	100	1.0E-06	1	117	10	50	25550	1.08E-09	1.10E-02	1.19E-11
Vinyl Chloride	3.82E-02	100	1.0E-06	1	117	10	50	25550	3.50E-09	1.90E+00	6.65E-09
bis(2-Ethylhexyl)phthalate	6.52E-01	100	1.0E-06	1	117	10	50	25550	5.97E-08	1.40E-02	8.36E-10
Delta-BHC	1.40E-03	100	1.0E-06	1	117	10	50	25550	1.28E-10		
Aluminum	1.63E+04	100	1.0E-06	1	117	10	50	25550	1.50E-03		
Arsenic	2.21E+01	100	1.0E-06	1	117	10	50	25550	2.02E-06	1.75E+00	3.54E-06
Barium	1.03E+02	100	1.0E-06	1	117	10	50	25550	9.46E-06		
Beryllium	8.09E-01	100	1.0E-06	1	117	10	50	25550	7.41E-08	4.30E+00	3.18E-07
Copper	1.99E+01	100	1.0E-06	1	117	10	50	25550	1.82E-06		
Cobalt	8.04E+00	100	1.0E-06	1	117	10	50	25550	7.37E-07		
Iron	1.94E+04	100	1.0E-06	1	117	10	50	25550	1.77E-03		
Lead	1.43E+02	100	1.0E-06	1	117	10	50	25550	1.31E-05		
Manganese	4.82E+02	100	1.0E-06	1	117	10	50	25550	4.42E-05		
Nickel	1.98E+01	100	1.0E-06	1	117	10	50	25550	1.81E-06		
Vanadium	2.55E+01	100	1.0E-06	1	117	10	50	25550	2.34E-06		
Zinc	6.87E+01	100	1.0E-06	1	117	10	50	25550	6.29E-06		

TOTAL RISK 3.87E-06

TABLE : 6
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : SURFACE SOIL
EXPOSURE TYPE : INGESTION/PRESENT AND FUTURE/YOUTH TRESPASSER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Ingestion Rate (mg/day)*	*kg/mg*	*Fraction Ingested*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Chronic Body Dose (mg/kg/day)*	*Chronic Protective Body Dose (mg/kg/day)*	*Chronic Hazard Quotient*
Acetone	1.05E-02	100	1.0E-06	1	117	10	50	3650	6.72E-09	1.00E-01	6.72E-08
Methylene Chloride	1.98E-02	100	1.0E-06	1	117	10	50	3650	1.27E-08	6.00E-02	2.12E-07
Styrene	1.40E-03	100	1.0E-06	1	117	10	50	3650	8.98E-10	2.00E-01	4.49E-09
Trichloroethylene	1.18E-02	100	1.0E-06	1	117	10	50	3650	7.58E-09	6.00E-03	1.26E-06
Vinyl Chloride	3.82E-02	100	1.0E-06	1	117	10	50	3650	2.45E-08		
bis(2-Ethylhexyl)phthalate	6.52E-01	100	1.0E-06	1	117	10	50	3650	4.18E-07	2.00E-02	2.09E-05
Delta-BHC	1.40E-03	100	1.0E-06	1	117	10	50	3650	8.98E-10		
Aluminum	1.63E+04	100	1.0E-06	1	117	10	50	3650	1.05E-02	1.00E+00	1.05E-02
Arsenic	2.21E+01	100	1.0E-06	1	117	10	50	3650	1.42E-05	3.00E-04	4.72E-02
Barium	1.03E+02	100	1.0E-06	1	117	10	50	3650	6.62E-05	5.00E-02	1.32E-03
Beryllium	8.09E-01	100	1.0E-06	1	117	10	50	3650	5.18E-07	5.00E-03	1.04E-04
Copper	1.99E+01	100	1.0E-06	1	117	10	50	3650	1.28E-05	4.00E-02	3.19E-04
Cobalt	8.04E+00	100	1.0E-06	1	117	10	50	3650	5.16E-06		
Iron	1.94E+04	100	1.0E-06	1	117	10	50	3650	1.24E-02	5.00E-01	2.48E-02
Lead	1.43E+02	100	1.0E-06	1	117	10	50	3650	9.19E-05		
Manganese	4.82E+02	100	1.0E-06	1	117	10	50	3650	3.09E-04	1.00E-01	3.09E-03
Nickel	1.98E+01	100	1.0E-06	1	117	10	50	3650	1.27E-05	2.00E-02	6.33E-04
Vanadium	2.55E+01	100	1.0E-06	1	117	10	50	3650	1.64E-05	7.00E-03	2.34E-03
Zinc	6.87E+01	100	1.0E-06	1	117	10	50	3650	4.40E-05	2.00E-01	2.20E-04

HAZARD INDEX 9.06E-02

TABLE : 7
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : SUBSURFACE SOIL
EXPOSURE TYPE : INGESTION/FUTURE/EXCAVATION WORKER
RISK TYPE : CARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Ingestion Rate (mg/day)*	*kg/mg*	*Fraction Ingested*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Intake (mg/kg/day)*	*Oral Slope Factor (mg/kg/day)-1*	*Increased CA Risk*
Acetone	2.84E-02	480	1.0E-06	1	65	1	70	25550	4.96E-10		
Methylene Chloride	1.70E-02	480	1.0E-06	1	65	1	70	25550	2.97E-10	7.50E-03	2.22E-12
Trichloroethylene	1.36E-02	480	1.0E-06	1	65	1	70	25550	2.37E-10	1.10E-02	2.60E-12
Vinyl Chloride	5.75E-02	480	1.0E-06	1	65	1	70	25550	1.00E-09	1.90E+00	1.91E-09
4-Methylphenol	6.80E-02	480	1.0E-06	1	65	1	70	25550	1.19E-09		
bis(2-Ethylhexyl)phthalate	1.60E-01	480	1.0E-06	1	65	1	70	25550	2.79E-09	1.40E-02	3.91E-11
Naphthalene	4.30E-02	480	1.0E-06	1	65	1	70	25550	7.50E-10		
Phenanthrene	6.00E-02	480	1.0E-06	1	65	1	70	25550	1.05E-09		
Heptachlor epoxide	5.80E-04	480	1.0E-06	1	65	1	70	25550	1.01E-11	9.10E+00	9.21E-11
Aluminum	1.94E+04	480	1.0E-06	1	65	1	70	25550	3.38E-04		
Antimony	7.82E+00	480	1.0E-06	1	65	1	70	25550	1.36E-07		
Arsenic	2.02E+01	480	1.0E-06	1	65	1	70	25550	3.53E-07	1.75E+00	6.17E-07
Barium	1.60E+02	480	1.0E-06	1	65	1	70	25550	2.79E-06		
Beryllium	9.60E-01	480	1.0E-06	1	65	1	70	25550	1.67E-08	4.30E+00	7.20E-08
Cobalt	1.21E+01	480	1.0E-06	1	65	1	70	25550	2.11E-07		
Copper	1.08E+02	480	1.0E-06	1	65	1	70	25550	1.88E-06		
Iron	2.62E+04	480	1.0E-06	1	65	1	70	25550	4.56E-04		
Lead	1.64E+01	480	1.0E-06	1	65	1	70	25550	2.85E-07		
Manganese	1.00E+03	480	1.0E-06	1	65	1	70	25550	1.75E-05		
Mercury	8.16E-02	480	1.0E-06	1	65	1	70	25550	1.42E-09		
Nickel	3.00E+01	480	1.0E-06	1	65	1	70	25550	5.23E-07		
Thallium	3.19E-01	480	1.0E-06	1	65	1	70	25550	5.57E-09		
Vanadium	3.34E+01	480	1.0E-06	1	65	1	70	25550	5.82E-07		
Zinc	1.04E+02	480	1.0E-06	1	65	1	70	25550	1.82E-06		
									TOTAL RISK		6.91E-07

TABLE : 8
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : SUBSURFACE SOIL
EXPOSURE TYPE : INGESTION/FUTURE/EXCAVATION WORKER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Ingestion Rate (mg/day)*	*kg/mg*	*Fraction Ingested*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Subchronic Body Dose (mg/kg/day)*	*Subchronic Protective Body Dose (mg/kg/day)*	*Subchronic Hazard Quotient*
Acetone	2.84E-02	480	1.0E-06	1	65	1	70	91	1.39E-07	1.00E+00	1.39E-07
Methylene Chloride	1.70E-02	480	1.0E-06	1	65	1	70	91	8.33E-08	6.00E-02	1.39E-06
Trichloroethylene	1.36E-02	480	1.0E-06	1	65	1	70	91	6.65E-08	6.00E-03	1.11E-05
Vinyl Chloride	5.75E-02	480	1.0E-06	1	65	1	70	91	2.82E-07		
4-Methylphenol	6.80E-02	480	1.0E-06	1	65	1	70	91	3.33E-07	5.00E-01	6.66E-07
bis(2-Ethylhexyl)phthalate	1.60E-01	480	1.0E-06	1	65	1	70	91	7.84E-07	2.00E-02	3.92E-05
Naphthalene	4.30E-02	480	1.0E-06	1	65	1	70	91	2.11E-07	4.00E-02	5.27E-06
Phenanthrene	6.00E-02	480	1.0E-06	1	65	1	70	91	2.94E-07		
Heptachlor epoxide	5.80E-04	480	1.0E-06	1	65	1	70	91	2.84E-09	1.30E-05	2.19E-04
Aluminum	1.94E+04	480	1.0E-06	1	65	1	70	91	9.50E-02	1.00E+00	9.50E-02
Antimony	7.82E+00	480	1.0E-06	1	65	1	70	91	3.83E-05	4.00E-04	9.57E-02
Arsenic	2.02E+01	480	1.0E-06	1	65	1	70	91	9.90E-05	1.00E-03	9.90E-02
Barium	1.60E+02	480	1.0E-06	1	65	1	70	91	7.84E-04	5.00E-02	1.57E-02
Beryllium	9.60E-01	480	1.0E-06	1	65	1	70	91	4.70E-06	5.00E-03	9.40E-04
Cobalt	1.21E+01	480	1.0E-06	1	65	1	70	91	5.92E-05		
Copper	1.08E+02	480	1.0E-06	1	65	1	70	91	5.28E-04	4.00E-02	1.32E-02
Iron	2.62E+04	480	1.0E-06	1	65	1	70	91	1.28E-01	5.00E-01	2.56E-01
Lead	1.64E+01	480	1.0E-06	1	65	1	70	91	8.01E-05		
Manganese	1.00E+03	480	1.0E-06	1	65	1	70	91	4.90E-03	1.00E-01	4.90E-02
Mercury	8.16E-02	480	1.0E-06	1	65	1	70	91	4.00E-07	3.00E-04	1.33E-03
Nickel	3.00E+01	480	1.0E-06	1	65	1	70	91	1.47E-04	2.00E-02	7.35E-03
Thallium	3.19E-01	480	1.0E-06	1	65	1	70	91	1.56E-06	7.00E-04	2.23E-03
Vanadium	3.34E+01	480	1.0E-06	1	65	1	70	91	1.64E-04	7.00E-03	2.34E-02
Zinc	1.04E+02	480	1.0E-06	1	65	1	70	91	5.11E-04	2.00E-01	2.55E-03
										HAZARD INDEX	6.62E-01

TABLE : 9
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : SEDIMENTS
EXPOSURE TYPE : INGESTION/PRESENT AND FUTURE/YOUTH TRESPASSER
RISK TYPE : CARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Ingestion Rate (mg/day)*	*kg/mg*	*Fraction Ingested*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Intake (mg/kg/day)*	*Oral Slope Factor (mg/kg/day)-1*	*Increased CA Risk*
Acetone	3.78E-02	100	1.0E-06	1	117	10	50	25550	3.46E-09		
Benzene	3.00E-03	100	1.0E-06	1	117	10	50	25550	2.75E-10	2.90E-02	7.97E-12
Methylene Chloride	4.04E-02	100	1.0E-06	1	117	10	50	25550	3.70E-09	7.50E-03	2.78E-11
Benzo(a)anthracene	2.10E-01	100	1.0E-06	1	117	10	50	25550	1.92E-08	5.79E-01	1.11E-08
Benzo(a)pyrene	2.50E-01	100	1.0E-06	1	117	10	50	25550	2.29E-08	5.79E+00	1.33E-07
bis(2-Ethylhexyl)phthalate	1.13E+00	100	1.0E-06	1	117	10	50	25550	1.03E-07	1.40E-02	1.45E-09
Phenanthrene	1.80E-01	100	1.0E-06	1	117	10	50	25550	1.65E-08		
4,4-DDE	1.71E-02	100	1.0E-06	1	117	10	50	25550	1.57E-09	3.40E-01	5.32E-10
4,4-DDT	2.85E-02	100	1.0E-06	1	117	10	50	25550	2.61E-09	3.40E-01	8.88E-10
Aldrin	2.00E-03	100	1.0E-06	1	117	10	50	25550	1.83E-10	1.70E+01	3.11E-09
Delta-BHC	5.40E-03	100	1.0E-06	1	117	10	50	25550	4.95E-10		
Dieldrin	2.25E-03	100	1.0E-06	1	117	10	50	25550	2.06E-10	1.60E+01	3.30E-09
Heptachlor epoxide	3.10E-03	100	1.0E-06	1	117	10	50	25550	2.84E-10	9.10E+00	2.58E-09
Aluminum	2.07E+04	100	1.0E-06	1	117	10	50	25550	1.89E-03		
Antimony	7.26E+00	100	1.0E-06	1	117	10	50	25550	6.65E-07		
Arsenic	2.76E+01	100	1.0E-06	1	117	10	50	25550	2.53E-06	1.75E+00	4.42E-06
Barium	1.26E+02	100	1.0E-06	1	117	10	50	25550	1.16E-05		
Beryllium	8.78E-01	100	1.0E-06	1	117	10	50	25550	8.04E-08	4.30E+00	3.46E-07
Cadmium	6.90E-01	100	1.0E-06	1	117	10	50	25550	6.32E-08		
Cobalt	1.13E+01	100	1.0E-06	1	117	10	50	25550	1.03E-06		
Copper	2.67E+01	100	1.0E-06	1	117	10	50	25550	2.44E-06		
Iron	2.91E+04	100	1.0E-06	1	117	10	50	25550	2.67E-03		
Lead	5.63E+01	100	1.0E-06	1	117	10	50	25550	5.16E-06		
Manganese	5.30E+02	100	1.0E-06	1	117	10	50	25550	4.85E-05		
Mercury	4.39E-01	100	1.0E-06	1	117	10	50	25550	4.02E-08		
Nickel	2.69E+01	100	1.0E-06	1	117	10	50	25550	2.47E-06		
Vanadium	3.56E+01	100	1.0E-06	1	117	10	50	25550	3.26E-06		
Zinc	1.55E+02	100	1.0E-06	1	117	10	50	25550	1.42E-05		
									TOTAL RISK		4.92E-06

TABLE : 10
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : SEDIMENTS
EXPOSURE TYPE : INGESTION/PRESENT AND FUTURE/YOUTH TRESPASSER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration* (mg/kg)	*Ingestion Rate* (mg/day)	kg/mg	*Fraction Ingested*	*Exposure Frequency* (days/yr)	*Exposure Duration* (years)	*Body Weight* (kg)	*Averaging Time* (days)	*Chronic Body Dose* (mg/kg/day)	*Chronic Protective Body Dose* (mg/kg/day)	*Chronic Hazard Quotient*
Acetone	3.78E-02	100	1.0E-06	1	117	10	50	3650	2.42E-08	1.00E-01	2.42E-07
Benzene	3.00E-03	100	1.0E-06	1	117	10	50	3650	1.92E-09		
Methylene Chloride	4.04E-02	100	1.0E-06	1	117	10	50	3650	2.59E-08	6.00E-02	4.32E-07
Benzo(a)anthracene	2.10E-01	100	1.0E-06	1	117	10	50	3650	1.35E-07		
Benzo(a)pyrene	2.50E-01	100	1.0E-06	1	117	10	50	3650	1.60E-07		
bis(2-Ethylhexyl)phthalate	1.13E+00	100	1.0E-06	1	117	10	50	3650	7.23E-07	2.00E-02	3.62E-05
Phenanthrene	1.80E-01	100	1.0E-06	1	117	10	50	3650	1.15E-07		
4,4-DDE	1.71E-02	100	1.0E-06	1	117	10	50	3650			
4,4-DDT	2.85E-02	100	1.0E-06	1	117	10	50	3650	1.83E-08	5.00E-04	3.66E-05
Aldrin	2.00E-03	100	1.0E-06	1	117	10	50	3650	1.28E-09	3.00E-05	4.27E-05
Dieldrin	2.25E-03	100	1.0E-06	1	117	10	50	3650	1.44E-09	5.00E-05	2.88E-05
Heptachlor epoxide	3.10E-03	100	1.0E-06	1	117	10	50	3650	1.99E-09	1.30E-05	1.53E-04
Delta-BHC	5.40E-03	100	1.0E-06	1	117	10	50	3650	3.46E-09		
Aluminum	2.07E+04	100	1.0E-06	1	117	10	50	3650	1.33E-02	1.00E+00	1.33E-02
Antimony	7.26E+00	100	1.0E-06	1	117	10	50	3650	4.66E-06	4.00E-04	1.16E-02
Arsenic	2.76E+01	100	1.0E-06	1	117	10	50	3650	1.77E-05	3.00E-04	5.90E-02
Barium	1.26E+02	100	1.0E-06	1	117	10	50	3650	8.09E-05	5.00E-02	1.62E-03
Beryllium	8.78E-01	100	1.0E-06	1	117	10	50	3650	5.63E-07	5.00E-03	1.13E-04
Cadmium	6.90E-01	100	1.0E-06	1	117	10	50	3650	4.43E-07	1.00E-03	4.43E-04
Cobalt	1.13E+01	100	1.0E-06	1	117	10	50	3650	7.23E-06		
Copper	2.67E+01	100	1.0E-06	1	117	10	50	3650	1.71E-05	4.00E-02	4.28E-04
Iron	2.91E+04	100	1.0E-06	1	117	10	50	3650	1.87E-02	5.00E-01	3.74E-02
Lead	5.63E+01	100	1.0E-06	1	117	10	50	3650	3.61E-05		
Manganese	5.30E+02	100	1.0E-06	1	117	10	50	3650	3.40E-04	1.00E-01	3.40E-03
Mercury	4.39E-01	100	1.0E-06	1	117	10	50	3650	2.81E-07	3.00E-04	9.38E-04
Nickel	2.69E+01	100	1.0E-06	1	117	10	50	3650	1.73E-05	2.00E-02	8.64E-04
Vanadium	3.56E+01	100	1.0E-06	1	117	10	50	3650	2.28E-05	7.00E-03	3.26E-03
Zinc	1.55E+02	100	1.0E-06	1	117	10	50	3650	9.94E-05	2.00E-01	4.97E-04
										HAZARD INDEX	1.33E-01

TABLE : 11
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : SEDIMENTS
EXPOSURE TYPE : INGESTION/FUTURE/EXCAVATION WORKER
RISK TYPE : CARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Ingestion Rate (mg/day)*	*kg/mg*	*Fraction Ingested*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Intake (mg/kg/day)*	*Oral Slope Factor (mg/kg/day)-1*	*Increased CA Risk*
Acetone	3.78E-02	480	1.0E-06	1	65	1	70	25550	6.59E-10		
Benzene	3.00E-03	480	1.0E-06	1	65	1	70	25550	5.23E-11	2.90E-02	1.52E-12
Methylene Chloride	4.04E-02	480	1.0E-06	1	65	1	70	25550	7.06E-10	7.50E-03	5.29E-12
Benzo(a)anthracene	2.10E-01	480	1.0E-06	1	65	1	70	25550	3.66E-09	5.79E-01	2.12E-09
Benzo(a)pyrene	2.50E-01	480	1.0E-06	1	65	1	70	25550	4.36E-09	5.79E+00	2.53E-08
bis(2-Ethylhexyl)phthalate	1.13E+00	480	1.0E-06	1	65	1	70	25550	1.97E-08	1.40E-02	2.75E-10
Phenanthrene	1.80E-01	480	1.0E-06	1	65	1	70	25550	3.14E-09		
4,4-DDE	1.71E-02	480	1.0E-06	1	65	1	70	25550	2.98E-10	3.40E-01	1.01E-10
4,4-DDT	2.85E-02	480	1.0E-06	1	65	1	70	25550	4.98E-10	3.40E-01	1.69E-10
Aldrin	2.00E-03	480	1.0E-06	1	65	1	70	25550	3.49E-11	1.70E+01	5.93E-10
Dieldrin	2.25E-03	480	1.0E-06	1	65	1	70	25550	3.93E-11	1.60E+01	6.28E-10
Heptachlor epoxide	3.10E-03	480	1.0E-06	1	65	1	70	25550	5.41E-11	9.10E+00	4.92E-10
Delta-BHC	5.40E-03	480	1.0E-06	1	65	1	70	25550	9.42E-11		
Aluminum	2.07E+04	480	1.0E-06	1	65	1	70	25550	3.61E-04		
Antimony	7.26E+00	480	1.0E-06	1	65	1	70	25550	1.27E-07		
Arsenic	2.76E+01	480	1.0E-06	1	65	1	70	25550	4.81E-07	1.75E+00	8.43E-07
Barium	1.26E+02	480	1.0E-06	1	65	1	70	25550	2.20E-06		
Beryllium	8.78E-01	480	1.0E-06	1	65	1	70	25550	1.53E-08	4.30E+00	6.58E-08
Cadmium	6.90E-01	480	1.0E-06	1	65	1	70	25550	1.20E-08		
Cobalt	1.13E+01	480	1.0E-06	1	65	1	70	25550	1.97E-07		
Copper	2.67E+01	480	1.0E-06	1	65	1	70	25550	4.66E-07		
Iron	2.91E+04	480	1.0E-06	1	65	1	70	25550	5.08E-04		
Lead	5.63E+01	480	1.0E-06	1	65	1	70	25550	9.83E-07		
Manganese	5.30E+02	480	1.0E-06	1	65	1	70	25550	9.25E-06		
Mercury	4.39E-01	480	1.0E-06	1	65	1	70	25550	7.66E-09		
Nickel	2.69E+01	480	1.0E-06	1	65	1	70	25550	4.70E-07		
Vanadium	3.56E+01	480	1.0E-06	1	65	1	70	25550	6.21E-07		
Zinc	1.55E+02	480	1.0E-06	1	65	1	70	25550	2.70E-06		

TOTAL RISK 9.38E-07

TABLE : 12
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : SEDIMENTS
EXPOSURE TYPE : INGESTION/FUTURE/EXCAVATION WORKER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Ingestion Rate (mg/day)*	*kg/mg*	*Fraction Ingested*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Subchronic Body Dose (mg/kg/day)*	*Subchronic Protective Body Dose (mg/kg/day)*	*Subchronic Hazard Quotient*
Acetone	3.78E-02	480	1.0E-06	1	65	1	70	91	1.85E-07	1.00E+00	1.85E-07
Benzene	3.00E-03	480	1.0E-06	1	65	1	70	91	1.47E-08		
Methylene Chloride	4.04E-02	480	1.0E-06	1	65	1	70	91	1.98E-07	6.00E-02	3.30E-06
Benzo(a)anthracene	2.10E-01	480	1.0E-06	1	65	1	70	91	1.03E-06		
Benzo(a)pyrene	2.50E-01	480	1.0E-06	1	65	1	70	91	1.22E-06		
bis(2-Ethylhexyl)phthalate	1.13E+00	480	1.0E-06	1	65	1	70	91	5.52E-06	2.00E-02	2.76E-04
Phenanthrene	1.80E-01	480	1.0E-06	1	65	1	70	91	8.82E-07		
4,4-DDE	1.71E-02	480	1.0E-06	1	65	1	70	91	8.38E-08		
4,4-DDT	2.85E-02	480	1.0E-06	1	65	1	70	91	1.40E-07	5.00E-04	2.80E-04
Aldrin	2.00E-03	480	1.0E-06	1	65	1	70	91	9.80E-09	3.00E-05	3.27E-04
Dieldrin	2.25E-03	480	1.0E-06	1	65	1	70	91	1.10E-08	5.00E-05	2.20E-04
Heptachlor epoxide	3.10E-03	480	1.0E-06	1	65	1	70	91	1.52E-08	1.30E-05	1.17E-03
Delta-BHC	5.40E-03	480	1.0E-06	1	65	1	70	91	2.64E-08		
Aluminum	2.07E+04	480	1.0E-06	1	65	1	70	91	1.01E-01	1.00E+00	1.01E-01
Antimony	7.26E+00	480	1.0E-06	1	65	1	70	91	3.56E-05	4.00E-04	8.89E-02
Arsenic	2.76E+01	480	1.0E-06	1	65	1	70	91	1.35E-04	1.00E-03	1.35E-01
Barium	1.26E+02	480	1.0E-06	1	65	1	70	91	6.18E-04	5.00E-02	1.24E-02
Beryllium	8.78E-01	480	1.0E-06	1	65	1	70	91	4.30E-06	5.00E-03	8.60E-04
Cadmium	6.90E-01	480	1.0E-06	1	65	1	70	91	3.38E-06	1.00E-03	3.38E-03
Cobalt	1.13E+01	480	1.0E-06	1	65	1	70	91	5.53E-05		
Copper	2.67E+01	480	1.0E-06	1	65	1	70	91	1.31E-04	4.00E-02	3.27E-03
Iron	2.91E+04	480	1.0E-06	1	65	1	70	91	1.43E-01	5.00E-01	2.86E-01
Lead	5.63E+01	480	1.0E-06	1	65	1	70	91	2.76E-04		
Manganese	5.30E+02	480	1.0E-06	1	65	1	70	91	2.60E-03	1.00E-01	2.60E-02
Mercury	4.39E-01	480	1.0E-06	1	65	1	70	91	2.15E-06	3.00E-04	7.16E-03
Nickel	2.69E+01	480	1.0E-06	1	65	1	70	91	1.32E-04	2.00E-02	6.60E-03
Vanadium	3.56E+01	480	1.0E-06	1	65	1	70	91	1.74E-04	7.00E-03	2.49E-02
Zinc	1.55E+02	480	1.0E-06	1	65	1	70	91	7.59E-04	2.00E-01	3.80E-03

HAZARD INDEX 7.02E-01

TABLE : 13
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : SEDIMENTS
EXPOSURE TYPE : DERMAL CONTACT/PRESENT AND FUTURE/YOUTH TRESPASSER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*kg/mg*	*Skin Surface Area Contacted (cm2/day)*	*Soil Skin Adherence Factor (mg/cm2)*	*Abs. Factor*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Chronic Body Dose (mg/kg/day)*	*Chronic Protective Body Dose (mg/kg/day)*	*Chronic Hazard Quotient*
Cadmium	6.90E-01	1.0E-06	7720	0.6	0.005	117	10	50	3650	1.02E-07	5.00E-05	2.05E-03
									HAZARD INDEX			2.05E-03

TABLE : 14
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : SEDIMENTS
EXPOSURE TYPE : DERMAL CONTACT/FUTURE/EXCAVATION WORKER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*kg/mg*	*Skin Surface Area Contacted (cm2/day)*	*Soil Skin Adherence Factor (mg/cm2)*	*Abs. Factor*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Subchronic Body Dose (mg/kg/day)*	*Subchronic Protective Body Dose (mg/kg/day)*	*Subchronic Hazard Quotient*
Cadmium	6.90E-01	1.0E-06	3120	0.6	0.005	65	1	70	91	6.59E-08	5.00E-05	1.32E-03
										HAZARD INDEX		1.32E-03

TABLE : 15
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : LEACHATE SOILS
EXPOSURE TYPE : INGESTION/PRESENT AND FUTURE/YOUTH TRESPASSER
RISK TYPE : CARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Ingestion Rate (mg/day)*	*kg/mg*	*Fraction Ingested*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Intake (mg/kg/day)*	*Oral Slope Factor (mg/kg/day)-1*	*Increased CA Risk*
1,4-Dichlorobenzene (para)	1.20E+00	100	1.0E-06	1	5	10	50	25550	4.70E-09	2.40E-02	1.13E-10
Methylene Chloride	6.70E-02	100	1.0E-06	1	5	10	50	25550	2.62E-10	7.50E-03	1.97E-12
4-Methylphenol	4.40E-01	100	1.0E-06	1	5	10	50	25550	1.72E-09		
Benzo(a)anthracene	2.10E-01	100	1.0E-06	1	5	10	50	25550	8.22E-10	5.79E-01	4.76E-10
bis(2-Ethylhexyl)phthalate	7.50E-01	100	1.0E-06	1	5	10	50	25550	2.94E-09	1.40E-02	4.11E-11
Naphthalene	2.40E-01	100	1.0E-06	1	5	10	50	25550	9.39E-10		
Phenanthrene	4.70E-01	100	1.0E-06	1	5	10	50	25550	1.84E-09		
Aldrin	2.10E-03	100	1.0E-06	1	5	10	50	25550	8.22E-12	1.70E+01	1.40E-10
4,4-DDE	6.90E-04	100	1.0E-06	1	5	10	50	25550	2.70E-12	3.40E-01	9.18E-13
Dieldrin	3.20E-03	100	1.0E-06	1	5	10	50	25550	1.25E-11	1.60E+01	2.00E-10
Delta-BHC	2.10E-03	100	1.0E-06	1	5	10	50	25550	8.22E-12		
Aluminum	1.17E+04	100	1.0E-06	1	5	10	50	25550	4.58E-05		
Arsenic	1.20E+01	100	1.0E-06	1	5	10	50	25550	4.70E-08	1.75E+00	8.22E-08
Barium	1.10E+02	100	1.0E-06	1	5	10	50	25550	4.31E-07		
Beryllium	6.00E-01	100	1.0E-06	1	5	10	50	25550	2.35E-09	4.30E+00	1.01E-08
Cadmium	7.10E-01	100	1.0E-06	1	5	10	50	25550	2.78E-09		
Cobalt	6.80E+00	100	1.0E-06	1	5	10	50	25550	2.66E-08		
Copper	4.78E+01	100	1.0E-06	1	5	10	50	25550	1.87E-07		
Iron	2.57E+04	100	1.0E-06	1	5	10	50	25550	1.01E-04		
Lead	1.10E+02	100	1.0E-06	1	5	10	50	25550	4.31E-07		
Manganese	5.11E+02	100	1.0E-06	1	5	10	50	25550	2.00E-06		
Mercury	1.20E+00	100	1.0E-06	1	5	10	50	25550	4.70E-09		
Nickel	1.87E+01	100	1.0E-06	1	5	10	50	25550	7.32E-08		
Vanadium	2.31E+01	100	1.0E-06	1	5	10	50	25550	9.04E-08		
Zinc	1.19E+02	100	1.0E-06	1	5	10	50	25550	4.66E-07		

TOTAL RISK 9.33E-08

TABLE : 16
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : LEACHATE SOILS
EXPOSURE TYPE : INGESTION/PRESENT AND FUTURE/YOUTH TRESPASSER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Ingestion Rate (mg/day)*	*kg/mg*	*Fraction Ingested*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Chronic Body Dose (mg/kg/day)*	*Chronic Protective Body Dose (mg/kg/day)*	*Chronic Hazard Quotient*
1,4-Dichlorobenzene (para)	1.20E+00	100	1.0E-06	1	5	10	50	3650	3.29E-08	1.00E-01	3.29E-07
Methylene Chloride	6.70E-02	100	1.0E-06	1	5	10	50	3650	1.84E-09	6.00E-02	3.06E-08
4-Methylphenol	4.40E-01	100	1.0E-06	1	5	10	50	3650	1.21E-08	5.00E-02	2.41E-07
Anthracene	1.90E-01	100	1.0E-06	1	5	10	50	3650	5.21E-09	3.00E-01	1.74E-08
Benzo(a)anthracene	2.10E-01	100	1.0E-06	1	5	10	50	3650	5.75E-09		
bis(2-Ethylhexyl)phthalate	7.50E-01	100	1.0E-06	1	5	10	50	3650	2.05E-08	2.00E-02	1.03E-06
Naphthalene	2.40E-01	100	1.0E-06	1	5	10	50	3650	6.58E-09	4.00E-03	1.64E-06
Phenanthrene	4.70E-01	100	1.0E-06	1	5	10	50	3650	1.29E-08		
Aldrin	2.10E-03	100	1.0E-06	1	5	10	50	3650	5.75E-11	3.00E-05	1.92E-06
4,4-DDE	6.90E-04	100	1.0E-06	1	5	10	50	3650	1.89E-11		
Dieldrin	3.20E-03	100	1.0E-06	1	5	10	50	3650	8.77E-11	5.00E-05	1.75E-06
Delta-BHC	2.10E-03	100	1.0E-06	1	5	10	50	3650	5.75E-11		
Aluminum	1.17E+04	100	1.0E-06	1	5	10	50	3650	3.21E-04	1.00E+00	3.21E-04
Arsenic	1.20E+01	100	1.0E-06	1	5	10	50	3650	3.29E-07	3.00E-04	1.10E-03
Barium	1.10E+02	100	1.0E-06	1	5	10	50	3650	3.01E-06	5.00E-02	6.03E-05
Beryllium	6.00E-01	100	1.0E-06	1	5	10	50	3650	1.64E-08	5.00E-03	3.29E-06
Cadmium	7.10E-01	100	1.0E-06	1	5	10	50	3650	1.95E-08	1.00E-03	1.95E-05
Cobalt	6.80E+00	100	1.0E-06	1	5	10	50	3650	1.86E-07		
Copper	4.78E+01	100	1.0E-06	1	5	10	50	3650	1.31E-06	4.00E-02	3.27E-05
Iron	2.57E+04	100	1.0E-06	1	5	10	50	3650	7.04E-04	5.00E-01	1.41E-03
Lead	1.10E+02	100	1.0E-06	1	5	10	50	3650	3.01E-06		
Manganese	5.11E+02	100	1.0E-06	1	5	10	50	3650	1.40E-05	1.00E-01	1.40E-04
Mercury	1.20E+00	100	1.0E-06	1	5	10	50	3650	3.29E-08	3.00E-04	1.10E-04
Nickel	1.87E+01	100	1.0E-06	1	5	10	50	3650	5.12E-07	2.00E-02	2.56E-05
Vanadium	2.31E+01	100	1.0E-06	1	5	10	50	3650	6.33E-07	7.00E-03	9.04E-05
Zinc	1.19E+02	100	1.0E-06	1	5	10	50	3650	3.26E-06	2.00E-01	1.63E-05
										HAZARD INDEX	3.33E-03

TABLE : 17
SITE : NIAGARA COUNTY REFUSE
ENV. MEDIUM : LEACHATE SOILS
EXPOSURE TYPE : DERMAL CONTACT/PRESENT AND FUTURE/YOUTH TRESPASSER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*kg/mg*	*Skin Surface Area Contacted (cm2/day)*	*Soil Skin Adherence Factor (mg/cm2)*	*Abs. Factor*	*Exposure Frequency (days/yr)*	*Exposure Duration (years)*	*Body Weight (kg)*	*Averaging Time (days)*	*Chronic Body Dose (mg/kg/day)*	*Chronic Protective Body Dose (mg/kg/day)*	*Chronic Hazard Quotient*
Cadmium	7.10E-01	1.0E-06	7720	0.6	0.005	5	10	50	3650	4.51E-09	5.00E-05	9.01E-05
									HAZARD INDEX			9.01E-05

APPENDIX F

TOXICITY PROFILES

VOLATILES

ACETONE

Use

Acetone is used as a solvent industrially and domestically. The most common household use is as a paint thinner. It is also used industrially in the manufacture of lubricating oil, chloroform, pesticides, and pharmaceuticals (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: $(CH_3)2CO$

MW:	58.08	MP:	-95°C
SG:	0.7899 at 20°C	BP:	56.2°C
FP:	-16°C	VP:	190 mmHg at 20°C

Sol.(water): Miscible

Sol.(organics): Alcohol, ether, chloroform, benzene

Fate and Transport

Acetone is a highly volatile compound, but because of its high solubility in water, it would be expected to remain in solution rather than volatilize from surface soils. Biodegradation and leaching are the two major fate processes in soils (ICF, 1985).

In the atmosphere, acetone degrades rapidly and, in surface waters, acetone would be expected to remain in solution and eventually settle into the sediments (EPA, 1984).

Pharmacokinetics

Dalhamn, et al. (1968) reported that 60 percent of the acetone inhaled in cigarette smoke was absorbed by humans within 2 seconds. The primary route of absorption appeared to be through the blood. Several studies report that absorption is doubled with light exercise. Absorbed acetone is eliminated through expired air and is excreted in the urine (EPA, 1984).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Limited information regarding oral exposure to acetone was located in the available literature. In a ninety-day gavage study in albino rats conducted by EPA (1986), increased liver weight and tubular degeneration of the kidneys were observed in the 2500 mg/kg group. There were no observed effects at 100 mg/kg/day. Sollman (1921) exposed rats to acetone in drinking water and,

upon termination of the experiment, the test animals were found to be in normal health. In oral exposure studies, laboratory animals exhibited signs of narcosis but no biochemical or histological changes were seen. In two studies, Parmeggiani and Sassi (1954) and Raleigh and McGee (1972) performed case studies on workers who were chronically exposed to 19-220 ppm and >750 ppm acetone, respectively. Both groups of workers complained of irritation of the mucosal membranes, including conjunctivitis, pharyngitis, bronchitis, and gastroduodenitis. In a more detailed experiment, Oglesby et al. (1949) regularly examined 800 men occupationally exposed to <2150 ppm acetone for 8 hours/day. No differences were noted between the workers and 800 control subjects.

Teratogenic and Other Developmental Effects

Acetone is known to cross the placental barrier, but, due to its low toxicity it would not be expected to cause any fetotoxic effects (EPA, 1984). No additional data regarding the teratogenicity of acetone were located in the available literature.

Mutagenic Effects

In seven studies reviewed by the U.S. EPA, only one indicated that acetone causes mutagenic effects. Kawachi, et al. (1980) reported that acetone caused chromosomal aberrations in unspecified cells. Acetone tested negatively for sister-chromatid-exchange, point mutations and cell binding in mouse lymphoma cells (EPA, 1984).

Carcinogenic Effects

U.S. EPA has considered the evidence regarding the carcinogenic effects of acetone to be inadequate. One experiment involving the effects of acetone painted on an unspecified laboratory animal gave negative carcinogenic results (EPA, 1984). No further data regarding the carcinogenicity of acetone were found in the available literature.

Ecotoxicity

The toxicity of acetone to aquatic organisms is known to be low. The LC_{50} value for sunfish is reported to be 14.2 g/l. Acetone's effects on terrestrial species have not been well documented, although it is assumed that they are similar to the effects on laboratory animals.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	$1x10^{-1}$ mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	$1 X 10^{0}$ mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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BENZENE

Use

Benzene is a clear, colorless, aromatic hydrocarbon that possesses a distinct sweet odor (NIOSH, 1987). It is extremely flammable and volatile. Benzene is widely used in the production of other industrial compounds, such as styrene and phenols, and in making common household materials such as rubber, plastic, and inks (Sittig, 1991). Exposure to the public is mainly through the combustion of gasoline and through cigarette smoke.

Chemical and Physical Properties

Chemical Formula: $C_6 H_6$

MW:	78	BP:	80.1°C
SG:	0.879 at 20°C	MP:	5.56°C
FP:	-11.1°C	VP:	100 mmhg at 26°C

Sol. (water): 1780 mg/l at 25°C

Sol. (organics): Miscible with ethanol, ether acetic acid, acetone, chloroform, carbon disulfide, and carbon tetrachloride.

Odor Threshold: 2 ppm (EPA, 1985)

Fate and Transport

Because of its volatility, benzene is photo-oxidized very rapidly in the atmosphere, with a half-life of less than 1 day (EPA, 1985). In moist soils, benzene is retained and slowly transported into the ground water, where it is known to remain stable (EPA, 1985). The bioaccumulation of benzene appears to be relatively low.

Pharmacokinetics

Benzene is highly lipid soluble and is readily absorbed via inhalation, ingestion, and dermal contact. Approximately 50 percent of benzene is absorbed through inhalation. Absorbed benzene is readily distributed to fatty tissues where it is sequestered. The prime target organ is the bone marrow. Detoxification of bodily fluids occurs in the liver. The major metabolite of benzene in humans is phenol sulfite, but it can also be excreted unchanged.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Target organs of benzene include bone marrow, central nervous tissues, and the respiratory system (NIOSH, 1987).

In a work environment, benzene can be acutely fatal to humans in extremely high concentrations (20,000 ppm) and can cause short-term effects such as dizziness and vomiting in humans exposed to lower levels (Sittig, 1991).

In elevated concentrations, benzene can be fatal within minutes (EPA, 1985). Initially, benzene causes central nervous system depression, followed by acute arrythmia and cardiovascular collapse. If benzene in a liquid state is aspirated into the lung, immediate pulmonary edema and hemorrhaging can occur (Sittig, 1991). Benzene is known to cause aplastic anemia, leukopenia, and thrombocytopenia in humans as a result of chronic exposure. The latency period can be as long as 10 years (Sittig, 1991).

Both gavage and inhalation exposure of rodents to benzene have resulted in the development of neoplasia. The incidence of tumors, carcinomas, leukemias, and lymphomas have been shown to be directly proportional to the dosage administered. (EPA, 1985).

Teratogenic and Other Developmental Effects

Benzene is known to be a potent inhibitor of growth *in utero*. It has also been shown to cause chromosomal aberrations in the bone marrow cells of workers as a result of chronic exposure (EPA, 1985).

Pregnant mice were given gavage doses of benzene at levels between 0.3 and 1.0 mg/kg daily on days 6-15 of gestation. Increased mortality among the dams and resorption of embryos occurred for all levels (EPA, 1984). No teratogenic effects were noted in rabbits, rats, or fruit flies exposed to less than 550 ppm in the air.

Mutagenic Effects

Signs of mutagenicity amongst rabbits, mice, rats, and fruit flies were not seen when they were exposed to benzene levels of 550 ppm in the air.

Carcinogenic Effects

Benzene is classified as a Class A human carcinogen. Aksoy, et al. reported effects of benzene exposure among 28,500 Turkish employed in the shoe industry. The peak exposure was reported to be 210-650 ppm over a period of 10 years. An incidence of 13/100,000 cases was reported for the workers, compared to 6/100,000 for the general public (IRIS).

In a second study, the effects of benzene exposure on 748 white males employed in a rubber manufacturing industry were examined.. A statistically significant increase in the mortality rate due to leukemia was noted for the population. There was no evidence of exposure to solvents other than benzene (IRIS).

Rinsky, et al. (1981) noted a statistically significant increase in the number of leukemia deaths in workers occupationally exposed to benzene. Exposure was estimated to be between 10 and 100 ppm, 8-hour TWA. Ott, et al. (1978) noted an increase in leukemia deaths in workers exposed to between 2 to 25 ppm, 8-hour TWA. The increase, however, was determined by EPA to be insignificant (IRIS, 1990). Wong, et al. (1983) noted statistically significant increases in leukemias, lymphatic, and hematopoietic cancers in chemical plant workers exposed to benzene for at least 6 months. No statistical details were reported (IRIS).

Benzene was shown to have caused leukemia directly, or as a result of bone marrow abnormalities in Sprague-Dawley rats (Maltoni and Scartano, 1979). Benzene was administered by gavage at doses of 50 and 250 mg/kg bw for 4 to 5 days/week for 52 weeks. Twenty five percent developed Zymbal gland tumors, 6.2 percent had skin carcinomas, and 12.1 percent had leukemias at the end of the 52 weeks (IRIS). Dogs exposed to 600 to 1000 ppm benzene for 12-15 days also developed leukemia (EPA, 1985).

Ecotoxicity

In aquatic toxicity bioassays, the EC_{50} values for benzene in a variety of invertebrate and vertebrate freshwater aquatic species range between 5300 µg/L and 386,000 µg/L. In these studies, however, only those performed on rainbow trout were obtained from a flow through test and were based on measured concentrations. Results based on unmeasured concentrations in static tests are likely to underestimate the toxicity for relatively volatile compounds, such as benzene. A chronic test on *Daphnia magna* was determined to be incomplete, however in this test, no adverse effects were observed at test concentrations as high as 98,000 µg/L (EPA, 1980).

Standards, Criteria and Guidelines

EPA Class A carcinogen

Oral Slope Factor:	2.9×10^{-2} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	2.9×10^{-2} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	Currently under review by EPA
Chronic Inhalation RfD:	Currently under review by EPA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	5.0 µg/L
AWQC:	Water and Fish Consumption - 0.66 µg/L
	Fish Consumption - 40 µg/L

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BROMOCHLOROMETHANE

Use

Bromochloromethane is a colorless, volatile liquid with a chloroform-like odor (U.S. EPA, 1988). It is produced by partial replacement of chloride in dichloromethane by reaction with anhydrous AlBr3, treatment with Br and Al, or by reaction with HBr followed by water washing and distillation. Bromochloromethane is used primarily as a fire-extinguishing fluid, particularly in aircraft and portable extinguishers (Stenger, 1978).

Chemical and Physical Properties

MF: CH_2BrCl MP: -86.5°C
MW: 129.39 BP: 68.1°C
Sol. (water): 0.9 g/100 g at 25°C
Sol. (organics): miscible or soluble in alcohols, ethers, and benzene

Fate and Transport

Mabey and Mill (1978) reported a half-life of 44 years for bromochloromethane in water at pH 7 and 25°C. They also reported a first-order hydrolysis rate constant of 5.0 x 10^{-10}/sec. indicating that hydrolysis is not a significant fate for this compound. Cadmand and Simons (1966) have predicted that photolysis of bromochloromethane is not a significant fate either. Studies by Tabak et al. (1981) indicate that biodegradation is a significant fate for bromochloromethane in aquatic media. The U.S. EPA (1985) reports that volatilization of bromochloromethane from aquatic media is rapid and significant.

The atmospheric chemical removal processes include reaction of bromochloromethane with different free radicals (OH·, 0·, Cl·, etc.), with ozone, or photolytic transformations (U.S. EPA, 1985). The U.S. EPA (1985) reports that atmospheric residence times for bromochloromethanes are less than two years. Cadman and Simons (1966) predict that photolysis of bromochloromethane in the atmosphere is not a significant fate. Physical removal processes, such as dissolution, adsorption on aerosol particles, and dry deposition, are not likely to be significant fates for atmospheric bromochloromethane either (U.S. EPA, 1985).

Calculations by Kenoga and Goring (1978) indicate that bromochloromethane has medium-to-high soil mobility. Therefore, leaching of bromochloromethane into ground water may occur. Wilson et al. (1981) has shown volatilization to be an important fate of bromochloromethane in soil. The U.S. EPA (1985) states that microbial degradation of bromochloromethane in soil may be in important fate.

Pharmacokinetics

The U.S. EPA (1985) reports that toxicity studies indicate that bromochloromethane is absorbed following ingestion. Anderson et al. (1980) found the maximum uptake of bromochloromethane (119 ppm) by rats in an inhalation chamber to be 11.4 mg/kg/hr.

Pfaffenberger et al. (1979) found that a similar halogenomethane, bromodichloromethane became slightly concentrated in the serum and adipose tissue of rats when administered by gavage, but had virtually disappeared from these tissues within 3-6 days.

Studies by Kubic et al. (1974) indicate that circulating carboxyhemoglobin is involved in the metabolism of bromochloromethane and that carbon monoxide is one of the products.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Torkelson et al. (1960) exposed groups of 20 male and 20 female rats, 10 male and 10 female guinea pigs, 2 male and 2 female rabbits and 10 female mice to 0, 500, 1000 ppm bromochloromethane by inhalation, for 7 hours/day, 5 days/week for 4-6 months. Additionally, a group of 10 female rats and 1 male and 1 female dog were exposed to 370 ppm bromochloromethane for 7 hours/day, 5 days/week for 4-6 months.

At the higher exposure levels, male and female rats exhibited increased liver and kidney weights (500 ppm, 1000 ppm) and hepatic histopathology (1000 ppm). The females exhibited liver histopathology at the 500 ppm level. The female rats exposed to 370 ppm exhibited no adverse effects other than increased liver weights. Torkelson et al. (1960) ascribed the increased liver weight to stress. Blood bromide levels were elevated in rats of both sexes at the higher exposure levels and in females at 370 ppm. Female guinea pigs exhibited an increased number of presumably circulating neutrophils, decreased body weight, and increased relative liver and kidney weights at the highest exposure level. Male guinea pigs also experienced these weight changes in addition to decreased spermatogenesis. These same changes occurred to a lesser degree in the 500 ppm-exposed-guinea pigs. One male rabbit exhibited decreased spermatogenesis at the 1000 ppm level. The 10 female mice exhibited decreased body weights and increased relative liver and kidney weights at the higher exposure levels. No adverse effects were exhibited by the dogs.

Teratogenic and Other Developmental Effects

Data regarding the teratogenicity of bromochloromethane was not found in the literature reviewed. However, data on the teratogenic effects of other bromochloromethanes was found.

Ruddick et al. (1983) exposed groups of 9-14 Sprague-Dowley rats to 0, 50, 100, or 200 mg/kg/day of either dibromochloromethane or bromodichloromethane on days 6-15 of gestation. The only adverse effects exhibited by the dams was increased liver and kidney weights and decreased body weights at the higher dose levels. The fetuses exhibited no significant adverse effects.

Groups of 10 male and 30 female IRC mice as well as subsequent generations were exposed to dibromochloromethane in emolphor (a polyoxyethylated nonionic surfactant oil) at 0, 0.1, 1.0, and 4.0 mg/ml concentrations in drinking water (Borzelleca and Carchman, 1982). Survival of the F1 generation was reduced in mice treated with 4 mg/ml. Additionally, litter size, gestation index, and the viability of the pups was also significantly reduced. Lactation index and postnatal body weight were reduced in the F2b generation after exposure to 4 mg/ml dibromochloromethane. No significant increase in malformations was reported at any dose level. At the 1 mg/ml concentration, significant decreases in litter size, pup viability, postnatal body weight and lactation index occurred.

Mutagenic Effects

IRIS reports that mutagenicity tests with bromochloromethane in microorganisms have yielded consistently positive results. Ostermann-Golkar et al. (1983) found bromochloromethane to be mutagenic in reverse mutation assays with *Salmonella typhimurium* TA100 and TA1535 in the absence of activation at 20-60 mm concentrations, as well as with or without activation in the vapor phase at 10 µliter/plate. Simmon (1978), Simmon et al. (1977), and Simmon and Tardiff (1978) also reported positive results in *S. typhimurium* exposed without activation to bromochloromethane at 10-50 µliter/desiccator.

When exposed without activation to 10 µliter/plate bromochloromethane, *Escherichia coli* WU361089 exhibited reverse mutations at the tyr locus and *E. coli* K394 exhibited prophage induction (Ostermann-Golkar et al., 1983).

Carcinogenic Effects

Data regarding the carcinogenicity of bromochloromethane were not found in the literature reviewed. However, the U.S. EPA (1985) reported on two studies that suggest a relationship between exposure to the bromochloromethanes, bromodichloromethane and dibromochloromethane and cancer.

Ecotoxicity

Data regarding the ecotoxicity of bromochloromethane were not found in the literature reviewed.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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2-BUTANONE

Use

2-Butanone, also known as methyl ethyl ketone, is a clear, colorless liquid with a fragrant, somewhat sharp, mint-like odor (Sittig, 1991). It is used as a solvent in vinyl film manufacture and nitrocellulose coating, in smokeless powder manufacture, in cements and adhesives, and in the dewaxing of lubricating oils (US EPA, 1980). It is also an intermediate in drug manufacture (Sittig, 1991).

Chemical and Physical Properties

Chemical formula: $CH_3COCH_2CH_3$ BP: 79-80°C
MW: 72.1 VP: 77.5 mmHg at 20°C
Sol. (water): 268 g/liter at 20°C
Sol. (organics): miscible with alcohol, ether, benzene

Fate and Transport

Lande et al. (1976), having determined an evaporative half-life for 2-butanone from water to be ~ 6 days, concluded that 2-butanone may biodegrade almost completely within 10 days from most surface waters.

The US EPA (1984) stated that 2-butanone is expected to have a high soil mobility based on its relatively high water solubility and a low octanol/water partition coefficient of 0.26. They also state that volatilization and biodegradation may account for significant loss of 2-butanone from soils. It has been speculated, based on occurrences in aquatic media, that the half-life of 2-butanone in soils is on the order of a few days (US EPA, 1984).

Pharmacokinetics

In a study of the dermal absorption of 2-butanone in humans, Mins and Wurster (1965) applied 100 ml of 2-butanone to the forearm under normal, hydrated, and dehydrated skin conditions. Using an absorption cell, the exposure lasted 8 hours. 15 minutes after exposure, a 3.6 mg/liter concentration of 2-butanone was detected in expired air and 2-3 hours after exposure a steady-state level of 6.5-6.6 mg/liter in expired air was achieved. One hour after administering a single intraperitoneal dose of 450 mg/kg, DiVincenzo and Krasavage (1974) found 11 percent of administered 2-butanone and metabolites in the serum.

4 hours after administering a single oral dose of 355 mg/kg 2-butanone in rats, Dietz and Traiger (1979) found blood concentration of 2-butanone, 2-butanol, 3-hydroxy-2-butanone, and 2,3-butanediol to be 94.1 mg/100 ml, 3.2 mg/100 ml, 2.4 mg/100 ml, and 8.6 mg/100 ml respectively.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

LaBelle and Brieger (1955) exposed 25 rats to 235 ppm of 2-butanone for 7 hrs/day, 5 days/week for 12 weeks. No effects were observed although only a few parameters were measured.

Similarly, Saida et al. (1976) found no signs of neuropathy, defined in terms of paralysis, in rats exposed to 1125 ppm concentration continuously for 5 months.

Takeuchi et al. (1983) found only slight neurological effects in rats exposed to 2-butanone at a level of 200 ppm for 12 hrs/day, 7 days/week for 24 weeks. Effects did not surface until 4 months of treatment. In an inhalation study, Cavender et al. (1983) exposed rats of both sexes to 0, 1250, 2500, or 5000 ppm concentrations of 2-butanone for 6 hrs/day, 5 days/week for 90 days. No effects were reported at the 1250 ppm level but female rats in the 2500 ppm group experienced elevated SGPT activity. A host of effects occurred at the 5000 ppm level including depressed mean body weight; slight increases in liver weight, liver to body weight ratio, and liver to brain weight ratio; and significantly decreased SGPT activity. Females also experienced increased alkaline phosphatase, potassium and glucose values.

Teratogenic and Other Developmental Effects

Schwartz et al. (1974) found no maternal toxicity when pregnant Sprague-Dawley rats were exposed to 2-butanone by inhalation at concentrations of 100 or 3000 ppm for 7 hrs/day during days 6-15 of gestation. However, somewhat decreased fetal body measurements and a significant increase in litters having fetuses with skeletal abnormalities were detected in the lower exposure level and a significant increase in litters having fetuses with gross external anomalies or internal soft-tissue anomalies was observed in the higher exposure level.

Mutagenic Effects

Florin et al. (1980) and Douglas et al. (1980) found that 2-butanone was not mutagenic in *S. typhimurium* strains TA98, TA100, TA1535, or TA1537 with or without rat hepatic homogenates. However, Zimmerman at al (1985) reported that 2-butanone induced aneuploidy in the diploid D61, M strain of *Saccharomyces cerevisae.*

Carcinogenic Effects

Pertinent data regarding the carcinogenicity of 2-butanone were no located in the available literature. 2-Butanone is designated Group D (not-classified) for the weight-of-evidence category for potential carcinogens.

Ecotoxicity

Wallen et al. (1957) found an LC_{50} concentration for Lanbusia affinis around 5600 µg/liter. Similar results were found in other studies on other freshwater fish (Turball et al., 1954). 2-Butanone was toxic to brine shrimp at LC_{50} levels of 1950 mg/liter (ICP, 1985).

Pertinent data regrading the toxicity of 2-butanone to terrestrial life, wild or domestic, was not found in the literature reviewed.

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	5.0×10^{-2} mg/kg/day
Chronic Inhalation RfD:	9.0×10^{-2} mg/kg/day
Subchronic Oral RfD:	5.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	9.0×10^{-1} mg/kg/day
MCL:	NA
AWQC:	NA

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A91-278.1

CARBON DISULFIDE

Use

Carbon disulfide is also known as carbon bisulfide and dithiocarbonic anhyride (U.S. EPA, 1985). It is a clear, colorless, very flammable and highly volatile liquid that has a mild, ethereal order when pure, but a disagreeable sulfurous odor when impure (Timmerman, 1978). Carbon disulfide is produced by high temperature reaction of methane and sulfur gas in the presence of a catalyst such as charcoal, Cr, W, and Mo compounds, and oxides, sulfides of metals (Timmerman, 1978). It is used in the manufacture of rayon, cellophane, carbon tetrachloride (Timmerman, 1978) and a variety of other uses including manufacture of rubber chemicals and flotation agents, ammonium thiocyanate, sodium thiocyanate, xanthogenates, electronic vacuum tubes, use as an insecticide (fumigant) and as a solvent for phosphorus, sulfur, selenium, bromine, iodine fats, resins, and rubber (CMR, 1983; Berg, 1981; Windholz, 1983).

Chemical and Physical Properties

Chemical Formula: CS_2

MW: 76.13
Sol. (water): 2,940 mg/liter
Sol. (organics): miscible with anhydrous methanol, ethanol, ether, benzene, chloroform, carbon tetrachloride, and oil

BP: 46.3°C
MP: -108.6 to -116.6°C
VP: 297 mmHg at 20°C

Fate and Transport

In aquatic media, hydrolysis of carbon disulfide is not an environmentally significant event (Peyton et. al., 1978). Peyton et. al. (1978) estimated the evaporation $t_{1/2}$ of carbon disulfide from saturated water to be 11 minutes. U.S. EPA (1986) states the volitization should be a rapid and important removal process based on Henry's Law constant. It appears from BCF estimations that carbon disulfide will not bioaccumulate (U.S. EPA, 1986).

Atkinson et. al. (1978) and Wood and Heicklen (1971) found that carbon disulfide does not photolyze directly under atmospheric conditions. The U.S. EPA (1986) reports that experimental data indicate that carbon disulfide is removed from the troposphere before it can enter the stratosphere.

Being a soil disinfectant, carbon disulfide is unlikely to biodegrade significantly in soils and hydrolysis on wet soil surfaces is also unlike (U.S. EPA, 1986). The expected volatilization of carbon disulfide from water suggests that this may be a major escape route from soils as well (U.S. EPA, 1986). In a study of the adsorption gaseous carbon disulfide onto soils, Bremner and Banwart (1976) suggest that soils have little if any potential for removing carbon disulfide from industrial emissions.

Pharmacokinetics

Numerous studies have shown that carbon disulfide is absorbed extensively from the lungs in humans and animals and that a steady-state is achieved between inhaled and exhaled carbon disulfide within one to two hours of exposure (U.S. EPA, 1986). Carbon disulfide and its metabolites were distributed rapidly to body fat and highly perfused tissues in studies on rats and mice by McKenna and DiStefano (1977) and Bergman et. al. (1984). It was also reported that free carbon disulfide is eliminated more rapidly from the tissues than its metabolites (McKenna an DiStefano, 1977). Bioacumulation in any tissue does not appear significant for free carbon disulfide or its metabolites (U.S. EPA, 1986).

The U.S. EPA (1986) reported on numerous studies that suggest two major routes for the metabolism of carbon disulfide: reaction with amino acids or reduced glutathione to form thiocarbamates or conjugated glutathione, and by reactions catalyzed by cytochrome P450 to form reactive sulfur which may react further to form thiourea, carbonyl sulfide, or monothiocarbonate.

Excretion of carbon disulfide occurs rapidly in expired air, urine and milk (U.S. EPA, 1986). Numerous studies have shown that unchanged carbon disulfide is eliminated principally through the lungs with a small percentage being eliminated in the urine while its metabolites are excreted more slowly and primarily through the urine (U.S. EPA, 1986).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Pilarska et. al. (1973) exposed 13 Wistar rats to 25 mg/kg/day carbon disulfide in arachis oil for 60 days. Carbon disulfide-exposed rats had normochromic and normocytic anemia, eosinopenia, and reticulocytosis. Dietzmann and Laas, as cited by U.S. EPA (1986), examined the tissues of the brain and spinal cord of male Wistar rats historically to study the effects of carbon disulfide on the CNS. The rats were treated by gavage with 0.06ml carbon disulfide/rat twice weekly for eight weeks and then twice weekly with 0.12nl carbon disulfide/rat for an additional 12 weeks. Exposed rats were killed at 4, 8, 12, 16, and 20 weeks. During the first few weeks, rats exhibited disorientation and a "reeling gait" that disappeared after three weeks. Hair loss occurred at 12 weeks and after 16 weeks of treatment, paraplegia of the hind and fore limbs occurred in "several animals". Histologically; destruction of ganglion cells in the cerebrum, cerebellum, and brain stem; elective parenchymal necrosis in frontal and parietal cortices; "micro-maculated fresh hemorraghic extravasates in the areas of nucleus rubber and pans"; and axonal swelling and destruction of the myelin sheath occurred after 12 weeks. Enzymatic changes (reductions in

activities of succinic dehydrogenase and acetylcholinesterase, arylsulfatases, and glutamic dehydrogenase) occurred only after 20 weeks.

The U.S. EPA (1986) reports that occupational exposure to carbon disulfide has been associated with cardiovascular, neurologic, immunologic and ocular effects. They also report that animal studies have demonstrated neurologic, cardiovascular, hepatic, renal, gastrointestinal, and hematological effects caused by inhalation of carbon disulfide.

Vigliani (1954) reported that exposure to carbon disulfide at 144-321 ppm may cause psychosis, polyneuritis (absent of weakened achilles or patellar reflexes) tremors, weakness of limbs, myopathy and vertigo in a study from the viscose rayon industry.

Teratogenic and Other Development Effects

Jones-Price et. al. (1984a,b) exposed pregnant CD rats and New Zealand White Rabbits to carbon disulfide in corn oil by gavage. CD rats were exposed to 0, 100, 200, 400, or 600 mg/kg/day for days 6-15 of gestation and killed on day 20. Rabbits were exposed to 0, 25, 75, or 150 mg/kg/day on days 6-19 of gestation and killed on day 30. Maternal toxicity effects (abnormal posture, rigidity or paralysis of hind limbs, ataxia, lethargy, rough or erect coat, decreased body weight, decreased gravid uterine weight, increased liver weight), occurred in rabbits at 75 and 150 mg/kg/day and in rats at all doses, with the most severe effects of 400 and 600 mg/kg/day. Rats exhibited no compound-related effects on number of implants/litter, proportion of litters with deal, resorbed or affected fetuses, percent resorbed, dead or affected fetuses/litter, number of live fetuses/live litter, and percent males/live litter. They did exhibit a significant dose-related decrease in average fetal body weight/litter. The rabbits exposed to 150 mg/kg/day exhibited an increase in percent resorbtions (litter and percent malformations/litter). The U.S. EPA (1986) states that a significantly increased number of resorptions/litter in rabbits at a level where no maternal toxicity occurred (25 mg/kg/day) suggests that ingested carbon disulfide may have a primary effect on the developing fetus.

Gondzik (1971) exposed "mongrel" rats to 12.5 mg/kg or 25 mg/kg distilled carbon disulfide dissolved in peanut oil intraperitoneally every second day for 60 days or 120 days. Rats exposed to 12.5 mg/kg/2 days for 60 days showed no testicular effects. Those exposed to 25 mg/kg/2 days for 60 days exhibited thickened vascular walls in the testis vessels engorged with RBCs, disorganization of the layers of seminiferous epithelium, and a reduction in the number of spermatozoa in the tubular lumen. Rats exposed to this higher dose for 120 days exhibited advanced regressive lesions involving all structural parts of the testis; including folding and shrinking of the tubular basement

membrane, scant stomal tissue, and loss of spermatogonia. Spermatogenesis was absent but Sertoli cells were present in every tubule. The number of leydig cells in the intertubular spaces was reduced and the cells showed signs of degenerative vacuolation. The authors suggest that the nature of these effects indicate irreversibility.

Mutagenic Effects

Hedenstedt et. al. (1979) found that carbon disulfide was not mutagenic in bacterial reverse mutation assays with *Salmonella typhimurium* strain TA100 with or without S-9. Donner et. al. (1981) reported similar results for strains. TA98 and TA100 and Haworth et. al. (1983) also had similar results for strains TA98, TA100, TA1535, and TA1537. Donner et. al. (1981) also reported negative results in sex-linked recessive lethal studies in *Drosophila melanogaster* and Beliles et. al. (1980) reported a lack of mutagenicity in bone marrow cytogenesis and in dominant-lethal studies in rats.

Carcinogenic Effects

Checkoway et. al. (1984) and Wilcosky et. al. (1984) reported an association between lymphocytic leukemia and multiple solvent exposure. Eleven male hourly workers from the U.S. rubber industry whose deaths were attributed to lymphocytic leukemia were compared to a control group. Of the 24 solvents, the workers were exposed to, carbon disulfide and carbon tetrachloride, had the strongest association with leukemia. There was no association between exposure to carbon disulfide and any other cancer. Wilcosky et. al. (1984) however, suggests "cautious interpretation" due to the large number of variables and that the interpretation of this apparent association between carbon disulfide and leukemia is unclear. In contradiction to these studies, Nurimen and Hernberg (1984) found no association between occupational exposure to carbon disulfide and cancer mortality in a 15-year prospective follow-up study on viscose rayon workers.

Ecotoxicity

No data regarding the toxicity of carbon disulfide to aquatic or terrestrial life, domestic or wild, were found in the literature reviewed.

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity.

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	1×10^{-1} mg/kg/day
Chronic Inhalation RfD:	2.86×10^{-3} mg/kg/day
Subchronic Oral RfD:	1.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	2.86×10^{-3} mg/kg/day
MCL:	NA
AWQC:	NA

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CHLOROBENZENE

Use

Chlorobenzene is a colorless liquid with a mild aromatic odor. It is used in the manufacture of aniline, phenol, and chloronitrobenzene as well as being produced as an intermediate in the manufacture of dye-stuffs and many pesticides (Sittig, 1991).

Chemical and Physical Properties

MF: C_6H_5Cl BP: 131-132°
MP: -45°C
MW: 112.56 FP: 28°C
Sol. (water): insoluble
Sol. (organics): soluble in alcohol, benzene, chloroform, ether

Fate and Transport

The U.S. EPA (1987) reports that chlorobenzene released to the atmosphere is expected to degrade slowly by free radical oxidation. Due to its volatility and insolubility, chlorobenzene in water is expected to partition rapidly to the air. In soils, chlorobenzene will most likely bind to soil and migrate slowly to ground water, resisting biodegradation (U.S. EPA, 1987). Chlorobenzene has been shown to bioaccumulate in fish, aquatic invertebrates, and algae, and in higher organisms it has been shown to be metabolized to other compounds (U.S. EPA, 1987).

Pharmacokinetics

The U.S. EPA (1987) reports that, based upon the high lipid solubility of chlorobenzene along with the absorption characteristics of benzene and the smaller chlorinated hydrocarbons that are also highly lipid soluble, the EPA will assume that 100 percent of an orally administered dose and 60 percent of an inhaled dose are absorbed over a period of one to several hours and that the dose is retained.

Sullivan et al. (1983) found that male Sprague-Dawley rats exposed to 100, 400, or 700 ppm ^{14}C-chlorobenzene acutely or over an 8-hour period by inhalation exhibited the highest concentration in adipose tissue. The kidney and the liver also showed significant accumulation. The data suggest a preferential distribution of chlorobenzene to the adipose tissue at all dose levels while it seems to be proportionally distributed to the kidney and liver with dose. Additionally, multiply-exposed rats (over 8 hours) tended to exhibit higher tissue burdens than rats exposed only once.

The U.S. EPA (1987) states that, upon termination of exposure, chlorobenzene would be expected to be released from the adipose tissue and become available for metabolic activation and potential continuation of induction of toxicity.

Williams et al. (1975) reported that the principal metabolites of chlorobenzene administered to mammals, including humans, are p-chlorophenol, p-chloroatechol, and p-chlorophenyl-mercapturic acid.

Spencer and Williams (1950) and Azouz et al. (1953) report that the chlorophenol metabolite is excreted as the ethereal sulfate or the gluconide. Williams et al. (1975) and Sullivan et al. (1983) report other excretion products to be chlorophenyl mercapturic acid, 4-chlorocatechol, and to a lesser degree in some species; phenol and hydroquinone.

In rats exposed to 100 ppm in air for eight hours, 5 percent was excreted via inhalation and 95 percent in the urine (Sullivan et al., 1983). At 700 ppm, 32 percent was exhaled and 68 percent excreted in the urine. The authors concluded that increasing amounts of the chemical are exhaled unchanged as the metabolic pathways for biotransformation become saturated.

Human Toxicity

Noncarcinogenic

Systemic Effects

In a study by the Monsanto Co. (1967a), male and female beagle dogs were given chlorobenzene orally by capsule at doses of 27.25, 54.5, or 272.5 mg/kg/day, 5 days/week, for 13 weeks. At 54.5 mg/kg/day slight bile duct proliferation, cytologic alternations and leukocytic infiltration of the stroma in liver occurred. The highest dose of 272.5 mg/kg/day resulted in death; body weight loss; hematologic changes; and pathologic changes in the liver which included bile duct hyperplasia, cytologic changes, leukocytic infiltration, and centrolobular degeneration; kidney changes; gastrointestinal mucosa changes; changes to hematopoietic tissue; and alterations in clinical chemistry and urine analysis.

The NTP (1985) conducted a 13-week range-finding study in groups of five male and five female rats and mice with chlorobenzene administered by gavage. Both species were administered 0, 60, 125, 250, 500 or 750 mg/kg/day, 5 days/week. 100 percent lethality occurred in male mice treated with 500 mg/kg or 750 mg/kg within one week. Death was accompanied by histopathological lesions in many organs. All female mice in the 750 mg/kg group died by week 10. At 250 mg/kg a 50 percent reduction in body weight gain and histopathological lesions were observed. Male mice at the 125 mg/kg dose exhibited increased liver weights and one male in this group and one male in the low dose group exhibited liver necrosis. In rats, the highest doses (500, 750 mg/kg) resulted in decreased body weight gain and histopathological alterations in both sexes. The females exhibited altered serum biochemistries. Decreased survival was observed in the highest dose group. AT 250 mg/kg the only observed adverse effect was minimal centrobular hepatocellular necrosis.

Most of the inhalation studies evaluated by the U.S. EPA (1987) reported no adverse effects or minimal effects.

Teratogenic and Other Developmental Effects

John et al. (1984) and Hayes et al. (1982) exposed pregnant rats and rabbits to 0, 75, 210, or 590 ppm chlorobenzene via inhalation for 6 hours/day during the period of major organogenesis (days 6-15 for rats, 6-18 for rabbits). Decreased body weight gain was observed in rats at the highest dose. No teratological effects were observed in rat fetuses at any dose level. Rabbits exhibited increased liver weights (relative and absolute) at the mid and high dose. No structural malformations were observed in rabbit fetuses either.

Mutagenic Effects

Chlorobenzene was not mutagenic for *Salmonella typhimurium* TA 98, TA100, TA1535, TA1537, TA1538; with or without addition of rat liver or hamster liver homogenate (duPont, 1977; Lawlor et al., 1979; Merck, 1978; Monsanto, 1976a; NTP, 1982; Simmon et al., 1979). Lawlor et al. (1979) and Simmons et al. (1979) report that chlorobenzene does not induce DNA damage in *E. coli* strains WP2 uvr A+rec+ or WP100 uvr A-rec- or *S. typhimurium* strains TA1978 uvr B+ or TA1538 uvr 8-. Additionally, it did not induce specific locus forward mutations in mouse lymphoma L5178Y cells, either with or without metabolic activation (Monsanto, 1976b).

Chlorobenzene did, however, cause increases in the number of revertants in *Actinomycetes antibioticus-400* (Keskinova, 1968) and *Asperigillus nidulans* (Prasad, 1970; Prasad and Promer, 1968), and mitotic disturbances in *Allium cepa* (Ostergen and Levan, 1943). The chemical also included reciprocal recombination in *Saccharomyces cerevasiae* strain D3 with metabolic activation (Simmon et al, 1979).

Carcinogenic Effects

In a gavage study of rats and mice exposed to between 30 and 120 mg/kg/day, 5 days/week for 103 weeks no statistically significant increase in the frequency of carcinomas of any type were observed (NTP, 1985).
No other data regarding the carcinogenicity of chlorobenzene were found in the literature reviewed.

Ecotoxicity

ICF (1985) reports that chlorobenzene was acutely toxic to fish at levels greater than 25 mg/liter and to aquatic invertebrates at levels greater than 10 mg/liter. ICF (1985) also stated

that chlorobenzene was shown to have a bioaccumulation factor of about 1,000 in fresh water species.

No data regarding the ecotoxicity of chlorobenzene to terrestrial life, wild or domestic, were found in the literature reviewed.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2.00×10^{-2} mg/kg/day
Chronic Inhalation RfD:	5.00×10^{-3} mg/kg/day
Subchronic Oral RfD:	2.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	5.0×10^{-2} mg/kg/day
MCL:	NA
AWQC:	Water and Fish Consumption - 488 µg/L
	Fish Consumption - 21,000 µg/L (recalculated)

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CHLOROETHANE

Use

Chloroethane, also known as ethyl chloride, is a colorless gas with a characteristic, ethereal odor. It is currently used largely as a blowing agent in foamed plastics (Lancky and Olson, 1989). ATSDR (1989) reports that, in the past, the single largest use of chloroethane was in the production of tetraethyl lead. In fact, as late as 1984, 80 percent of the chloroethane used in the U.S. was used in the production of this compound. Other uses of chloroethane include production of ethyl cellulose, dyes, chemicals, and pharmaceuticals; as a solvent, refrigerant, topical anesthetic, pulp vitality tester in dentistry, medication to alleviate pain associated with insect burns and stings, an adjunct in the treatment of tinea lesions and creeping eruptions, and as a counter-irritant for relief of myofacial and visceral pain syndromes (ATSDR, 1989).

Chemical and Physical Properties

M.F: C_2H_5Cl BP: 12.4°C
MW: 64.52 MP: -138.3°C
Sol. (Water): 5,678 mg/liter at 20°C
Sol. (Organics): miscible with principle organic solvents.

Fate and Transport

Volatilization appears to be the most likely fate of chloroethane in surface waters. Although it is highly soluble and may in fact be introduced to water from the air by washout, Gossett (1987) and Thomas (1982) found the half-life of chloroethane from a model river (1m deep, 1 m/sec flow, 3 m/sec wind speed) to be 2.4 hours.

Chemical hydrolysis to ethanol and hydrochloric acid appears to be the dominant fate of chloroethane in ground water (ATSDR, 1989), since losses from other degradation and transport processes are expected to be negligible (Vogel and McCarty, 1987).

Atmospherically, ATSDR (1989) predicts the dominant removal process for chloroethane to be reaction with photochemically generated hydroxyl radicals in the troposphere. This reaction is thought to occur via hydrogen abstraction with a half-life of 40 days (Atkinson, 1985; Howard and Evenson, 1976). Callahan et al. (1979) concluded that, based on this half-life, less than 1 percent of atmospherically-released chloroethane would diffuse into the stratosphere where it would be destroyed by photolysis. Several authors cited by ATSDR (1989) state that photolysis of chloroethane below the ozone layer will not occur because chloroethane contains no chromophores that absorb light in the visible part of the spectrum.

Volatilization is expected to be the dominant fate for chloroethane in surface soils based on its relatively high vapor pressure (1008 mm Hg) (ATSDR, 1989). Swann et al. (1983) concluded that, based on a relatively low Koc partition coefficient of 1.52, chloroethane is highly mobile in soils and may undergo significant leaching.

In subsurface soils, ATSDR (1989) reports that chemical hydrolysis may be the dominant fate of chloroethane, particularly in moist systems. Biodegradation may also occur under anaerobic conditions (Haider, 1980; Kobayashi and Rittman, 1982).

Bioconcentration of this compound in aquatic organisms does not appear to be significant (Bysshe, 1982; Hansch and Leo, 1985; Horvath, 1982).

Pharmacokinetics

Lehman and Flury (1943) report that chloroethane is expected to be rapidly absorbed through the lungs and skin in humans and animals. ATSDR (1989) reports that the rapidity of anesthia in man and animals following inhalation exposure supports Lehman and Fury's claim.

Konietzko (1984) reported that the highest concentration of chloroethane in the animal body was found in the fatty tissues around the kidney while the lowest was found in the cerebrospinal fluid. It was also reported that the brain accumulated twice the concentration of chloroethane as the blood. The chloroethane concentration was found to be especially high in the brain and medulla oblongata of the animals studied (Lehman and Flury, 1943).

Konietzko (1984) detected slight metabolism of chloroethane to ethanol via dechlorination in animals administered high anesthetic doses. Less than 0.5 percent of administered chloroethane was dechlorinated by rat liver microsomes in vitro (Van Dyke and Wineman, 1971).

ATSDR (1989) reports that excretion of chloroethane by the lungs is expected to be rapid. Morgan et al. (1970) found that humans exposed briefly by inhalation to chloroethane expired 30 percent of the retained dose within one hour. Excretion through the urine appeared to occur at a very slow rate (<0.0190/min.) one hour after inhalation (Morgan et al., 1970).

Human Toxicity

Noncarcinogenic

Systemic Effects

ATSDR (1989) reports that use of chloroethane as a general anesthetic has occasionally resulted in the death of human patients. Dawkins (1964) reports that 71 deaths between the years 1945-1964 were attributed to chloroethane. Death from respiratory paralysis (Kuschinsky, 1970) and toxic injury to the heart (Lehman and Fury, 1943) have been reported following anesthesia with chloroethane.

Sayers et al. (1929) reported mild abdominal cramps in humans who inhaled two breaths of 40,000 ppm chloroethane or three or four breaths of 20,000 ppm chloroethane. Similarly, Davidson (1925) found that an 8 minute exposure to 33,600 ppm chloroethane caused nausea and vomiting in humans. Subjects exposed to 25,000 ppm did not become nauseous, however, even after 21 minutes.

Mild eye irritation occurred when volunteers were exposed briefly to 40,000 ppm chloroethane but did not occur when they were exposed to 20,000 ppm (Sayers et al., 1929). Adams et al. (1939) reported no adverse effects in rabbits exposed to 60 doses of up to 1000 mg/kg/dose of chloroethane by gavage.

Teratogenic and Other Developmental Effects

No adverse effects were reported in female mice or their fetuses when the mice were exposed to 500 ppm chloroethane for six hours on days 6-15 of gestation (Scortichini et al., 1986).

Van Liere et al. (1966) reported reduced uterine motility and muscle tonus in dogs exposed acutely to a high concentration of chloroethane. ATSDR (1989) has questioned the relevance of this endpoint to other reproductive effects, however. Testes weight in rats was unaffected after a 10-day exposure to 438 ppm (Schmidt et al., 1972). Similarly, NTP (1988) found no adverse effects in rats and mice exposed to 19,000 ppm chloroethane for 13 weeks.

Mutagenic Effects

NTP (1988) and Riccio et al. (1983) reported positive results for mutagenicity in reverse mutation assays using *Salmonella typhimurium* with and without activation in a desiccator. However, Natarajan and Obe (1986) reported negative results for a similar test not performed in a desiccator. It has been speculated by NTP (1988) that mutagenic tests of chloroethane will only give positive results in a desiccator due to the compounds volatility.

NTP (1988) also reported negative results in a micronucleus test on bone marrow samples from mice that were exposed to 15,000 ppm chloroethane vapor for two years. Similarly, Natarajan and Obe (1986) reported negative results in a micronucleus test for chromosomal aberrations in mice.

Carcinogenic Effects

NTP (1988) found that 86 percent of female mice chronically exposed to chloroethane vapor developed highly malignant uterine carcinomas. The incidence of hepatocellular

carcinomas also increased significantly in female mice. Male mice had an increased incidence of alveolar and bronchiolar adenomas.

No data regarding the carcinogenicity of chloroethane to humans were found in the literature reviewed.

Ecotoxicity

No data regarding the ecotoxicity of chloroethane specifically were found in the literature reviewed. Data on the ecotoxicity of chlorinated ethanes in general were found, however. The available freshwater data indicate that toxicity decreases greatly with decreasing chlorination. Therefore, chloroethane is expected to be less toxic than 1,2-dichloroethane. 1,2-dichloroethane was found to be acutely toxic at concentrations as low as 118,000 µg/liter and chronically toxic at 20,000 µg/liter (U.S. EPA, 1980). Saltwater data illustrate the same correlation between toxicity and degree of chlorination. 1,2-dichloroethane is acutely toxic at concentrations as low as 113,000 µg/liter (U.S. EPA, 1980).

No data regarding the toxicity of chloroethane to terrestrial life, domestic or wild, were found in the literature reviewed. However, based on laboratory findings, some toxic effects should be expected.

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	2.9×10^{-3} mg/kg/day
Inhalation Slope Factor:	NA
Chronic Oral RfD:	4.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	2.86×10^{0} mg/kg/day
Subchronic Oral RfD:	4.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	2.86×10^{0} mg/kg/day
MCL:	NA
AWQC:	NA

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CHLOROFORM

Use

Chloroform was first used in the medical field as an anesthetic and as an inhalant for asthmatics. Because of its toxic effects, chloroform's use was eventually abandoned in medicine (Sittig, 1991). It is presently used in the production of chlorofluorocarbons, as a solvent, and in the extraction of vitamins, penicillin, and other antibiotics. Chloroform is often produced during the chlorination of public drinking water. As a result, a large percentage of the population is exposed daily to small quantities of chloroform (IARC, 1979).

Chloroform is also used as an insecticidal fumigant, in the manufacture of artificial silks and plastics, and as a residual additive in a number or drugs (IARC, 1979).

Chemical and Physical Properties

Chemical Formula: $CHCl_3$

MW:	119.38	BP:	61.7°C
SG:	1.483 at 20°C	MPP:	-63.5°C
FP:	>140°F	VP:	150.5 mmhg at 20°C

Sol. (water): 8200 mg/l at 20°C
Sol. (organics): soluble in acetone; miscible with alcohol, ether and benzene

Fate and Transport

Volatilization is chloroform's primary means of transport from surface soils and water. In groundwater and in subsurface soil, chloroform remains stable and leaches readily (EPA, 1984). In the atmosphere, chloroform is hydroxylated to form phosgene and chlorine oxide, with small quantities returning, in precipitation, as chloroform (EPA, 1984).

Pharmacokinetics

Fry, et al. (1972) and Brown, et al. (1974) report that both humans and animals absorb approximately 100 percent of ingested chloroform through the gastrointestinal lining. When inspired, up to 77 percent of chloroform is absorbed by lung tissues (EPA, 1984).

When absorbed by the body, chloroform is distributed to all organs, with high concentrations found in the nervous tissues (IARC, 1979). In rats injected with radio-isotopically labelled chloroform, 75 percent of the radioactivity was expired within 18 hours, all but 5 percent as chloroform (IARC, 1979). The majority of the retained chloroform was metabolized in the kidneys and liver. Most of the chloroform ingested by monkeys was excreted in the urine unchanged (IARC, 1979).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Chronic exposure to chloroform in humans results primarily in effects on the central nervous system and, to a lesser degree, the liver, kidneys, and heart (EPA, 1984). One study reported severe digestive disturbances, mental dullness and lassitude in workers exposed to 80 to 240 ppm (ACGIH, 1984). Symptoms appeared to be dose related in the same workers. Another reported enlarged livers in 25 percent of workers handling chloroform at a chemical plant (ACGIH, 1984).

Acute exposure to chloroform is known to be fatal in humans. Death from cardiac arrest and kidney and liver damage as a result of acute exposure (unknown concentration) in chemical plant workers has been noted (IARC, 1979).

In laboratory rats, doses between 90 and 180 mg/kg-bw/day resulted in an increased incidence of noncancerous respiratory disease. Lower doses, 60 mg/gh-bw/day, resulted in lowered body weight, decreased liver weight and decreased levels of serum cholinesterase (EPA, 1984). In another study, researchers reported necroses of the liver and gonad dysfunction at dose levels of 150 and 410 mg/kg-bw/day (EPA, 1985).

Teratogenic and Other Developmental Effects

Chloroform is known to be highly fetotoxic. Decreased body weight and an increase in fetal resorption has been noted in Sprague-Dawley rats (ACGIH, 1984). Another study reported acaudia, decreased crown-rump length, imperforated anus, missing ribs and delayed skeletal ossification in Sprague-Dawley rats that inhaled chloroform for 7 hours/day on days 6-15 of gestation at dose levels between 30 and 300 ppm.

Murray et al. (1979) reported an increased incidence of cleft palate in CF/1 mice exposed to 100 ppm for 7 hours/day on days 6-15 of gestation.

Mutagenic Effects

IRIS reports that the majority of tests for genotoxicity of chloroform have been negative. These negative results include covalent binding to DNA, mutation in *Salmonella*, a *Drosophila* sex-linked recessive test for DNA damage, a micronucleus test, and transformation of BHK cells.

IRIS states, however, that DiRenzo (1982) reported binding to radiolabeled chloroform to calf thymus DNA following metabolism by rat liver microsomes, and Callen et al. (1980) found that chloroform caused mitotic recombination in *Saccharomyces.*

Carcinogenic Effects

Although there have been no epidemiologic studies of chloroform's effects on humans, case studies have consistently reported increased levels of bladder, colon, and rectal cancer in populations exposed to relatively high chloroform levels in the drinking water (IRIS). Other suspected carcinogens were present, however in these water supplies.

Numerous animal studies have been performed that indicate chloroform to be carcinogenic. NCI (1976) reported an increase in malignant mammary tumors in female rats given 60 mg chloroform/kg-bw/day for 96 weeks in a toothpaste base. Male mice, given the same dosage, exhibited an increase in kidney tumors. A significant increase in renal tumors was observed in Osborne-Mendel rats administered chloroform in drinking water at 1800 mg/L for 104 weeks (IRIS).

In a major study performed at the Huntington Research Center, no carcinogenic effects were noted in Beagle dogs, Sprague-Dawley rats, and in 3 of 4 strains of experimental mice (ACGIH, 1984). In the fourth strain, the incidence of renal tumors increased in male ICI-Swiss mice when given 60 mg/kg/day (IARC, 1979).

Ecotoxicity

Chloroform is not known to be strongly bioaccumulated in a food chain.

Further information concerning chloroform's ecological effects was not located in the available literature.

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	6.1×10^{-3} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	8.05×10^{-2} mg/kg/day
Chronic Oral RfD:	1×10^{-2} mg/kg/day
Chronic Inhalation RfD:	Currently under review by EPA
Subchronic Oral RfD:	1×10^{-2} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.10 mg/L (total trihalomethanes)
AWQC:	Water and Fish Consumption - 0.19 µg/L
	Fish Consumption - 15.7 µg/L

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CHLOROMETHANE

Use

Chloromethane, or methyl chloride as it is often called, is a colorless, liquified gas with a faint sweet odor. It is used as a methylating and chlorinating agent in organic chemistry. Petroleum refineries use it as an extractant for greases, oils, and resins. Chloromethane is used as a solvent in the synthetic rubber industry, as a refrigerant, and as a propellant in polystyrene foam production. In the past it has been used as a local anesthetic (freezing). It is also an intermediate in drug manufacture (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: CH_3C
MW: 50.49 BP: 23.7°C
SG: 0.9159 AT 20°C MP: 97°C
Sol (water): 6,450 - 7,250 mg/liter at 20°C
Sol.(organics): Miscible with chloroform, ether, and glacial acetic acid, soluble in alcohol.

Fate and Transport

Chloromethane is a gas at typical ambient temperatures, and therefore is unlikely to remain in soil or water. A relatively low log octanol/water partition co-efficient of 0.91 suggests that partition occurs primarily into air or water with little sorption to soil or sediment. The half-life of chloromethane in agitated water was found to be 27 minutes, suggesting the atmosphere as the final fate of this halomethane.

The major process of environmental degradation of chloromethane is probably through oxidation in the troposphere. At this level in the atmosphere, the chloromethane molecule is attacked by hydroxyl radicals via the mechanism of hydrogen abstraction primarily forming formyl chloride (ICF, 1985).

Pharmacokinetics

Considerable metabolism of chloromethane occurs but the exact nature of this metabolism is uncertain. Sperling et al. (1950) found that intravenously injected chloromethane disappeared rapidly from the blood but only about 5 percent appeared in the expired air in 1 hr. and only small amounts in the bile and urine. However, Bus (1978) reported 63.9, 32.2, and 3.9 percent of the radioactivity of inhaled ^{14}C chloromethane was excreted by rats in exhaled air, urine, and feces during the first 24 hrs. Very little radioactivity remained in the body 24 hours after exposure.

Stewart et al. (1977) found that expired air falls below detectable levels within minutes after exposure to concentrations considered acceptable for industrial exposure.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Stewart et al. (1977) performed an extensive study in which human males were given single or repeated exposures to 0, 20, 100, or 150 ppm and females to 0 or 100 ppm chloromethane. Exposures were 1, 3, or 7.5 hr/day, 5 days/week. A variety of clinical tests including behavioral, neurological, electromyographic, and chemical were performed and no significant decrements were found. No increase in methyl alcohol was found in the urine and chloromethane in expired air dropped so rapidly as to be of little or no value in quantitating exposure.

Hansen et al. (1953) observed that fifteen workers exposed to excessive concentrations of chloromethane after a spill showed signs of dizziness, blurred vision, incoordination and gastrointestinal complaints. Recovery was complete in 10-30 days.

Smith and von Oettingen (1947) exposed animals to chloromethane for 6 hrs/day, 6 days/week: Guinea pigs, mice, dogs, rabbits, and rats showed injury at 1,000 ppm over varying periods up to 175 days: At 500 ppm, rats showed no effects but other animals, including dogs and monkeys showed significant responses including marked neuromuscular damage and death. At 300 ppm, no effects were observed on any animals exposed for 64 weeks.

Teratogenic and Other Developmental Effects

Chloromethane has been shown to cause reproductive effects in male rats. Morgan et al. (1982) found dose-related testicular degeneration in groups of 10 male Fisher 344 rats exposed to 1000, 3500, and 5000 ppm methylchloride vapor, 6 hr/day for 5 days, to filtered air for 2 days, and then to chloromethane for 4 more days. No testicular degeneration was observed in rats exposed to 0 ppm methylchloride.

Wolkowski-Tyl et al. (1983) exposed groups of 74-77 female 57B46 mice that were mated to C3H male mice to 0,250, 500 or 750 ppm concentrations of chloromethane for 6 hrs/day during days 6-18 of gestation. Dams were killed on the last day. Those exposed to 750 ppm had decreased body weights, tremors, convulsions and ataxia, and were hypersensitive to touch and sound. 6 mice died and one was killed in extremis in the 750 ppm group during the exposure period. The fetuses of the 500 ppm and 750 ppm groups had significantly increased incidences of heart defects. No chloromethane-induced effects were reported in the 250 ppm group.

In a 2-generation reproductive study, Hamm et al. (1985) exposed Fischer rats to chloromethane vapor. Groups of 40 males and 80 females were exposed to 0, 150, 475, and 1500 ppm concentrations of chloromethane for 6 hrs/day, 5 days/week for 10 weeks. After this, each male was mated to 2 exposed females and the exposure was changed to 6 hrs/day for 7 days/week. After a 2-week mating period males were removed from exposure and mated with unexposed females for an additional 2 weeks. Females in treated groups were not exposed from gestation day 18 to postnatal day 4. Members of the F1 generation were exposed just as their parents had been (0,150, and 475 ppm chloromethane for 10 weeks) and were then mated.

Although no effects on litter size, sex ratio, pup viability, pup survival or pup growth were noted in the 150 and 475 ppm groups, no litters were produced from males exposed to 1500 ppm when mated with either exposed or unexposed females. Exposed and unexposed females produced significantly fewer litters when mated with 475 ppm males as well. A trend toward decreased fertility at 475 ppm was the only effect detected in the breedings.

Mutagenic Effects

Andrews et al. (1976) performed mutagenic studies in *Salmonella typhimurium* TA 1535 with an without microsomal enzyme activation. The results of these studies indicate that chloromethane induces reversions.

Fostel et al. (1985) found that chloromethane was also positive for forward mutations in *S. typhimurium* T677 and human lymphoblasts and for SCE in human lymphoblasts without Metabolic activation. Working et al. (1985) reported that chloromethane induced dominant lethal mutations in mature sperm of Fischer 344 rats exposed to 3000 ppm, 6 hrs/day for 5 days.

Carcinogenic Effects

CIIT (1985) exposed group of 120 male and 120 female B6C3F1 Mice and equal numbers of male and female Fischer rats to 0, 50, 225, or 100 ppm concentrations of chloromethane Vapor for 6 yrs/day, 5 days/week for 104 weeks (2 yrs.).

5-20 rats and mice of each sex from each group were killed every six months except for mice in the 1000 ppm group which suffered high mortality. (only 2 survived to 2 months at which time they were sacrificed). A significant increase in the incidence of renal tumors was observed in male mice. The first tumor was detected at 12 months and following that the type of tumors detected included renal cortical adenomas, renal cortical adenocarcinomas, papillary cystadenomas, tubular cystadermas and papillary cystaderocarcinomas. The incidences of kidney tumors were dose-related and occurred in the 115 and 1000 ppm groups. No treatment-related development of oncogenicity was detected in female mice or male or female rats.

Ecotoxicity

The available data for halomethanes in general indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 11,000 ug/L and would be expected to occur at lower concentrations in species that are more sensitive than those studied. (AWQC, 1986). Data on the chlorinated methanes other than chloromethane indicate that aquatic toxicity declines with decreased chlorination. Thus, chloromethane should be less toxic than chloroform or carbon tetrachloride, neither of which had any effect on Daphnic Magna or the fathead minnow, respectively, during chronic exposure to 3400 ug/liter (ICF, 1985).

The available data for halomethanes in saltwater environments indicate that acute and chronic toxicity occurs at concentrations as low as 12,000 and 6,400 ug/liter, respectively, and would likely occur at lower concentration among species more sensitive than the tested (AWQC, 1986). A decrease in algal cell numbers occurs at concentrations as low as 11,500 ug (AWQC, 1986).

No information as to the toxicity of chloromethane to terrestrial animals, wild or domestic, was found in the literature reviewed.

Standards, Criteria and Guidelines

EPA Class C Carcinogen

Oral Slope Factor:	1.3×10^{-2} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	6.3×10^{-3} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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DIBROMOCHLOROMETHANE

Use

Dibromochloromethane, a clear colorless liquid, is used as an intermediate in the manufacture of fire extinguishing agents, aerosol propellants, refrigerants, and pesticides (Sittig, 1991).

Chemical and Physical Properties

MF: $CHBr_2Cl$ BP: 119-120°C
MW: 208.29
Sol. (Water): 1050 ppm at 30°C
Sol. (Organics): miscible or soluble in alcohols, ethers, and benzene.

Fate and Transport

Mabey and Mill (1978) reported a half-life of 274 years for dibromochloromethane in water at pH 7 and 25°C. They also reported a first-order hydrolysis rate constant of 0.8×10^{-10}/sec indicating that hydrolysis is not a significant fate for this compound. Photolytically, Ogita et al. (1983) reported that dibromochloromethane in aqueous solution at room temperature is decomposed by UV irradiation $\geq$ 200 nm. Dibromochloromethane is not easily oxidized in aquatic systems because there are no functional groups that react strongly with hydroxyl radicals (Callahan et al., 1979). Studies have revealed that dibromochloromethane can be biodegraded under anaerobic or methanogenic conditions but not under aerobic ones (U.S. EPA, 1985). Kaczmar et al. (1984) found volatilization to be an important mechanism for the removal of dibromochloromethane from the aquatic environment.

It is thought that dibromochloromethane is often formed during the chlorination of drinking water (U.S. EPA, 1985).

The atmospheric chemical removal processes include reaction with different free radicals ($OH\cdot$, $O\cdot$, $Cl\cdot$, etc.), with ozone, or photolytic transformations (U.S. EPA, 1985). The U.S. EPA (1985) states that atmospheric residence times for bromochloromethanes are less than two years. Physical removal processes, such as dissolution, adsorption on aerosol particles, and dry deposition, are not likely to be significant fates for atmospheric dibromochloromethane (U.S. EPA, 1985).

Calculations by Kenaga and Goring (1978) indicate that dibromochloromethane has medium-to-high soil mobility. Therefore, leaching of dibromochloromethane into ground water may occur. Wilson, et al. (1981) has shown volatilization to be an important fate of bromochloromethanes in soil. The U.S. EPA (1985) states that microbial degradation of dibromochloromethane in soil may also be an important mechanism of removal.

Pharmacokinetics

The U.S. EPA (1985) reports that toxicity studies indicate that dibromochloromethane is absorbed following ingestion.

Pfaffenberger et al. (1979) found that bromodichloromethane administered by gavage became slightly concentrated in the serum and adipose tissue of rates, but had virtually disappeared from these tissues within three to six days.

Anders et al. (1978) reported carbon monoxide in the blood of rats after they were intraperitoneally injected with dibromochloromethane at above average concentrations. This suggests carbon monoxide as one of the products of the metabolism of dibromochloromethane.

Human Toxicity

Noncarcinogenic

Systemic Effects

NTP (1985) performed both short-term and long-term studies on the effects of dibromochloromethane in rats and mice. In the short-term study, groups of 10 F344/N rats of each sex and 10 B6C3F1 mice of each sex were administered 0, 15, 30, 60, 125, or 250 mg/kg/day of dibromochloromethane by gavage for five days/week for 13 weeks. Male mice exhibited increased incidence of vacuolar change (fatty metamorphosis) in the liver and toxic nephrosis at 250 mg/kg/day. Rats of both sexes showed increased incidences of liver vacuolar change, centrilobular necrosis, toxic neuropathy, and salivary gland inflammation and squamous metaplasia at 250 mg/kg/day. Male rats at the lower dose levels also exhibited vacuolar changes.

In the long-term study, 50 F344/N rats of each sex and 50 B6C3F1 mice of each sex were exposed to 0, 40, and 80 mg/kg/day of dibromochloromethane by gavage five days/week for 104 and 105 weeks, respectively. An increase in liver fatty changes and ground glass cytoplasmic changes was found to be dose-related in rats of both sexes. Treated female rats also had higher incidences of kidney nephrosis. Higher incidences of hepatomegaly, fatty metamorphosis, calcification, and liver necrosis occurred in treated mice of both sexes. Male mice also exhibited increased incidence of nephrosis and the females exhibited incidence of thyroid follicular cell hyperplasia.

Teratogenic and Other Developmental Effects

Ruddick et al. (1983) exposed groups of 9-14 Sprague-Dawley rats to 0, 50, 100, or 200 mg/kg/day of either dibromochloromethane or

bromodichloromethane on days 6-15 of gestation. The only adverse effects exhibited by the dams was increased liver and kidney weights and decreased body weights at the higher dose levels. The fetuses exhibited no significant adverse effects.

Groups of 10 male and 30 female IRC mice as well as subsequent generations were exposed to dibromochloromethane in emulphor (a polyoxyethylated nonionic surfactant oil) at 0, 0.1, 1.0, and 4.0 mg/ml concentrations in drinking water (Borzelleca and Carchman, 1982). Survival of the F1 generation was reduced in mice treated with 4 mg/ml. Additionally litter size, gestation index, and the viability of the pups was also significantly reduced. Lactation index and postnatal body weight were reduced in the F2b generation after exposure to 4 mg/ml dibromochloromethane. No significant increase in malformations was reported at any dose level. At the 1 mg/ml concentration, significant decreases in litter size, pup viability, postnatal body weight and lactation index occurred.

Mutagenic Effects

Simmon (1978) reported that dibromochloromethane gave a positive dose-related response for reverse mutation without metabolic activation in *Salmonella typhimurium* TA100. Borzelleca and Carchman (1982) found that dibromochloromethane was negative in a dominant lethal study conducted on IRC mice. Dibromochloromethane treatment produced dose-related increased incidences of sister chromatid exchange in mouse bone marrow cells following oral treatment of male mice with 0, 25, 50, of 100 mg/kg/day for four days and in human lymphocytes in vitro (Morrimoto and Koizumi, 1983).

Carcinogenic Effects

Data regarding the carcinogenicity of dibromochloromethane specifically to humans were not found in the literature reviewed. However, Aldrich and Peoples' (1982) survey of melanoma cases in Brevard County, Florida did reveal a relationship between observed new cases of melanoma and levels of the trihalomethanes, bromodichloromethane and dibromochloromethane, in drinking water. Although factors such as the age of the population surveyed, sunlight, frequency of outdoor activity and presence of other contaminants in the drinking water (chloroform) may be playing a role, the data do suggest a relationship between increased cancer risk and trihalomethanes.

In the aforementioned NTP (1985) long-term study, the rats exhibited no signs of carcinogenicity. Mice exhibited increased incidences of hepatocellular adenomas and carcinomas in the high dose group (100 mg/kg/day) although it was only statistically significant in the females.

Voronin et al. (1987) observed no significant tumor increases in groups of 50 CBAX C57B1/6 mice/sex treated with 0, 0.4, 4.0, or 400 mg/liter dibromochloromethane in drinking water.

Ecotoxicity

No data regarding the ecotoxicity of dibromochloromethane were found in the literature reviewed.

Standards, Criteria and Guidelines

EPA Class C Carcinogen

Oral Slope Factor:	8.4×10^{-2} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2×10^{-2} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	0.10 mg/l
AWQC:	NA

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DICHLOROBENZENE

Use

The compound dichlorobenzene (DCB) has three geometric isomers. 1,2-DCB is a pale yellow liquid with a pleasant, aromatic odor. Its major uses are as a process solvent in the manufacturing of toluene diisocyanate and as an intermediate in the synthesis of dyestuffs, herbicides, and degreasers. Information is not available regarding the use of 1,3-DCB. It may occur as a contaminant of 1,2-DCB and 1,4-DCB. 1,4-DCB is a colorless solid with a mothball-like odor. It is used primarily as an air deodorant and an insecticide, which account for 90 percent of the total production of this isomer (Sittig, 1991).

Physical and Chemical Properties

Chemical Formula: $C_6H_4Cl_2$

	BP: 180.5°C
MW: 147.0 25°C	FP: -17.03°C
Sol. (Water): 154 mg/l at 25°C	VP: 1.5 mm Hg at 20°C

Sol. (organics): Miscible with alcohol, ether, and benzene

Fate and Transport

Dichlorobenzenes exist primarily in a vapor phase in the atmosphere and react with photochemically produced hydroxyl radicals (EPA, 1987). Pankow et al. (1984), detecting dichlorobenzenes in rain water, suggested washout as a possible means of atmospheric removal.

Several studies in the Great Lakes area (Oliver and Nicol, 1982; Oliver, 1983; Oliver and Charlton, 1984) have revealed adsorption to sediments as a major fate process for dichlorobenzenes. Oliver and Nicol (1982) found dichlorobenzenes to be persistently present in Lake Ontario sediment cores. Although Lyman et al. (1982) illustrated the potential of dichlorobenzenes to volatilize from the water column, the U.S. EPA (1987) reports that adsorption to sediments will greatly reduce this fate process.

Swann et al. (1984) reports that, based on the range of log octanol/water partition coefficients (3.38 - 3.60), dichlorobenzenes can be expected to be adsorbed moderately to tightly in soils. The ability of dichlorobenzenes to leach from soils into ground water was illustrated by Page (1981) and Hutchins et al. (1983). The U.S. EPA (1987) reports that volatilization may occur from surface soil but suggests that this is a less important fate process. Haider et al. (1974) reports that, under aerobic conditions, slow biodegradation in soil may take place.

Pharmacokinetics

Ware and West (1977) and U.S. EPA (1985) have concluded that all three dichlorobenzene isomers (1,2-, 1,3-, and 1,4-) are absorbed through the gastrointestinal tract of humans and experimental animals. Studies on two female CFY rats (Hawkins et al., 1980) have revealed the gastrointestinal absorption of 1,4-DCB to be greater than 90 percent.

Ware and West (1977) and U.S. EPA (1985) also concluded that all three dichlorobenzene isomers are absorbed by the lungs of humans and experimental animals. Hawkins et al. (1980) concluded that 1,4-DCB is substantially absorbed from the lungs based on inhalation studies with CFY rats.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

In an extensive study by NTP (1985), various groups of F344/N rats and B6C3F1 mice were exposed to 1,2-DCB in corn oil by gavage at various concentrations for various durations. Fifty animals of each sex and species were exposed to 0, 60, or 120 mg/kg/day, 5 days/week for 103 weeks. No significant adverse effects occurred. Ten animals of each sex and species exposed to 0, 30, 60, 125, 250, or 500 mg/kg/day, 5 days/week, for 13 weeks exhibited liver necrosis at the 250 mg/kg/day dose level. Male rats exhibited death, degeneration and necrosis of the liver, lymphocyte depletion in the spleen and thymus, and renal tubular degeneration at the 500 mg/kg/day dose level. Slight decreases in hemoglobin, hematocrit, and red blood cell counts occurred in rats exposed to this high dose.

Rats dosed by gavage with 1,2-DCB at 18.8, 188, or 376 mg/kg/day, 5 days/week for 192 days exhibited increased liver and kidney weights at 188 mg/kg/day and liver pathology and increased spleen weight at 376 mg/kg/day. No effects were observed at the low dose (Hollingsworth et al., 1958). Hollingsworth et al. (1958) also exposed rats, guinea pigs, mice, rats, and monkeys to 1,2-DCB by inhalation at concentrations of 49 or 93 ppm for 7 hours/day, 5 days/week for 6 to 7 months. The only adverse effects reported were reduced body weight gain in rats and reduced spleen weight in guinea pigs at the high dose.

Data regarding the noncarcinogenic systemic effects of 1,3-DCB were not available in the literature reviewed.

In a study by NTP (1986) 50 B6C3F1 mice of each sex and 50 F344 rats of each sex were exposed to 0, 150 (rats only), 300, or 600 (mice only) mg/kg/day of 1,4-DCB 5 days/week for 103 weeks. High-dose male rats exhibited a significantly increased mortality rate relative to controls. High-dose female rats had a 5-7 percent lower mean body weight relative to controls. All the dosed animals except for the male rats exhibited an increased incidence of nonneoplastic nephropathy, characterized by renal tubular atrophy, degeneration and regeneration; tubular dilation; thickening of the basement membrane; minimal accumulation of interstitial collagen; and the presence of granular casts.

Loeser and Litchfield (1983) exposed groups of 76-79 male and 76-79 female Alderly Park Wistar-derived rats to 0, 75, or 500 ppm of 1,4-DCB, 5 hours/day, 5 days/week for 76 weeks. No immediate effects on body weight, food or water consumption, mortality rate, blood biochemistry, hematology, or the histology of major organs were observed. Thirty-six weeks following treatment, however, those rats exposed to the 500 ppm dose level exhibited increased liver, lung, kidney, and heart weights and a "slightly" elevated urinary coproporphyrin levels.

Teratogenic and Other Developmental Effects

Hayes (1985) exposed pregnant F344/N rats and New Zealand rabbits by inhalation to 0, 100, 200, or 400 ppm of 1,2-DCB, 6 hours daily on days 6 to 15 (rats) or 6 to 18 (rabbits) of gestation. Body weight gain was lower in rats at all doses and in rabbits at 400 ppm during the first 3 days of exposure. Liver weights (absolute and relative) were increased in rats at 400 ppm. No developmental toxicity was observed at any dose.

Data regarding the teratogenicity of 1,3-DCB were not available in the literature reviewed.

Loeser and Litchfield (1983) summarized a report by Hodge et al. (1977) in which groups of 20 pregnant SPF rats were exposed to 0, 75, 200, or 500 ppm of 1,4-DCB for 6 hours/day on days 6-15 of gestation. No evidence of maternal toxicity was observed. One fetus in each group was reported to have a malformation. No significant teratogenic, embryo, or fetotoxic effects were associated with inhalation exposure to 1,4-DCB. Hayes et al. (1985) found no evidence of teratogenicity, embryotoxicity, or fetotoxicity in a similar study with New Zealand rabbits.

Mutagenic Effects

1,2-DCB was not mutagenic in *S. typhimurium* strains TA98, TA100, TA1535, or TA1537 with and without rat or hamster liver homogenates at concentrations as high as 333 µg/plate (NTP, 1985). Lawlor et al. (1979) reported that 1,2-DCB did not produce reverse mutations in TA98, TA100, TA1535, TA1537, or TA1538 with and without rat liver homogenates. Prasad and Promer (1968), however, did report an increase in the frequency of mutations in the auxotrophic strain of *Aspergillus nidulans* following exposure to 1,2-DCB. Similarly, Zapata-Gayan et al. (1982) reported a statistically significant increase in chromosomal alterations in workers occupationally exposed to 1,2-DCB vapors for 4 days (8 hours/day).

Prasad and Promer (1968) report that an increase in the number of revertants of an auxotrophic strain of *Aspergillus nidulans* occurred when spores were treated with 1,3-DCB. However, the increase, however, was not statistically significant.

1,4-DCB was not mutagenic in *S. typhimurium* strains TA98, TA100, TA1535, TA1537, and TA1538 with or without rat liver microsomal activation (Lawlor et al., 1979; NTP, 1986). NTP (1986) found that 1,4-DCB did not increase the mutation frequency in cultivated L5178Y/TI$^{+/-}$ mouse lymphoma cells with or without metabolic activation, nor did it increase the frequency of sister-chromatid exchange or chromosomal aberrations in Chinese Hamster ovaries. Several studies reviewed by Loeser and Litchfield (1983) revealed no signs of mutagenicity as well.

Carcinogenic Effects

The NTP (1985) exposed groups of 50 F344N rats/sex and 50 B6C3F1 mice/sex to 0, 60, or 120 mg/kg/day of 1,2-DCB by gavage in corn oil, 5 days/week for 103 weeks. No statistically significant increases in tumors was observed in the rats. Mice exhibited an increase in the incidence of malignant histiocytic lymphoma. Male mice had an increased incidence of alveolar and bronchiolar carcinomas. One high-dose male had a testicular interstitial cell tumor. Contrary to these findings, male mice exhibited a decrease in the incidence of hepatocellular adenomas and carcinomas.

Data regarding the carcinogenicity of 1,3-DCB were not available in the literature reviewed.

Fifty male and 50 female B6C3F1 mice and 50 female F344/N rats were exposed to 0, 300, or 600 mg/kg/day of 1,4-DCB in corn oil by gavage for 5 days/week for 103 weeks in an NTP (1986) bioassay. Fifty male F344/N rates were exposed to 0, 150, or 300 mg/kg/day for the same duration. Mice exhibited a significant increase in the incidence

of hepatocellular adenomas and/or carcinomas. Male rats exhibited a significant increase in the incidence of renal tubular adenocarcinoma and adenoma.

No treatment-related increase in the incidence of tumors was observed in groups of 75-79 Alderly Park Swiss mice or Wistar-derived rats exposed to 0, 75, or 500 ppm 1,4-DCB for 5 hours/day, 5 days/week for 57 and 76 weeks, respectively (Loeser and Litchfield, 1983).

Ecotoxicity

ICF (1985) reports that all the 48- and 96-hour LC_{50} values for *Daphnia* and bluegills tested under static conditions are; 2,440 and 5,590 µg/l (1,2-DCB); 28,100 and 5,020 µg/l (1,3-DCB); and 11,000 and 4,280 µg/l (1,4-DCB), respectively. The 96-hour LC_{50} values around 3,000 µg/l were obtained in two flow through tests using fathead minnows and rainbow trout. The fathead minnow has a chronic toxicity value of 2,000 µg/l. A whole body bioconcentration factor of approximately 80 was reported in the bluegill. In the freshwater alga *Selenastrum capricornutum*, the 96-hour median effect levels for chlorophyll *a* and cell numbers are 179,000 and 149,000 µg/l, respectively.

In saltwater systems, acute values for the mysid shrimp and sheepshead minnow were 1970 and 9,660 µg/l, respectively. No saltwater chronic values are available. The 96-hour median effect levels for chlorophyll *a* and cell numbers in the saltwater alga *Skeletonema capricornutum* are 44,200 and 44,100 µg/l, respectively.

Standards, Criteria, and Guidelines

1,2-DCB EPA Class D Carcinogen
1,3-DCB EPA Class D Carcinogen
1,4-DCB EPA Class C Carcinogen

Oral Slope Factor:	1,2-DCB:	NA
	1,3-DCB:	NA
	1,4-DCB:	2.4×10^{-2} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	1,2-DCB:	NA
	1,3-DCB:	NA
	1,4-DCB:	NA
Chronic Oral RFD:	1,2-DCB:	9×10^{-2} mg/kg/day
	1,3-DCB:	NA
	1,4-DCB:	NA

Chronic Inhalation RfD:	1,2-DCB:	4×10^{-1} mg/kg/day
	1,3-DCB:	NA
	1,4-DCB:	7×10^{-1} mg/m^3
Subchronic Oral RfD:	1,2-DCB:	9×10^{-1} m/kg/day
	1,3-DCB:	NA
	1,4-DCB:	NA
MCL:	1,2-DCB:	600 µg/l
	1,3-DCB:	600 µg/l
	1,4-DCB:	75 µg/l
AWQC:	Water and Fish Consumption: 0.4 mg/l	
	Fish Consumption: 2.6 mg/l	

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1,1-DICHLOROETHANE

Use

1,1-dichloroethane, also known as asymmetrical dichloroethane, ethylidene chloride and 1,1-ethylidene chloride; is used as a solvent and cleaning and degreasing agent. It is also an intermediate in organic synthesis. (Sittig, 1991)

Physical and Chemical Properties

Chemical Formula: CH_2 Cl CH_2 Cl

MW: 98.96	BP: 83-84°C
SG: 1.253 at 20°C	MP: -35.4°C
Sol. (water): 8 g/liter	VP: 61 mmHg at 20°C

Sol. (organics): miscible with alcohol, chloroform, and ether

Fate and Transport

Volatilization is the most significant means of escape of 1,1-dichloroethane from surface waters (ICF, 1985). The chemical is rapidly broken down by hydroxylation in the atmosphere. Some may be absorbed by atmospheric water and return to the earth by precipitation, however (ICF, 1985). Due to a low octanol/water partition coefficient (1.48) and reasonable solubility in water, leaching through soil into the ground water is an expected fate (ICF, 1985).

Pharmacokinetics

Specific data regarding the uptake and metabolism of 1,1-dichloroethane was not found in the literature reviewed. Data on the uptake of its isomer, 1,2-dichloroethane, was found, however. A similar uptake of the two isomers may be suspected.

Reitz et al. (1982) administered 150 mg ^{14}C-1,2-dichloroethane/kg bw in corn oil to rats. Recovery of radioactivity in exhaled air, urine and carcass at the end of 48 hours was virtually complete. Spreafico et al. (1978, 1979, 1980) found that peak blood levels occurred within 20 minutes and appeared to be linearly related to dose level when rats were exposed to 25, 50, or 150 mg 1,2-dichloroethane/kg bw in corn oil by gavage. Tissue levels, however were not linearly related. The authors concluded from this that passive transport across the GI tract occurred.

Urusova (1953) reported that women exposed to $\simeq$ 15.5 ppm 1,2-dichloroethane in air during a normal work day accumulated the chemical in breast milk. Immediately following exposure, exhaled air contained 14.5 ppm 1,2-dichloroethane indicating absorption through their lungs and the achievement of blood and total body equilibrium with inspired air within

the daily work period. Reitz et al. (1980, 1982) found that equilibrium was reached in approximately 1 hour and was maintained at ~ 9 mg/liter when 4 Osborne-Mendel rats were exposed to 150 ppm 1,2-dichloroethane for 6 hours. Blood levels approached zero 1.5 hours after exposure was terminated.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Smyth (1956) reported that rats survived an 8-hour exposure to 4000 ppm but were killed by 16000 ppm. Lehmann and Plaa et al. (1965) reported no renal necrosis in mice exposed to 1000 mg/kg dose. Some tubular swelling occurred at this dose and at higher doses urinary protein (2000 mg/kg) and urinary glucose (4000 mg/kg) increased. Hoffman et al. (1971) exposed rats, guinea pigs, rabbits, and cats to 500 ppm concentration of 1,1-dichloroethane to 6 hours/day, 5 days/week for 13 weeks. No adverse effects were observed. After an additional 13 weeks however, the cats exhibited evidence of kidney injury histologically and by increased blood urea.

Teratogenic and Other Development Effects

Schwetz et al. (1974) exposed pregnant female rats on days 6-15 of gestation to 3800-6000 ppm 1,1-dichloroethane vapors for 7 hour/day. No adverse effects were observed in the dams or the fetuses except for slight, although statistically significant, decreases in food consumption and weight gain by the dams and delayed ossification in the fetuses. No teratological effects were related to exposures.

Mutagenic Effects

Riccio et al. (1983) and Mitoma et al. (1984) reported positive results in *S. typhimurium* strains TA1535, TA98, and TA100 when tested by plate incorporation in a desiccator in the presence and absence of metabolic activation systems. Strain TA1537, however, yielded negative results in this test. Similarly, Tu et al. (1985) and A.D. Little, Inc. (1983) reported negative results for 1,1-dichloroethane in a cell transformation assay with BALB/C-3T3 cells tested in the absence of an exogenous metabolic activation system in a sealed glass incubation chamber. When tested in a similar manner, however, 1,1-dichloroethane produced positive results in a DNA repair assay with hepatocyte primary cultures from rats or mice (Williams, 1977).

Carcinogenic Effects

Limited evidence of carcinogenicity was revealed in an NCI (1978a) bioassay. In this study Osborne-Mendel rats and B6C3F1 mice were administered technical grade 1,1-dichloroethane in corn oil by gavage, 5 days/week for 78 and 70 weeks, respectively. The dosing was incontinuous (3 weeks on, 1 week off) due to toxicity resulting in doses of 382 (low) and 764 (high) mg/kg/day for male rats, 475 (low) and 950 (high) for female rats, 1442 (low) and 2885 (high) mg/kg/day for male mice, and 1665 (low) and 3331 (high) mg/kg/day for female mice. Unexposed and vehicle-control groups were monitored as well. Female rats exhibited a statistically significant dose-related increase in the incidence of hemangiosarcomas and those females surviving 52 weeks showed a significant increase in the incidence of mammary gland adenocarcinomas. Male rats exhibited no significant incidences of carcinomas. Female mice exhibited liver carcinomas in the vehicle-control and low-dose groups, an increase in benign uterine endometrial stromal polyps in the high-dose group. A dose-related increase in the incidence of hepatocellular carcinomas was observed in male mice.

In another NCI (1978b) study, the isomer of 1,1-dichloroethane, 1,2-dichloroethane, produced an increase in the incidence of forestomach squamos cell carcinomas and hemangiosarcomas in male rats and an increase in the incidence of mammary adenocarcinomas in female rats and mice. Additionally, mice of both sexes exhibited alveolar and bronchiolar adenomas, females exhibited endometrial stromal polyps and sarcomas, and males exhibited hepatocellular carcinomas.

Klaunig et al. (1986) concluded that 1,1-dichloroethane was not carcinogenic based on a 56-week study in B6C3F1 mice. The mice were exposed to 0, 835, or 2500 mg/liter in drinking water following a 4-week exposure to 10 mg/liter diethyl nitrosamine (DENA-initiated groups) or deionized water (uninitiated groups). Mice were sacrificed at 24 and 52 weeks and no adverse effects were observed. IRIS, however, has questioned the adequacy of the duration of the study.

Chlorinated ethanes and ethylenes were investigated by Milman et al. (1988) and Story et al. (1986) to detect their potential tumor initiating or promoting effects in a liver foci assay in Osborne-Mendel rats. 1,1-dichloroethane did not show any signs of initiation or complete carcinogenicity in the absence of initiation or promotion. It did exhibit promotional effects with DENA as initiator. However, the assumption that the liver foci seen in this assay are precancerous has not been validated (IRIS).

Ecotoxicity

The available freshwater data for chlorinated ethanes indicate that toxicity increases greatly with increasing chlorination (AWQC, 1986). ICF (1985) reports that 1,1,1-trichloroethane is less active than the 1,1,2-isomer. Based on this, 1,1-dichloroethane is probably no more toxic

than the 1,2-isomer. 1,2-dichloroethane is acutely toxic at 100-500 mg/liter concentrations and chronically toxic at 20 mg/liter concentration (ICF, 1985). In saltwater systems, 1,2-dichloroethane was acutely toxic at 113 mg/liter (AWQC, 1986).

No data regarding the toxicity of 1,1-dichloroethane to terrestrial life, wild or domestic, was found in the literature reviewed.

Standards, Criteria, and Guidelines

EPA Class C Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	1.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	1.0×10^{-1} mg/kg/day
Subchronic Oral RfD:	1.0×10^{0} mg/kg/day
Subchronic Inhalation RfD:	1.0×10^{0} mg/kg/day
MCL:	NA
AWQC:	NA

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1,2 DICHLOROETHANE

Use

1,2 dichloroethane is a thick, man-made liquid that is volatile and possesses a sweet odor. It is used as a precursor in the production of vinyl chloride and numerous industrial degreasers. It can be found in cleaning agents and pesticides and also in such household items as adhesives and paint removers (ATSDR, 1989). 1,2 dichloroethane is also used as a gasoline additive.

Chemical and Physical Properties

Chemical formula: $C_2H_4Cl_2$

MW:	98.97	BP:	83.5°C
SG:	1.253 at 20°C	MP:	-35.4°C
FP:	15°C	VP:	63.8 mmhg at 20°C

Sol.(water): 8690 mg/l at 20°C
Sol.(organics): miscible with alcohol, chloroform and ether

Fate and Transport

The primary avenue of exposure to 1,2 dichloroethane is through the air. The majority of releases of 1,2 dichloroethane occur from accidental spills or improper disposal techniques. When released to surface waters, 1,2 dichloroethane volatilizes to the atmosphere within three days. In the atmosphere, it degrades, through hydroxylation, within two to three months. Because it does not sorb well into soils, 1,2 dichloroethane is readily transported to ground water where it may remain for years (EPA, 1985).

Pharmacokinetics

1,2 dichloroethane is readily absorbed through the tissues of the lungs following inhalation and is respired rapidly (ATSDR, 1989). In a separate study, it was reported that nursing women exposed to 0.063 ppm 1,2 dichloroethane in the workplace rapidly accumulated it in breast milk. Maximum concentrations were reached within one hour after work ended (ATSDR, 1989).

1,2 dichloroethane is known to absorb, via passive diffusion across the mucous membranes of the gastrointestinal tract. Reports indicate that peak concentrations of 1,2 dichloroethane in rats were reached 15 minutes after oral exposure (ATSDR, 1989). Because of it's lipophilic qualities, 1,2 dichloroethane is expected to accumulate in the body's fatty tissues.

1,2 dichloroethane is expelled from the body in the urine after being metabolized to non-volatile organic compounds. It is thought that when this metabolic process is exhausted

through acute exposure to 1,2 dichloroethane, the compound recirculates through the body and causes increased toxic effects.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Chronic inhalation exposure to humans is known to cause anorexia, nausea, vomiting, fatigue and nervousness (EPA, 1984). Suveev and Babichenko (1969) reported cold sweats, brachycardia, systolic murmurs and enlarged livers in a high percentage of 12 workers chronically exposed to 1,2 dichloroethane. Numerous studies have reported signs of central nervous system dysfunction amongst occupationally exposed workers.

Kozik (1957) reported increases in gastrointestinal disorders, liver and gall bladder diseases and diseases of the muscle, tendons and neuronal ganglia amongst workers exposed to aircraft glue fumes containing 1,2 dichloroethane.

In a chronic oral exposure study performed by NCI (1978), Osborne-Mendel rats were exposed, through gavage doses, to 47 and 95 mg/kg-bw/day for 78 weeks. A high mortality rate was noted for rats in the high exposure group as early as 15 weeks into the study. The early deaths were attributed to unspecified toxic effects rather than carcinogenic effects. Seventy-two percent of female rats exposed to the high dose died as a result of tumors between 60 and 80 weeks.

Teratogenic and Other Developmental Effects

No teratogenic effects were reported in a study performed by Alumot et al. (1976). Rats were fed diets containing 250 or 500 ppm 1,2 dichloroethane. Litter size, mortality rate and weight of young were not effected. 1,2 dichloroethane is known to accumulate in human and animal fetuses (ATSDR, 1989).

Mutagenic Effects

1,2 dichloroethane was shown to be mildly mutagenic in *Salmonella typhimurium* and in *E. coli*. It also increases the amount of sex-linked recessive-lethal alleles in *Drosophila melanogaster* (EPA, 1985).

Carcinogenic Effects

A statistically significant increase in incidences of colon and rectal cancer in men of age 55 years or older was noted by Isacson (1985) as a result of 1,2 dichloroethane in drinking water. These data may be impertinent because of possible exposure to other contaminants (ATSDR, 1989). No other human carcinogenicity data was found in the available literature.

Numerous animal studies, however, indicate 1,2 dichloroethane to be a carcinogen. Doses as low as 47 mg/kg-bw/day produced tumors in rats and mice when administered by gavage (NCI, 1978). In this study, increases in forestomach squamous cell carcinomas and circulating system hemangiosarcomas were noted. Alveolar adenomas, hepatocellular carcinomas and endometrial sarcomas were all noted in mice of both sexes. Mammary adenocarcinomas were observed in female mice and rats (IRIS, 1990).

Inhalation exposure of Wistar and Sprague-Dawley rats and Swiss mice to 1,2 dichloroethane did not result in an increase in tumor incidence, although severe toxic effects were noted in Sprague-Dawley rats exposed to 250 ppm for 7 hours/day, 5 days/week for 78 weeks by Maltoni et al. (1980).

Ecotoxicity

1,2 dichloroethane is known to be less toxic to aquatic animals than most chlorinated ethanes. No information regarding effects to plants or terrestrial animals was found in the available material.

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	9.1×10^{-2} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	9.1×10^{-2} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	5 µg/L
AWQC:	Water and Fish Consumption - 0.94 µg/L
	Fish Consumption - 243.0 µg/L

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1,1 DICHLOROETHYLENE

Use

1,1 Dichloroethylene (1,1 DCE) is a clear liquid with the sweet smell typical of a chlorinated solvent. 1,1 DCE is used in the manufacture of paint, varnish, lacquer, soap and finish removers. It is also frequently used as a solvent for cellulose esters, napthalenes, oils, fats, tar and gum and as a cleaning agent in the dry cleaning industry (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: CH_2CCl_2

MW: 96.94
SG: 1.218 at 20°C
FP: none
BP: 37°C
MP: -122.1°C
VP: 500 mmHg at 20°C
Sol. (water): 400 mg/l at 20°C
Sol. (organics): slightly soluble in alcohol, ether, acetone, benzene, and chloroform.

Fate and Transport

Volatilization is the primary route of removal of 1,1 DCE from surface waters. Once in the atmosphere 1,1 DCE is photo-oxidized through hyroxylation. 1,1 DCE will most likely volatilize from surface soils with low organic content but will adsorb to any organic matter present (ICF, 1985). It is speculated, because of work done with similar compounds, that 1,1 DCE would leach readily from soils and would migrate with ground water (EPA 1985).

Pharmacokinetics

1,1 DCE is known to be absorbed rapidly into the digestive tract of rats upon oral administrations (EPA 1985). McKenna et al. (1978) reported the rapid appearance of labelled 1,1 DCE in the urine and expired air of rats given an intragastric dose of ^{14}C labeled 1,1 DCE.

Andersen et. al., (1979) exposed fasted male rats to various concentrations of 1,1-DCE in a closed chamber. They observed an initial rapid phase followed by a slow phase of uptake. They concluded that the rapid phase represented whole body equilibrium while the slow phase represents metabolism.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

In 3 studies (Rampy et al., 1977; Quast et al., 1983) of lab animals orally exposed to 1,1 DCE, no significant effects were noted other than an increased incidence of cytoplasmic vacuolization of hepatocytes.

Inhalation studies revealed that subchronic exposure can lead to liver and kidney damage. Prendergast et al. (1967) exposed rats, guinea pigs, rabbits, dogs and monkeys to atmospheric concentrations of 1,1 DCE ranging from 20 to 395 mg/m^3 for up to 90 days. Continuous exposure to 189 mg/m^3 produced dose related mortality in guinea pigs and monkeys. At high doses, growth depression was noted in all species, as were renal lesions, hepatic lesions and/or enzyme alterations.

The U.S. EPA (1985) reports that chronic studies, both inhalation and oral, generally resulted in hepatocellular fatty changes and periportal hepatocellular hypertrophy. This condition is reversible upon termination of treatment. No increase in mortality, other than from carcinogenesis, was noted in any of the studies.

Teratogenic and Other Developmental Effects

Short et al. (1977) and Murray et al. (1979) both noted signs of fetal toxicity, skeletal alterations and soft-tissue alterations in rats, rabbits and mice as a result of inhalation. The alterations were considered to be manifestations of maternal toxicity.

Mutagenic Effects

Drevon and Kuroki (1979) reported that 1,1 DCE was not mutagenic for V79 cells exposed to vapor in vitro and Cerna and Kypenova (1977) found that it did not produce chomosomal aberrations in bone marrow cells of ICR mice given single or repeated i.p. treatment in vivo.

Reitz et. al., (1980) reported CD-1 mice and Sprague-Dawley rats exposed in vivo to 1,1 DCE showed signs of DNA alkylation and subsequent repair which was specific to liver and kidney, with the kidney of both species exhibiting higher alkylation.

Carcinogenic Effects

Ott et al. (1976) investigated occupational exposure of 138 Dow Chemical Company workers to 1,1 DCE. No statistically significant differences were noted between workers exposed to various concentration of 1,1 DCE.

Of eighteen studies performed on laboratory animals, only one was deemed acceptable in implying 1,1 DCE as a carcinogen (IRIS). Maltoni et al. (1985) exposed Swiss mice to 10 and 25 ppm 1,1 DCE for 4-5 days/week for 12 months. A statistically significant increase in kidney adenocarcinoma was noted in the male Swiss mice. An increase in the incidence of mammary carcinomas was noted, but no dose-response characteristics were observed (IRIS). In a similar study Maltoni noted mammary tumors in Sprague-Dawley rats exposed to concentrations of 10 and 100 ppm 1,1 DCE (IRIS).

Ecotoxicity

1,1 DCE is not extremely toxic to freshwater or saltwater organisms, with LC_{50} values ranging between 80 and 200 mg/l (ICF, 1985).

No data regarding the toxicity of 1,1 DCE to aquatic or terrestrial organisms were located in the literature reviewed.

Standards, Criteria and Guidelines

EPA Class C Carcinogen

Oral Slope Factor:	6.0×10^{-1} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	1.2×10^{0} (mg/kg/day)$^{-1}$
Chronic Oral RfD:	9.0×10^{-3} mg/kg/day
Chronic Inhalation RfD:	currently under review by EPA
Subchronic Oral RfD:	9.0×10^{-3} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	7 µg/L
AWQC:	Water and Fish Consumption - .033 µg/L
	Fish Consumption - 1.85 µg/L

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1,2-DICHLOROETHYLENE

Use

1,2-dichloroethylene exists in two isomers, cis 60 percent and trans 40 percent. The toxicity of these two forms varies. At room temperature, 1,2-dichloroethylene is a liquid with a slight acrid, ethereal odor. It is also known as acetylene dichloride and symdichloroethylene.

It is used as a solvent for acetylcellulose, resins, and waxes. 1,2-dichloroethylene is utilized in the extraction of rubber, in the extraction of oils and fats from fish and meat, as a refrigerant, and in the manufacture of pharmaceuticals and artificial pearls (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: ClCH - CHCl
MW: 96.94
SG: 1.2565 at 20°C
Sol. (water): 600 mg/liter
Sol. (organics): Miscible with alcohol, ether, and acetone. Very soluble in benzene and chloroform.

BP: 47.5°C
MP: -50°C
VP: 200 mmHg at 14°C
FP: 3°C (undef. isomers)

Fate and Transport

The half-life of the trans isomer of this compound has been estimated by the EPA to be 1-6 days with the cis isomer being even lower (U.S. EPA, 1984). Volatilization is probably the main means of dispersion (ICF, 1985).

1,2-dichloroethylene is broken down rapidly by hydroxylation. Some may be absorbed by water vapor and returned to the earth in precipitation, however. (ICF, 1985).

Given that both isomers have low octanol/water partition coefficients, it is expected that evaporation will be the major fate of this compound in surface soils (U.S. EPA, 1984). Tabak et al. (1981) concluded that biodegradation of 1,2-dichloroethylene in subsurface soil is likely to be a slow process. Therefore, the compound is expected to leach from subsurface soil into ground water. In fact, Page (1981) reported a frequency of 51 percent for 1,2-t-dichloroethylene in New Jersey ground waters.

Pharmacokinetics

The U.S. EPA (1980) has estimated that "virtually 100 percent of ingested DCE (dichloroethylene) may be absorbed systematically" based on the studies of Daniel (1963) and Monster et al. (1976) using trichloroethylene. These same studies led the U.S. EPA (1980) to estimate that "35 to 50 percent of inhaled DCE... may be absorbed systematically."

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Springer (1965) administered a mixture of the 1,2- dichloroethylene isomers to rats for seven weeks at concentrations of 0.05, 0.25, 0.5 or 1.0 g/kg. Whether these were daily, weekly or total doses is unclear. No adverse effects were reported at any dose level. Barnes et al. (1985) reported no dichloroethylene-induced changes in gross pathology or terminal body weight at any dose level when male and female CD-1 mice were exposed to the trans isomer in their drinking water at concentrations of 0.1, 1.0 or 2.0 mg/ml.

Contrary to these reports, Jenkins et al. (1974) reported increases in a series of hepatic enzymes in rats, indications of hepatotoxicity resulting from single dose exposures of 400 or 1500 mg/kg of cis-1,2-dichloroethylene introduced by gavage in corn oil. The authors suggested that the cis isomer appears to be slightly more hepatotoxic than the trans isomer with respect to these endpoints. Freundt et al. (1977) found progressive damage to the lungs and fatty changes in the liver when groups of six female Wistar rats were exposed to 100 ppm atmospheric concentrations of trans-1,2-dichloroethylene 8 hrs/day, 5 days/week for 1,2,8 or 16 weeks.

Freundt and Machotz (1978) found that exposure of rats to 100 ppm of cis-1,2-dichloroethylene for 8 hours resulted in inhibition of the MFO system as measured by hexobarbital sleeping time, zoxazolamine paralysis and formation of amino-antipyrine from aminopyrine. They also reported that the cis isomer was a more potent inhibitor than the trans isomer.

Teratogenic and Other Developmental Effects

Pertinent data regarding the teratogenicity of either isomer of 1,2-dichloroethylene were not found in the literature reviewed.

Mutagenic Effects

Greim et al. (1975) reported negative results for mutagenicity by either isomer of 1,2-dichloroethylene using *E,coli K12* as the indicator organism. Cerna and Kypernala (1977) found that both isomers were not mutagenic in *Salmonella* tester strains. They did find that the cis isomer produced a dose-dependent increase in mutations using the host-media bioassay and that it induced

chromosomal aberrations as indicated by cytogenic analysis of bone marrow cells isolated from given repeated intrapetitoned injections while the trans isomer did not.

Carcinogenic Effects

Pertinent data regarding the carcinogenicity of either isomer of 1,2-dichloroethylene was not found in the literature reviewed. The trans isomer has been evaluated by the U.S. EPA for evidence of human carcinogenic potential, and the cis isomer is classified Class D, not classifiable as to human carcinogenicity.

Ecotoxicity

The U.S. EPA (1986) reports that acute toxicity to freshwater aquatic life occurs at concentrations as low as 11,600 µg/liter and it is expected that it would occur at lower concentrations in species more sensitive than those tested.

They also report that the available data indicate that acute and chronic toxicity to saltwater aquatic life occurs at concentrations as low as 224,000 µg/liter and it is expected that it would occur at lower concentrations in species more sensitive than those tested. (U.S. EPA, 1986).

Standards, Criteria and Guidelines

EPA Class D Carcinogen (cis isomer)

Oral Slope Factor:	cis:	NA
	trans:	NA
Inhalation Slope Factor:	cis:	NA
	trans:	NA
Chronic Oral RFD:	cis:	1×10^{-2} mg/kg/day
	trans:	2×10^{-2} mg/kg/day
Chronic Inhalation RFD:	cis:	NA
	trans:	NA
Subchronic Oral RfD:	cis:	1.0×10^{-1} mg/kg/day
	trans:	2.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	cis:	NA
	trans:	NA
MCL:	cis:	70 µg/L
	trans:	100 µg/L
AWQC:	Water and Fish Consumption - 0.033 µg/l	
	Fish Consumption - 1.9 µg/l (for dichloroethylenes)	

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ETHYLBENZENE

Use

Ethylbenzene is a volatile aromatic hydrocarbon that is colorless and highly flammable. It is used as an anti-knock agent in airplane fuels; a solvent; a constituent of asphalt and naphtha; and in the manufacture of styrene and acetophenone (EPA, 1985).

Chemical and Physical Properties

Chemical Formula: C_8H_{10}

MW: 106.2
SG: 0.867 at 20°C
FP: 17.2°C
Sol. (water): 161 mg/l at 25°C
Sol. (organics): soluble in most organic solvents

BP: 136.2°C
MP: -95°C
VP: 7 mmhg at 20°C

Fate and Transport

The transport of ethylbenzene in the environment is not well documented. The major route of elimination from surface water and soils is most likely volatilization. High quantities of organics in the soil would likely cause retention and adsorption of ethylbenzene.

In the atmosphere, ethylbenzene is photooxidized rapidly (EPA, 1985).

Pharmacokinetics

Ethylbenzene is absorbed through the lung, gastrointestinal tract, and skin into the bloodstream. Inhalation studies with ethylbenzene have shown that humans absorb approximately 64 percent of the inhalation dose. Absorbed ethylbenzene is distributed throughout the body but is concentrated in the kidneys, lung, adipose tissue, digestive tract, and liver. The primary metabolites of ethylbenzene formed in humans are mandelic acid and phenylglyoxylic acid, while 1-phenylethanol, benzoic acid, and mandelic acid are the major metabolites formed in rodents. The inhaled ethylbenzene dose is almost completely excreted by humans within 24 hours after exposure is ceased (EPA, 1985).

When ethylbenzene is coadministered with xylenes, xylenes are preferentially metabolized, causing ethylbenzene metabolism to be delayed (EPA, 1985).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Acute dermal exposure (17.8 ml/kg or 15,400 mg/kg) has been demonstrated to produce death in humans. Death occurred in rats after acute exposure to 4,000 ppm (17,400 mg/cu meter) via inhalation. The target organs of acute exposure are the central nervous system and lungs, however, toxic effects have also been observed in the liver and kidneys, (EPA, 1984). A concentration of 100 ppm (435 mg/m^3) did not produce adverse health effects in humans following an 8-hour inhalation exposure. Higher concentrations (values not specified) produced sleepiness, fatigue, headache, and mild eye and respiratory irritation (EPA, 1985).

Chronic oral exposure of ethylbenzene to rats resulted in liver and kidney changes. Increases in liver and kidney weights, cloudiness and swelling of the

hepatocytes and renal tubular epithelium were produced by doses of 408 mg/kg/day. Doses of 13.6 mg/kg/day did not produce effects (EPA, 1985).

Ethylbenzene potentiates the toxicity of acrylonitrile (EPA, 1985).

Teratogenic and Other Developmental Effects

Embrytoxicity, fetotoxicity, and teratogenicity were not observed in rats or rabbits exposed to ethylbenzene via inhalation. Inhalation of 1,000 ppm of ethylbenzene elicited slight maternal toxicity in rats (EPA, 1985).

Mutagenic Effects

There is no evidence of mutagenic activity in *S. typhimurium* following ethylbenzene exposure in assays with and without metabolic activation at concentrations up to 3 mg/plate. No mutations were observed in yeast cells nor in rat liver epithelial cells exposed to ethylbenzene at 0.2 to 2,000 µg/plate. In *Drosophila melanogaster*, there was no increased frequency of recessive lethals (EPA, 1985).

Carcinogenic Effects

Due to the lack of animal and human studies, EPA has not classified ethylbenzene as a Class D carcinogen, not classifiable as to carcinogenicity.

Ecotoxicity

At concentrations greater than 23 mg/L, freshwater species experienced acutely toxic effects. No chronic effects were observed following exposure to 440 µg/L. A bioconcentration factor of 95 was calculated based on the log octanol/water partition coefficient (EPA, 1985). With regard to impacts on vegetation, no adverse effects on chlorophyll production by *Selenatrum capriconatum* or *Skeletonema costratum* was observed at concentrations as high as 438,00 µg/L (EPA, 1980).

Standards, Criteria and Guidelines

EPA Class D carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	1.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	2.86×10^{-1} mg/kg/day
Subchronic Oral RfD:	1.0×10^{0} mg/kg/day
Subchronic Inhalation RfD:	2.86×10^{-1} mg/kg/day
MCL:	0.7 mg/L
AWQC:	Water and Fish Consumption - 1.4 mg/L
	Fish Consumption - 3.3 mg/L

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METHYLENE CHLORIDE

Use

Methylene chloride is a widely used industrial degreaser and paint remover. It is also used as a low temperature extractant and as a solvent for oil, fats, waxes and cellulose acetate (Sittig, 1991). Commercially, methylene chloride is used in aerosols as a flammability depressant, as a weight additive and as a caffeine extractant for coffee and tea (BNA, Inc., 1985).

Chemical and Physical Properties

Chemical Formula: $CH_2 Cl_2$

MW: 84.93	BP: 40°C
SG: 1.32 at 20°C	MP: -95.1°C
Sol. (water): 13,200 mg/l at 20°C	JP: 362.4 mmHg at 20°C
Sol. (organics): alcohol and ether.	

Fate and Transport

Methylene chloride is removed from surface soils and water primarily through volatilization. In the atmosphere, methylene chloride is photo-oxidized and broken down by hydroxyl radicals. Its byproducts include carbon dioxide and, to a lesser extent, carbon monoxide and phosgene (ICF, 1985). Atmospheric methylene chloride may be returned to earth via wet and dry deposition (ICF, 1985). It appears as though methylene chloride does not sorb well to soils and is not heavily bioaccumulated. Because of this, methylene chloride likely leaches readily to the groundwater (ICF, 1985).

Pharmacokinetics

Most cases of human absorption of methylene chloride involve inhalation. Methylene chloride reaches a steady state in the body after less than seven hours of continuous exposure. DiVincenzo and Kaplan (1981) exposed groups of volunteers to between 50 and 200 ppm methylene chloride for 7.5 hours. The pulmonary system was the primary route of absorption. Respiration eliminated less than 5 percent of the methylene chloride absorbed. Methylene chloride is metabolized to carbon monoxide in the body.

In the body, methylene chloride is concentrated in adipose tissue. Savolainen et al. (1977) noted that rats exposed to 200 ppm methylene chloride, for 6 hours/day for 5 days concentrated methylene chloride in the brain, blood, liver, and perirenal fat.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The National Coffee Association (1982) exposed groups of 85 rats/sex to doses of 5, 50, 125, and 250 mg/kg/day methylene chloride for 2 years. Doses of 50 mg/kg/day and larger resulted in histological alterations of the liver.

Subchronic exposure of rats to methylene chloride caused toxic effects in groups exposed to >100 ppm. Narcosis and lethargy were the two most pronounced effects (EPA, 1974). Chronic inhalation studies on workers occupationally exposed to methylene chloride revealed no indications of increased mortality rates from circulatory heart disease or cancer (EPA, 1989).

Direct contact with methylene chloride causes irritation of the mucous membranes in humans. Lassitude, anorexia, numbness and light-headedness are a few of the side effects of chronic exposure (ICF, 1985). Acute exposure is known to cause heart arrythmia and death in humans and liver and kidney damage in laboratory animals.

Haun et al. (1972) observed no effects in rats exposed via inhalation to 87 mg/m^3.

Teratogenic and Other Developmental Effects

Methylene chloride appears not to cause developmental or teratogenic effects in laboratory animals. Elevated levels of carboxyl hemoglobin, resulting from presence of carbon monoxide as a metabolite, were noted in rat fetuses. Mouse fetuses appear to exhibit advanced ossification of the sternebrae (EPA, 1989).

Mutagenic Effects

Methylene chloride was found to be mutagenic to *Salmonella typhimurium* and was noted to cause mitotic recombination in yeast cells (IRIS).

Carcinogenic Effects

NTP (1986) exposed rats and mice to levels of methylene chloride between 0 and 4000 ppm. A significant increase in mammary adenomas, fibroadenomas, hepatocellular adenomas, and carcinomas was evident. In a separate study, methylene chloride was shown to cause a slight increase in the incidence of hepatocellular carcinomas and neoplastic nodules in female rats. In this study, the National Coffee Association (1983) exposed rats to between 5 and 250 mg methylene chloride/kg/day.

Human case studies concerning occupational exposure to methylene chloride have shown little positive carcinogenic data. Friedlander et al (1978) provided evidence which suggested that methylene chloride increased the incidence of pancreatic tumors (IRIS). This evidence was eventually deemed inconclusive.

Ecotoxicity

Very little pertinent information concerning the toxic effects of methylene chloride on wildlife was located. Acute toxicity levels for saltwater species range between 193,000 and 224,000 mg/l. Saltwater species appear to be more tolerant, with acute toxicity levels ranging between 256,000 and 331,000 mg/l.

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	7.5×10^{-3} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	1.60×10^{-3} (mg/kg/day)$^{-1}$
Chronic Oral RfD:	6×10^{-2} mg/kg/day
Chronic Inhalation RfD:	8.57×10^{-1} mg/kg/day
Subchronic Oral RfD:	6×10^{-2} mg/kg/day
Subchronic Inhalation RfD:	8.57×10^{-1} mg/kg/day
MCL:	NA
AWQC:	Water and Fish Consumption - 4.7 µg/L (recalculated)
	Fish Consumption - 1600 µg/L (recalculated)

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STYRENE

Use

Styrene, also known as cinnamene, cinnamol, and vinyl benzene, is a colorless to yellowish, very refractive, oily liquid with a penetrating odor. It polymerizes to form the plastic polystyrene when heated to 200°C. Styrene is combined with 1,3-butadiene or acrylonitrile to form copolymer elastomers, butadiene-styrene rubber, and acrylonitrile-butadiene-styrene (ABS). It is also used in the production of resins, polyesters, insulators and various pharmaceuticals (Sittig, 1991).

Chemical and Physical Properties

MF: $C_6H_5CH = CH_2$

MW: 104.14 BP: 145-146°
Fl.Pt. 31°C (closed up) FP: -30.6°
Sol. (water): sparingly
Sol. (organics); soluble in alcohol, ether, methanol, acetone, and carbon disulfide

Fate and Transport

The U.S. EPA (1984) reports that the atmospheric fate of styrene is determined by its chemical and photochemical reactivity, as well as the activity of atmospheric physical processes. Studies by Dalta and Rao (1979) and Graedel (1978) indicate that the reaction of styrene with singlet oxygen is not significant in determining the fate of atmospheric styrene, while several other studies indicate that reaction of styrene with ozone, OH radicals, and NO_x and natural sunlight appear to be much more significant. The U.S. EPA (1984) reports that physical processes such as dry deposition and washout via rain or snow are unlikely to play a significant role in determining the fate of styrene considering the relatively high chemical and photochemical reactivity of this compound.

In natural aquatic media, U.S. EPA (1984) reports that the fate of styrene is likely to be determined by its ability to undergo chemical, photochemical and microbial reactions, as well as physical processes such as volatilization and sorption. Howard and Ingold (1968) and Mill et al. (1982) performed studies indicating that reaction of styrene with peroxy radicals is not a significant fate process in aquatic media. The U.S. EPA (1984) reports that photopolymerization may possibly play a significant role in determining styrene's fate in aquatic media. Microbial degradation appears to occur relatively rapidly in aquatic media, with studies reporting between 42% and 80% degradation of styrene within 5 days, depending on the nature of the microbial inoculum. The U.S. EPA (1984) reports that the formation of chlorohydrin from chlorine and styrene may be significant during chlorination of drinking water. Styrene will volatilize from water relatively rapidly while some may be removed through sorption and subsequent sedimentation of particulate matter (U.S. EPA, 1984).

Studies on the fate of styrene in soils have focused on the biodegradability of this chemical. Sielicki et al. (1978) found that 95% degradation of styrene occurred in 16 weeks in landfill soil; 87% degradation occurred in sandy loam soil during the same time period. Sielicki et al. (1978) also found that degradation was slower at higher levels of styrene. Wilson et al. (1983) found degradation of styrene in subsurface soils to be slow (2.3-12%/week). The U.S. EPA (1984) reports that volatilization of styrene from surface soil appears likely to be a significant loss mechanism.

Roberts et al. (1980) studied the transport characteristics of styrene in aquifers. They report that styrene was adsorbed relatively strongly by a sand aquifer and that, in cases where adsorption is the removal process, the aquifer capacity ultimately is exhausted and breakthrough occurs. A study by Grossman (1970) clearly demonstrates that styrene may leach through soil to ground water under certain conditions. His data indicate that styrene may persist in certain soils for at least 2 years.

Pharmacokinetics

The U.S. EPA (1985) reports that absorption of styrene from the GI tract has been shown by Plotnick and Weigel (1979) to be rapid and virtually complete. The U.S. EPA (1988) also cites several studies that have illustrated styrene uptake and absorption from inhalation. These human studies indicate that pulmonary retention of styrene is approximately two-thirds of the administered dose with dramatic variation in uptake between individuals and studies.

Plotnick and Weigel (1979) found that 20 mg/kg doses of ^{14}C-styrene administered in corn oil by gavage were distributed to the kidneys, liver, and pancreas of rats preferentially, with lower concentrations occurring in the lungs, heart, spleen, adrenals, brain, testes, and ovaries. Inhalation studies by Withey and Collins (1979) illustrated widespread distribution with relatively high concentrations in adipose tissue. In humans, Dowty et al. (1976) found concentrations of transplacentally transferred styrene to be somewhat higher than those of maternal blood, which suggests a selective one-way transplacental transfer.

Ohtsuji and Ikeda (1971) showed styrene to be metabolized to hippuric acid or phenylglyoxylic acid with several intermediates including benzoic acid.

The U.S. EPA (1988) states that a number of studies indicate that styrene is eliminated relatively rapidly from all tissues in animals. Plotnick and Weigel (1979) found tissue and organ concentrations of ^{14}C-styrene in rats to be <1 µg/g 24 hours after oral administration of 20 mg/kg.

Human-Toxicity

Noncarcinogenic Effects

Systemic Effects

Quast et al. (1979) exposed beagle dogs (4/sex) to 0, 200, 400, and 600 mg/kg bw/day doses of styrene in peanut oil by gavage for 560 days. Adverse effects were only observed in the two higher dose groups and included increased number of Heinz bodies in the red blood cells, decreased packed cell volume, and sporadic decreases in hemoglobin and red blood cell counts.

Increased iron deposits and elevated numbers of Heinz bodies were detected in the liver as well. The severity of these effects varied among individual animals at the same dose level.

Ponomarkov and Tomatis (1978) reported liver, kidney, and stomach lesions in rats exposed to 500 mg/kg styrene weekly for 120 weeks. Mice exposed to 300 mg/kg for the same duration illustrated no significant effects. Similarly, Wolf et al., as cited by IRIS, found no adverse effects in rats receiving an average daily oral dose of 95 mg styrene/kg bw for 185 days while doses of 285 or 475 mg/kg/day produced reduced growth and increased liver and kidney weights.

Teratogenic and Other Developmental Effects

Murray et al. (1976, 1978) administered styrene to pregnant Sprague-Dowley rats by gavage at doses of 0, 180, or 300 mg/kg/day on days 6 through 15 of gestation. Maternal toxicity was exhibited on days 6 through 9 and included reduced body weight gain and food consumption. No other adverse effects were observed in the rats or their fetuses.

Hemminiki et al. (1980) found a positive correlation between exposure to styrene and the incidence of spontaneous abortion in female members of the Finnish Union of Chemical Workers. Conclusive evidence could not be drawn due to the large number of variables inherent in the study, however.

Mutagenic Effects

The U.S. EPA (1988) reports that six mutagenicity tests using *Salmonella typhimurium* tests systems with and without S-9 metabolic activation yielded negative results. Similarly, De Meester et al. (1977, 1981) and Vainio et al. (1976) reported negative results in bacterial strains sensitive to frameshift mutagens. However, these studies produced positive results with mutant strains sensitive to base pair substitution.

Carcinogenic Effects

The U.S. EPA (1988) reports that data regarding the carcinogenicity of styrene are inconclusive. Several long-term bioassays (Jersey et al., 1978; Ponomarkov and Tomatis, 1978; NTP, 1983; Mattoni et al., 1982) have resulted in inconsistent incidences of tumor formation and excessive mortality among treated animals. Similarly, a host of retrospective cohort mortality and case-control studies conducted on workers exposed to styrene in the styrene-polystyrene manufacturing industry or the styrene-butadiene synthetic rubber industry provided inadequate data because of the relatively small cohort sizes and multiple chemical exposures of workers (including exposure to benzene). It should be noted, however, that these studies did reveal an elevated incidence of tumors of the hematopoietic and lymphatic tissues (McMichael et al., 1976; Smith and Ellis, 1977; Meinhardt et al., 1978).

Ecotoxicity

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor:	3.00×10^{-2} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	2.00×10^{-3} (mg/kg/day)$^{-1}$
Chronic Oral RfD:	2×10^{-1} mg/kg/day
Chronic Inhalation RfD:	currently under review by EPA
Subchronic Oral RfD:	2.0×10^{0} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.1 mg/l
AWQC:	NA

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1,1,2,2-TETRACHLOROETHANE

Use

1,1,2,2-Tetrachloroethane is a colorless, man-made liquid with a chloroform-like odor. It has been used as a metal degreaser, a solvent, and in paints and pesticides. Currently, it is largely used as a chemical intermediate (ATSDR, 1989)

Chemical and Physical Properties

Chemical Formula: $C_2H_2Cl_4$

MW:	167.85	MP:	-43.8°C
SG:	1.59 at 20°C	BP:	145.1°C
VP:	5.95 mmHg at 25°C		
Sol. (water):	2,870 mg/L at 20°C		

Fate and Transport

In the atmosphere, 1,1,2,2-tetrachloroethane is relatively unreactive. It's theoretical half-life in the atmosphere is 53.3 days (Atkinson, 1987). This decay is mainly caused by reactions with photochemically-produced hydroxyl radicals.

The fate of 1,1,2,2-tetrachloroethane in soils is unknown. Theoretically it is expected to biodegrade anaerobically and hydrolyze (ATSDR, 1989).

According to Cooper et al. (1987) 1,1,2,2-tetrachloroethane undergoes base-catalyzed hydrolysis to form trichloroethane in aquatic media. At typical environmental pHs, Cooper obtained a half-life of 102 days for 1,1,2,2-tetrachloroethane. Klecka and Gonsior (1983) conducted a similar study under sterile anaerobic conditions. Although half-lifes varied, 1,1,2,2-tetrachloroethane underwent hydrolytic dehalogenation to trichloroethane (ATSDR, 1989).

Under aerobic conditions, Tabak et al. (1981) found that 1,1,2,2-tetrachloroethane did not noticeably degrade when incubated with sewage seed in water.

Pharmacokinetics

Morgan et al. (1970) showed that 97 percent of an inhaled dose of 1,1,2,2-tetrachloroethane is absorbed by humans. The only study located regarding the dermal absorption of 1,1,2,2-tetrachloroethane was conducted on mice and guinea pigs. Jakobsen et al. (1982) and Tsuruta (1975) reported that these two mammals absorbed up to 1 ml of 1,1,2,2-tetrachloroethane applied to skin within thirty minutes. Mitoma et al. (1985) reported that rats and mice metabolized 70 percent of an orally administered dose of 1,1,2,2-tetrachloroethane within 48 hours.

Once absorbed into a mammalian body 1,1,2,2-tetrachloroethane is thought to accumulate primarily in the liver (ATSDR, 1989). Mitoma et al. (1985) reported that mice and rats hepatic proteins bound 1,1,2,2-tetrachloroethane at a high rate

No studies were located regarding 1,1,2,2-tetrachloroethane's metabolism in humans. Yllner (1971) and Mitoma et al. (1985) reported that 1,1,2,2-tetrachloroethane is metabolized in rats and mice to trichloroethanol, trichloroacetic acid, and dichloroacetic acid. These compounds are commonly broken down further into glyoxylic acid and oxalic acid (ATSDR, 1989). 1,1,2,2-tetrachloroethane also degrades non-enzymatically through dehydrochlorination by alkali into trichloroethylene and tetrachloroethylene.

Human Toxicity

Non-Carcinogenic Effects

Systemic Effects

Excessive inhalation of 1,1,2,2-tetrachloroethane has been shown to cause death in workers exposed occupationally in varnishing shops. Levels in the air during these occurrences are unknown (ATSDR, 1989). Smyth et al. (1969) showed that atmospheric concentrations in excess of 1,000 ppm kills rats and mice.

Hepple (1927) reported that a human committed suicide by drinking approximately 285 mg/kg-bw of 1,1,2,2-tetrachloroethane. One human death from dermal exposure was reported by Coyer (1944). Animal studies conducted by Smyth (1969) indicate that oral exposure to 200 mg/kg-bw and dermal exposure to 6.38 g/kg-bw caused death to rats and rabbits, respectively.

No significant respiratory or cardiovascular effects have been noted in humans or animals after inhalation exposure to 1,1,2,2-tetrachloroethane. The most notable systemic effects of acute and chronic exposure to 1,1,2,2-tetrachloroethane in humans and animals were noted in the liver. Jeney et al. (1957) reported that ambient concentrations of 1,1,2,2-tetrachloroethane between 1.5 and 36 ppm caused jaundice in workers exposed occupationally. Other hepatic effects of exposure to 1,1,2,2-tetrachloroethane include shrunken liver, neurosis, enzymatic changes, and centrilobular vacuolization (ATSDR, 1989). Kroner et al. (1981) reported that dermal application to guinea pigs of 513 mg/cm^2 of 1,1,2,2-tetrachloroethane for 16 hours caused karyopyknosis and pseudoe-osinophilic infiltration (ATSDR, 1989).

Teratogenic and Other Developmental Effects

Only one study was located regarding the developmental effects of 1,1,2,2-tetrachloroethane. Schmidt (1976) reported that 1,1,2,2-tetrachloroethane administered intraperitoneally to mice during gestation caused moderate effects to skeletal development at high doses (700 mg/kg). The authors consider the chemical to be embryotoxic but only weakly teratotoxic.

Mutagenic Effects

Studies regarding the mutagenic effects of 1,1,2,2-tetrachloroethane were not located in the literature reviewed.

Carcinogenic Effects

No studies regarding 1,1,2,2-tetrachloroethane's carcinogenicity to humans were located in the available literature. NCI (1978) reported an increased incidence of hepatocellular carcinomas in rats and mice exposed by gavage, to 1,1,2,2-tetrachloroethane in corn oil for 5 days per week for 78 weeks. The lowest dose at which these effects were noted was 142 mg/kg/day.

Ecotoxicity

No studies regarding the ecotoxicity effects of 1,1,2,2-tetrachloroethane were located in the available literature.

Standards, Criteria and Guidelines

Class C Carcinogen

Oral Slope Factor:	2.0×10^{-1} mg/kg/day
Inhalation Slope Factor:	2.0×10^{-1} mg/kg/day
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - .17 µg/L
	Fish Consumption Only - 10.7 µg/L

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TETRACHLOROETHYLENE

Use

Tetrachloroethylene, often called perchloroethylene (PCE), is a clear liquid with an odor similar to that of ether. Its major uses are as a dry-cleaning solvent and as a degreaser. PCE is also used as a fumigant, a chemical intermediate, and medically as an anthelmintic (ACGIH, 1984).

Chemical and Physical Properties

Chemical Formula: C_2Cl_4

MW:	165.83	BP:	121°C
SG:	1.63 at 20°C	MP:	-22.7°C
FP:	none	VP:	14 mmhg at 20°C

Sol. (water): 150 to 200 mg/l at 20°C
Sol. (organics): alcohol, ether and benzene.

Fate and Transport

Tetrachloroethylene volatilizes rapidly when released to surface waters and soils. In the atmosphere, tetrachloroethylene interacts with hydroxyl radicals to produce carbon dioxide, carbon monoxide, and hydrogen chloride (ICF,1985).

In soils, tetrachloroethylene adsorbs to the organic material present. In soils of low organic content, tetrachloroethylene leaches and is transported readily in the ground water (EPA 1985). Tetrachloroethylene is known to degrade slowly in ground water, where it can remain for months to years. Its degradation products in aquatic media are reported to be vinyl chloride and dichloroethylene (EPA, 1985).

Pharmacokinetics

When absorbed into the bloodstream, tetrachloroethylene is distributed mainly to fatty tissues. Much lower concentrations can be found in the blood and liver of humans. Rats absorb tetrachloroethylene into most body tissues, with concentration levels in the brain, lungs, and fat increasing proportionally with exposure. Blood and liver concentrations tend to level off after a three hour period (EPA, 1985).

Only 4 percent of tetrachloroethylene absorbed by humans is metabolized. Metabolites include trichloroethanol, trichloroacetic acid and other unidentified chlorinated products (EPA, 1985). Absorbed tetrachloroethylene is primarily respired through the lungs. Its metabolites are eliminated via the urine, with a half-life of 144 hours (EPA, 1985).

When taken orally, tetrachloroethylene is absorbed through the gastrointestinal lining. Fats and oils are known to facilitate absorption in dogs (EPA, 1984).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

No significant case studies on human exposure to tetrachloroethylene were located in the available literature, although chronic exposure is reported to effect the central nervous system, mucous membranes, eyes, and skin. Unconsciousness, dizziness and vertigo are reported to have occurred after acute occupational exposure and several fatalities have been reported as a result of massive accidental exposure (unspecified concentrations) (ACGIH, 1984). For mice, the oral LD_{50} has been reported to be 8.85 mg/kg-bw, with the LC_{50} in air reported as 6000 ppm over a 4 hour period (ACGIH, 1984).

Buben and O'Flaherty (1985) reported that Swiss-Cox mice exposed, by gavage to between 20 and 2000 mg tetrachloroethylene/kg-bw exhibited signs of toxicity in the liver. At higher doses, decreased DNA content, increased SGPT and hepatocellular necroses were noted.

Rowe et al. (1952) reported that rats, when exposed to 1600 ppm tetrachloroethylene for 7 hours/day, 5 days/week over a 25 day period, initially exhibited drowsiness and depression. Enlarged livers and kidneys were noted after 4 weeks.

In the same study, Rowe exposed rabbits, guinea pigs and monkeys to 100-400 ppm tetrachloroethylene for 7 hours/day, 5 days/week for approximately 6 months. No abnormal growth, organ function or histopathological findings were noted.

In a study of chronic oral exposure, the National Cancer Institute (NCI) administered, by gavage, doses between 300 and 949 mg/kg/day tetrachloroethylene to Osborne-Mendel rats and B6C3F1 mice. Toxic nephropathy was observed at all dose levels.

Teratogenic and Other Developmental Effects

Tetrachloroethylene is known to cause increased fetal resorption, subcutaneous edema, split sternebrae, and delayed skull ossification in mice and rats after exposure to 300 ppm for 7 hours/day on days 6-15 of gestation (Schwetz et al., 1975).

No information concerning developmental effects on humans was found in the available literature.

Mutagenic Effects

In an abstract, Cerna and Kypenova (1977) reported that tetrachloroethylene caused mutagenic effects in a *Salmonella* strain, but since details of methodology were not presented, the reliability of the experiment has been questioned.

Carcinogenic Effects

Tetrachloroethylene was found to be carcinogenic in mice and rats. No studies with definitive findings are available showing the carcinogenic effects of tetrachloroethylene on humans, although Blair et al. (1979) observed an excess of lung, cervical and skin cancers and a slight excess of leukemia amongst 330 deceased laundry and dry-cleaning workers. The workers, however, were also exposed to carbon tetrachloride and trichloroethylene.

NCI (1977) noted a significant increase in hepatocellular carcinoma in B6C3F1 mice exposed, by gavage, to between 386 and 1072 mg/kg-bw/day, 5 days/week for 78 weeks. No increase in tumor incidence was noted in rats exposed to similar concentrations.

No significant increase in malignant tumors was noted in an inhalation study performed by Rampy et al. (1977) on Sprague-Dawley rats.

Ecotoxicity

Tetrachloroethylene is considered to be moderately toxic to aquatic organisms. Trout were reported to exhibit on LC value of 4,800 µg/l. This was the most sensitive species tested (ICF, 1985)

No information concerning tetrachloroethylene's toxicity to terrestrial organisms was located in the available literature.

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	5.1×10^{-2} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	1.82×10^{-3} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	1×10^{-2} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	1×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.005 mg/l
AWQC:	Water and Fish Consumption - 0.8 µg/L
	Fish Consumption - 8.9 µg/L

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TOLUENE

Use

Toluene is a clear, colorless, organic compound with a benzene-like odor. It is highly flammable and extremely volatile. In industry, toluene is used in the production of benzene, as a solvent in paint thinners, and as additive to gasoline and other petroleum products. It is estimated that 100,000 workers in the United States are exposed to toluene annually (Sittig, 1991).

The majority of toluene releases to the environment occur from spills of gasoline and from improper disposal of toluene containing products. Every-day exposure to toluene occurs from gasoline and from the use of commercial paints and paint thinners.

Chemical and Physical Properties

Chemical Formula: $C_6H_5CH_3$

MW: 92	BP: 110.6°C
SG: 0.867 at 20°C	MP: -95°C
FP: 4.4°C	VP: 28.7 mmhg at 25°C

Sol. (Water): 0.05%
Sol. (organics): acetone, carbon disulfide; miscible with alcohols, ether, benzene, chloroform, and glacial acetic acid.

Fate and Transport

Volatilization is the predominant route of removal of toluene from soils and aquatic environments. Toluene degrades rapidly in the air where it has a half life of 1.3 days (EPA, 1985c). It readily biodegrades in soils and surface waters. Toluene is transported easily in ground water, where it is known to remain stable. A 1988 EPA study found toluene present at 29 percent of hazardous waste sites surveyed. The average ground water concentration was 21 ppb.

Toluene occurs at low levels in drinking water, food and air. In urban settings, toluene is found in the air at levels of approximately 10 ppb. According to an EPA National Screening Survey, approximately 3 percent of all surface water derived drinking water systems are contaminated with toluene at levels higher than 0.1 µg/l (EPA, 1985b).

Pharmacokinetics

Studies on humans and animals have shown that toluene is absorbed quickly through the respiratory tract. In humans, inhalation exposures of 100 to 130 ppm for 4 hours resulted in a 40 to 60 percent uptake and retention of toluene. Absorption in the gastrointestinal tracts of

male rats was relatively rapid with maximal blood-toluene levels being reached within 2 hours after gastric incubation (EPA, 1985); maximal blood levels following inhalation were reached in 15 to 30 minutes. Dermal absorption of aqueous toluene across human skin is related directly to concentration. Due to its lipophilic nature and low water solubility, toluene is expected to distribute to and accumulate in lipid tissue. A study with male rats revealed that toluene was distributed through the body with the greatest accumulation in lipid tissue. Toluene and its metabolites were also found in relatively high concentrations in tissues active in metabolism and excretion such as the liver and kidneys (EPA, 1985c). Toluene appears to be metabolized in humans and in animals through similar pathways (EPA, 1985). From inhalation studies, it is seen that side-chain hydroxylation to benzyl alcohol occurs. Benzyl alcohol is then conjugated with glycine to form hippuric acid, and is then excreted rapidly in the urine, generally within 12 hours of exposure. The half-life for toluene in adipose tissue of male humans exposed to 300 ppm toluene for 2 hours ranged from 0.5 to 2.7 days (ATSDR, 1989).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Human exposure to toluene is usually a result of inhalation of vapors in occupational settings. Intentional abuse and experimental inhalation also frequently occur (EPA, 1985b). Acute exposure to 200 ppm toluene for 8 hours caused fatigue, headache and nausea (EPA, 1985c). Subacute exposure resulted in similar but proportionately less severe symptoms. Studies of workers exposed to 200-500 ppm toluene occupationally for "many years" show that coordination, memory, and visual aptitude are all impaired. Cerebral dysfunction, such as tremors and ataxia, were also noted (EPA, 1985c). In a study done by Greenberg et al, it was shown that chronic exposure to toluene can also cause kidney dysfunction. A number of studies have reported chromosomal damage in the bone marrow (EPA, 1984). There is, however, no definitive evidence that toluene causes serious irreversible organ damage following chronic exposure (EPA 1985c).

Teratogenic and Other Developmental Effects

In one abstract, scientists reported an increase in fetal mortality in mice. In this study, gavage doses of 0.3, 0.5, and 1.0 ml/kg bw were administered daily on days 6-15 of the gestation period (EPA, 1985a).

Mutagenic Effects

Several studies cited by IRIS report no signs of mutagenic activity with toluene exposure. IRIS did cite a few Russian studies, however, that report toluene as effective in causing chromosomal damage in bone marrow cells of rats.

Carcinogenic Effects

There is no evidence that toluene is carcinogenic to humans.

No carcinogenic effects were seen in studies done on acute or chronic exposure of toluene to rats. These studies include topical, inhalation, and gavage exposures.

Ecotoxicity

In a 13 week gavage study on rats, animals that received 5,000 mg/kg-bw/day died during the first week (U.S. DHHS, 1990). Lower doses over the same period resulted in a reduction in body weight (16 percent) and an increase in organ size. The livers, lungs, hearts and kidneys were all seen to be greater in size and weight than those of the control rats.

Inhalation studies on rats showed that levels of 1200 ppm toluene over a period of 2 years resulted in an increase in the degeneration of olfactory and respiratory epithelial tissues. No weight changes or survival differences were noted (U.S. DHHS, 1990).

Five freshwater species of zooplankton displayed LC_{50} values for toluene of 12,700 to 313,000 µg/l. This is considered "practically nontoxic" (U.S. DHHS, 1990).

Standards, Criteria and Guidelines

EPA Class D carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	5.71×10^{-1} mg/kg/day
Subchronic Oral RfD:	2.0×10^{0} mg/kg/day
Subchronic Inhalation RfD:	5.71×10^{-1} mg/kg/day
MCL:	1.0 mg/L
AWQC:	Water and Fish Consumption - 14.3 mg/L
	Fish Consumption - 424 mg/L

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1,2,4-TRICHLOROBENZENE

Use

1,2,4-Trichlorobenzene (TCB) is used as a dye carrier (46 percent), herbicide intermediate (28 percent), heat-transfer medium, dielectric fluid in transformers, degreaser, lubricant, and potential insecticide against termites. The other TCB isomers are not used in any quantity (Sittig, 1991).

Physical and Chemical Properties

Chemical Formula: $C_6H_3Cl_3$

MW: 181.46	BP: 213.5°C
VP: 0.45 mm Hg at 25°C	MP: 17°C
Sol. (water): 25-35 mg/l at 25°C	
Sol. (organics): NA	

Fate and Transport

TCB is expected to exist primarily in the vapor phase in the atmosphere and is removed by reaction with photochemically generated HO radicals (U.S. EPA, 1987). Atkinson (1985) estimates the half-life of this removal process to be approximately 18.8 days. In aquatic systems, TCB most likely adsorbs to suspended solids and sediments and may bioaccumulate in some organisms (U.S. EPA, 1987) Wakeham et al. (1983) illustrated volatilization as an important fate process for TCB in mesocosm experiments with aerated sea water. Zoeteman et al. (1980) reports the half-lives of TCB in rivers, lakes, and ground water to be 0.3 - 3, 3 - 30, and 30 - 300 days, respectively. TCB is expected to remain strongly sorbed to soils and will not, therefore, leach appreciably into ground water (U.S. EPA, 1987). However, TCB has been detected in ground water indicating that it can be transported in soils under certain conditions. Marinucci and Bartha (1979) report that TCB is expected to biodegrade slowly in soils while Roberts et al. (1980) reports that it is not expected to biodegrade in ground water.

Pharmacokinetics

Data on the pharmacokinetics of TCB were limited. The only available data are on the absorption of TCB. Lingg et al. (1982) reports that male Charles River rats and female rhesus monkeys absorb at least 89 percent and 99 percent of a TCB dose through the gastrointestinal tract, respectively. Twenty-four hours after orally administering 10 mg of radioactive TCB, 84 percent and 40 percent of the radioactivity was recovered from the urine of the rats and monkeys, respectively, while only 11 percent and 1 percent, respectively, was detected in the feces.

Kociba et al. (1981) reports that TCB is absorbed from the respiratory tract. Absorption rates were not provided.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The only available data on the systemic toxicity of TCB are from subchronic exposures studies. Carlson and Tardiff (1976) report increased liver-to-body weight ratios and altered liver enzyme activities in male CD rats throughout a 30-day recovery period following 90 days of exposure to 40 mg/kg/day. Carlson (1977) exposed groups of five female rats to 0, 50, 100, or 20 mg TCB/kg/day in corn oil by gavage for 30, 60, 90, or 120 days. After 30 days, liver porphyrin and urinary porphyrin increases were observed in the 100 mg/kg/day group and 200 mg/kg/day group, respectively. Sixty days of exposure resulted only in increased liver weights. After 90 days of exposure, however, increases in liver weights at ≥50 mg/kg/day, in liver porphyrins at ≥100 mg/kg/day, and in urinary porphyrins at 200 mg/kg/day were observed. A significant increase in liver porphyrins was found after 120 days of exposure at levels ≥50 mg/kg/day.

Goto et al. (1972) reports no adverse effects on ICR-ICL mice exposed to 600 ppm TCB in the diet for 6 months. Parameters observed included body weight, liver, heart, and kidney weights, and the incidence of hepatic and other lesions.

In an inhalation study by Kociba et al. (1981), 20 male Sprague-Dawley rats, 4 male New Zealand rabbits, and 2 male beagle dogs were exposed to 0, 30, or 100 ppm TCB for 7 hours/day, 5 days/week for a total of 30 exposures in 44 days. Gross and comprehensive histological examinations revealed no significant treatment-related effects in any species tested. At 100 ppm, dogs exhibited increased liver weights, and rats exhibited increased liver and kidney weights. At 30 ppm and 100 ppm, rats exhibited increased urinary excretion of porphyrin. This was considered a compound-specific physiological effect rather than a toxic effect.

Teratogenic and Other Developmental Effects

Pregnant Sprague-Dawley rats were exposed to 0, 36, 120, 360, and 1200 mg/kg TCB in corn oil on days 9-13 of gestation (Kitchin and Ebron, 1983). In the 120 mg/kg and 360 mg/kg groups, mortality increased to 100 percent and 22%, respectively. In addition, rats in these two dose groups exhibited reduced body weight gain, moderate hepatocellular hypertrophy, and induction

of hepatic enzymes. Only fetuses from the 0 and 360 mg/kg groups were examined. Fetuses from the 360 mg/kg group exhibited significantly decreased embryonic head length, crown-rump length, somite number, and total embryo protein content.

Robinson et al. (1981) report no adverse effects on fertility, survival, growth, locomotor activity or blood chemistries in a 3-generation reproductive-teratogenic effect study with Charles River rats. The rats were exposed to 0, 25, 100, or 400 ppm TCB in drinking water. The only observed effect was significantly enlarged adrenals at 95 days of age in both sexes of the F_0 and F_1 generation at 400 ppm exposure.

Mutagenic Effects

Schoeny et al. (1979) and Lawlor et al. (1979) both report negative results for mutagenicity tests with *Salmonella typhimurium* test strains. No other data on the mutagenicity of TCB were available in the literature reviewed.

Carcinogenic Effects

Yamamoto et al. (1982) applied TCB in acetone to the dorsal skin of Slc.ddy mice twice weekly for 2 years. The doses were 60 percent and 30 percent TCB in 0.03 ml volume/application. Survival was reduced as low as 60 percent by week 40 at both doses. Histopathology revealed increased non-neoplastic lesions in the lung, liver, kidney, adrenal, spleen, and lymph node of the male mice exposed to 60 percent TCB; and in all these tissues except the lymph node for the female mice exposed to 60 percent TCB.

Although there was no statistically significant increase in the incidence of tumors, male mice exhibited nine tumor types at the high dose, as opposed to two in the low dose and controls. Female mice exhibited eleven tumor types at the high dose while low dose females exhibited three types and control females, eight.

Ecotoxicity

ICF (1985) reports that the acute LC_{50} values of 1,2,4-TCB for the freshwater species *Daphnia Magna*, rainbow trout, and fathead minnow are 50.2, 1.5, and 2.87 mg/l, respectively. Chronic toxicity of 1,2,4-TCB occurred at 0.206-0.705 mg/l for the fathead minnow in its early life stage. In saltwater, the mysid shrimp and sheepshead minnow had acute LC_{50} values of 0.45 and 21.4 mg/l, respectively. Algae in freshwater and saltwater had EC_{50} values for 1,2,4-TCB effects on chlorophyll of 35.3 and 8.75 mg/l, respectively, and for 1,2,4-TCBs effect on cell numbers, values of 36.7 and 8.93 mg/l, respectively.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slop Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	1.31 x 10^{-3} mg/kg/day
Chronic Inhalation RfD:	3.0 x 10^{-3} mg/kg/day
Subchronic Oral RfD:	1.31 x 10^{-2} mg/kg/day
Subchronic Inhalation RfD:	3.0 x 10^{-2} mg/kg/day
MCL:	0.009 mg/l (1,2,4-TCB)
AWQC:	NA

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1,1,2-TRICHLOROETHANE

Use

1,1,2-Trichloroethane (1,1,2-TCA) belongs to the class of halogenated hydrocarbons. It is used as a chemical intermediate and as an organic solvent in the timber, plastics, and synthetics industries. 1,1,2-TCA may be manufactured; it may also be formed naturally from the breakdown of 1,1,2,2-tetrachloroethane in anaerobic environments. 1,1,2-TCA is also known as vinyl trichloride, beta-trichloroethane, and ethane trichloride.

Chemical and Physical Properties

MF: $CH_2ClCHCl_2$
MW: 133.42
PD: Nonflammable, colorless liquid; sweet odor
MP: -35°C
BP: 113°C
SG: 1.44 at 20°C
VP: 22.49 mm Hg at 25°C
Sol. in water: 4,400 mg/l at 20°C
Sol. in organics: Miscible with alcohols, ethers, esters, and ketones

Fate and Transport

Due to its low soil adsorption coefficient, 1,1,2-TCA in soil will tend to leach into ground water and/or to volatilize. The fate of 1,1,2-TCA in ground water is eventual biodegradation; in anaerobic environments this will occur more rapidly. The primary fate of 1,1,2-TCA in surface soils or surface water is volatilization. The estimated half-life of 1,1,2-TCA in a river is 4.5 hours (ATSDR, 1988).

Atmospheric 1,1,2-TCA will gradually oxidize, with an estimated half-life in air of 49 days (ATSDR, 1988). Some amount may be "washed out" by precipitation, but will most likely re-volatilize shortly afterwards. Since the fate processes for atmospheric 1,1,2-TCA occur slowly, considerable dispersal of the contaminant from the source area is expected.

Pharmacokinetics

1,1,2-TCA is quickly absorbed into the bloodstream after inhalation (Morgan et at., 1970 and 1972). Animal studies also indicate oral and dermal doses are absorbed quickly and almost completely (ATSDR, 1988).

Due to its moderate solubility in lipids, 1,1,2-TCA is easily distributed and retained in fat

tissues, the kidneys, the liver, and the brain (Morgan, 1972). The major metabolic process for 1,1,2-TCA is glutathione-dependent and results in the formation of 5-carboxymethyl cysteine, thiodiacetic acid, and chloroacetic acid (ATSDR, 1988).

Excretion of 1,1,2-TCA is fairly slow relative to its metabolism. Most of an inhaled or ingested dose is excreted as metabolites in the urine, with an estimated half-life of 70 minutes (Morgan, 1970).

Simultaneous exposure to 1,1,2-TCA and polybrominated biphenyls increases the renal toxicity of 1,1,2-TCA; simultaneous exposure with phenobarbital increases hepatotoxicity. Diabetics and other people with hyperglycemic tendencies also exhibit increased hepatotoxic effects from exposure to 1,1,2-TCA (ATSDR, 1988).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

There is no evidence of mortality in humans resulting from exposure to 1,1,2-TCA.

Various species tested all exhibited mortality after exposure to 1,1,2-TCA by ingestion, inhalation, contact, or subcutaneous injection. Inhalation LC_{50} values for rats and mice have been recorded at 1654 and 416 ppm respectively (Bonnet et al., 1980; Gradiski et al., 1978). Oral doses administered by gavage resulted in LD_{50} values of 837 mg/kg for rats and 378 mg/kg for mice. Differing reactions to exposure were noted among different species and between males and females of each species.

Both ingestion and inhalation of 1,1,2-TCA have been shown to cause liver damage, incuding necrosis, elevated liver enzyme levels, and reduced glycogen levels, in rats, mice, guinea pigs, and dogs. At least one study (White et al., 1985) reported different reactions to 1,1,2-TCA exposure in male and female mice. Males exhibited decreased glutathione levels, while females showed increased glutathione and also elevated enzyme levels.

A limited amount of evidence indicates that 1,1,2-TCA may be associated with kidney damage, including swelling and congestion (ATSDR, 1988). Plaa et al. (1958, 1965, 1966, 1967) conducted a series of studies in mice and dogs receiving injections of 1,1,2-TCA. Subcutaneous injections of 173 mg/kg in mice caused tubular lesions with microscopic necrosis in the renal cortex, while intraperitoneal injections of 289 mg/kg resulted in necrosis, swelling, and general renal dysfunction. Female mice exhibited less severe or no effects even at high doses, further suggesting a gender-specific

reaction to 1,1,2-TCA.

Sanders et al. (1985) reported a lowest observable adverse-effects level (LOAEL) of 44 mg/kg/day in a 90-day study of the immunological effects of 1,1,2-TCA in mice. Affected functions, which differed between males and females, included humoral immune function, reticuloendothelial fixed macrophage activity, macrophage phagocytosis, and spleen lymphocyte response.

Exposure to 1,1,2-TCA has been observed to cause neurological effects, including anesthesia and central nervous system depression, in mice, rats, and dogs. Borzelleca (1983) reported motor impairment in mice at approximately a third of the LD_{50}. Other studies observed depression of reflexes, taste aversion, and seizures as a result of inhaling the chemical (ATSDR, 1988).

Teratogenic and Other Developmental Effects

There is no available evidence of teratogenic or other developmental effects related to exposure to 1,1,2-TCA.

Mutagenic Effects

There is no evidence of mutagenic effects in humans as a result of exposure to 1,1,2-TCA.

A series of assays have been conducted to study the genotoxic effects of 1,1,2-TCA. Positive results were obtained in an *in vitro* mutagenic assay in *Saccharomyces cervisiae* and a test of DNA repair in cultured rat hepatocytes (Williams, 1983), but a DNA repair test in mouse hepatocytes was negative. *In vivo* DNA adduct formation occurred to a greater degree in mouse liver than rat liver (Mazzullo et al., 1986). Other studies obtained negative results; but it appears that 1,1,2-TCA has some genetic effects in at least some mammalian species (ATSDR, 1988).

Carcinogenic Effects

There is no evidence of carcinogenic effects in humans as a result of exposure to 1,1,2-TCA.

1,1,2-TCA has been shown to cause hepatocellular carcinomas and adrenal pheochromocytomas in mice (NCI, 1978). However, the same conditions produced no evidence of increased neoplasms in rats. Other studies have also found no carcinogenic effects of exposure to 1,1,2-TCA in rats.

Ecotoxicity

1,1,2-TCA has not been reported in food, plants, or animals. Due to its low bioconcentration factor (< 10), 1,1,2-TCA is not anticipated to accumulate in the food chain (ATSDR, 1988). Acute LC_{50} values for freshwater aquatic organisms have been reported ranging from 18,000 to 81,700 ug/l (Clement Associates, 1985).

Standards, Criteria, and Guidance

1,1,2-TCA is classified as an EPA Class C Carcinogen (possible carcinogen) based on a study indicating increased incidence of hepatocellular carcinomas and adrenal pheochromocytomas in mice (IRIS, 1992).

Oral Slope Factor:	5.70×10^{-2} mg/kg/day^{-1}
Chronic Oral RfD:	4.00×10^{-3} mg/kg/day^{-1}
Inhalation Slope Factor:	5.70×10^{-2}
ACGIH TWA in air:	10 ppm (55 mg/m^3)
ACGIH IDLH:	500 ppm
Aquatic Life Protection:	18,000 ug/l (freshwater acute) 9,400 ug/l (freshwater chronic)
EPA AWQC:	0.6 ug/l

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2,4,6-TRICHLOROPHENOL

Use

2,4,6-Trichlorophenol (2,4,6-TCP) belongs to the class of chlorinated phenols. It is used as a germicide, bacteriacide, adhesive, wood preservative, antimildew agent, and as a chemical intermediate in the production of other chlorinated phenols.

Chemical and Physical Properties

MF: $HOC_6H_2Cl_3$
MW: 197.5
PD: Solid crystals; phenolic odor
MP: 68-69°C
BP: 246°C
FP: 61°C
VP: 1 mm Hg at 76.5°C
Sol. in water: 0.09 mg/l at 25°C
Sol. in organics: Alcohol, ether, acetone, benzene, methanol, toluene

Fate and Transport

Due to its low vapor pressure, 2,4,6-TCP is unlikely to volatilize from surface water. In the presence of an electron acceptor, photooxidation is the most likely fate process for 2,4,6-TCP, resulting in the formation of 2,6-dichlorobenzoquinone and 2,6-dichlorohydroquinone. 2,4,6-TCP in soil undergoes microbial degradation within 1 to 9 days (Clement Associates, 1985).

Pharmacokinetics

2,4,6-TCP is absorbed in the gastroenteric tract or from parenteral sites of injection. Target organs include the liver, kidney, lungs, and central nervous system. 2,4,6-TCP appears to act in part by inhibiting oxidative phosphorylation (Merck, 1983).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

There is no evidence of mortality in humans resulting from exposure to 2,4,6-TCP.

Sixteen male and 16 female A/J mice were administered varying doses of 2,4,6-TCP

in tricaprylin, either intraperitoneally or by gavage, 3 times a week for 8 weeks. Mortality occurred within 24 weeks of the initiation of the dosage (Stoner et al., 1986).

Other studies have observed the following effects in response to exposure to 2,4,6-TCP: restlessness, weight loss, increased rate of respiration, motor weakness, tremors, convulsions, and coma (Sittig, 1991).

Teratogenic and Other Developmental Effects

There is no available evidence of teratogenic or other developmental effects related to exposure to 2,4,6-TCP.

Mutagenic Effects

There is no evidence of mutagenic effects in humans as a result of exposure to 2,4,6-TCP.

A series of assays have been conducted to study the genotoxic effects of 2,4,6-TCP. Positive results were obtained in a mutagenic assay in *Saccharomyces cervisiae*, though there was no effect on mitotic recombination (Fahrig et al., 1978). A mouse spot test also produced positive results for 2,4,6-TCP, though the effects were among the weakest of chemicals tested (Clement Associates, 1985). 2,4,6-TCP did not prove mutagenic for *Salmonella typhimrium* (Rasanen et al., 1977).

Carcinogenic Effects

There is no evidence of carcinogenic effects in humans as a result of exposure to 2,4,6-TCP.

In a National Cancer Institute assay (NCI, 1979), 50 male and 50 female F344 rats were fed 5000 or 10,000 ppm 2,4,6-TCP for 106 or 107 weeks, while 50 male and 50 female B6C3F1 mice were fed either the same levels or from 2500 to 20,000 ppm for 105 weeks. Female rats only exhibited increased incidence of lymphomas and leukemia at the following doses: 25/50 at 258 mg/kg/day and 29/50 at 544 mg/kg/day. Other female rats exhibited leukocytosis, monocytosis of peripheral blood, and hyperplasia of bone marrow. Increased incidence of hepatocellular carcinomas and adenomas were observed in both male and female mice in the study.

No skin tumors were observed as a result of dermal applications of 2,4,6-TCP.

Ecotoxicity

No evidence is available on the ecotoxicity of 2,4,6-TCP.

Standards, Criteria, and Guidance

2,4,6-TCP is classified as an EPA Class B2 Carcinogen (probable human carcinogen) based on a study indicating increased incidence of lymphomas and leukemia in female rats and hepatocellular carcinomas and adenomas in male and female mice (IRIS, 1992).

Oral Slope Factor: 1.10×10^{-2} mg/kg/day^{-1}

Aquatic Life Protection: 970 ug/l (freshwater chronic)

EPA AWQC: 12.0 ug/l for excess cancer risk of 10^{-5}

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1,1,2-TRICHLORO-1,2,2-TRIFLUOROETHANE

Use

1,1,2-Trichloro-1,2,2-trifluoroethane, also known as Freon-113 or TTE, is used as a solvent and refrigerant; in fire extinguishers; as a blowing agent; and as an intermediate in the production of chlorotrifluoroethylene monomer by reaction with zinc (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: $C_2Cl_3F_3$

MW: 187.38	MP: -35°C
Dens.: 1,565 g/ml at 25°C	BP: 46°C
VP: 270 mm Hg at 20°C	VD: 6.47
Sol. (water): 170 mg/l at 25°C	

Fate and Transport

The U.S. EPA (1990) reports that TTE's very high vapor pressure, low water solubility, and chemical inertness are the key factors affecting its fate in the environment. In aquatic media, the U.S. EPA (1990) reports that volatilization will be very rapid, with the rate of volatilization being limited by the ability of TTE to diffuse through water. Thomas (1982) reports that the half-life of TTE in a model river 1 m deep, flowing at 1 m/sec, with a wind speed of 3 m/sec is approximately 4.0 hours. The U.S. EPA (1990) reports that TTE's moderate soil sorption coefficient (K_{oc}) indicates that adsorption to sediment and particulate matter in the water column would not be a significant fate process. This moderate K_{oc}, in addition to TTE's high vapor pressure and Henry's Law constant, suggests that TTE will volatilize rapidly from both dry and moist soil (U.S. EPA, 1990). Swann et al. (1983) reports that TTE's K_{oc} indicates a moderate potential for leaching into ground water.

Howard et al. (1975) reports that fluorocarbons are highly resistant to attack by oxidizing agents under environmental conditions. Hubrich and Stahl (1980) report that direct photolysis of TTE should not be significant given its lack of any UV absorption above 290 nm. TTE's rapid volatilization would limit, if not preclude, biodegradation (Howard et al., 1975). The U.S. EPA (1990) reports that TTE should have a low potential for bioconcentration in aquatic organisms.

TTE will partition into air due to its high volatility (U.S. EPA, 1990). Borchers et al. (1987) report that TTE is extremely stable in the troposphere, it will not directly photolyze or react with photochemically produced hydroxyl radicals. Borchers et al. (1987) report that TTE will disperse over the globe and diffuse slowly into the stratosphere, where it will be destroyed by photolysis by short wavelength UV radiation, and to a lesser extent by $O(^1D)$ attack. Following a seven-year study of seven atmospheric locations around the globe, Khalil and

Rasmussen (1988) report an atmospheric half-life of 48.5 years. The U.S. EPA (1990) reports that TTE released to the atmosphere will accumulate and become uniformly distributed over the world. TTE will be removed from the atmosphere by dry and wet deposition and will return via volatilization (U.S. EPA, 1990).

Pharmacokinetics

Absorption of TTE via the pulmonary system in humans was studied by Morgan et al. (1972). Volunteers were administered vapors of ^{33}Cl-labeled TTE in single breaths. The change in concentration of radioactivity in alveolar air with breath-holding time and the elimination of radioactivity in breath during normal breathing over 30 minutes were measured. After 40 seconds of breath-holding, approximately 70-80 percent of the initial TTE concentration remained in the alveolar air. After only 10-20 seconds of breath-holding, only 10 percent of the initial TTE concentration remained. 80.2 percent of the TTE inhaled in a single breath (and held for five seconds) was exhaled after 30 minutes in normal breathing experiments. This indicates that 19.8 percent of the inhaled TTE was retained in the body. In addition, almost 50 percent of the inhaled TTE was exhaled within the first minute (Morgan et al., 1972). Unpublished data on dermal absorption of TTE from the Haskell Laboratory (1968) was reviewed by the U.S. EPA (1983). Liquid TTE applied to the scalp (15 minutes) or the hands and forearms (30 minutes) was detected in expired air thus indicating dermal absorption. No quantitative estimates were available. No data regarding oral absorption of TTE were available in the literature reviewed.

Following inhalation in dogs and rats, absorbed TTE is rapidly distributed by the blood to various organs and tissues including the brain, liver, adrenal, heart, and thyroid; and is preferentially deposited in fat (Trochimowicz et al., 1974; Carter et al., 1970; Salvolainen and Pfaffli, 1980). A study with rats by Vainio et al. (1980) indicates that TTE may be oxidized by enzymes associated with P-450; specific metabolites, however, have not been identified.

Carter et al. (1970) reports that TTE is rapidly cleared from the body. Following a 14-day exposure regime, TTE could not be detected in the above-mentioned tissues of rats 24 and 48 hours after termination of exposure. In fat, TTE levels were reduced by 80-90 percent during the same time. The absorption studies conducted by Morgan et al. (1972) and Haskell Laboratories (1968) indicate that pulmonary exhalation is a significant excretory route in humans.

Human Toxicity

Non-Carcinogenic Effects

Systemic Effects

The U.S. EPA (1990) reports that adverse systemic effects in animals caused by subchronic or chronic exposure to vapors of TTE at concentrations ≤2,000 ppm have not been identified. Trochimowicz et al. (1988) report that adverse body weight changes were observed in rats exposed to concentrations ≥10,000 ppm. No adverse effects were reported for dogs, guinea pigs and rats exposed to TTE vapors of 5100 ppm for approximately four weeks (Steinberg et al., 1969).

In humans, no adverse effects were noted in a comparison by physical examination of chronically exposed workers to unexposed workers. Exposed workers worked an average of 2.8 years in rooms in which average concentrations of TTE were estimated to be 699 ppm. However, studies by Rasmussen and Sabroe (1986), Rasmussen et al. (1988), and Raffi and Violante (1981) suggest that occupational exposure to TTE vapors may cause neuropsychological effects and/or neuropathy.

Stopps and McLaughlin (1967) report that acute exposure (≤2 hours) to concentrations ≥2500 ppm TTE altered the performance of volunteers in psychophysiological tests. Reinhardt et al. (1971), however, found that exposure of volunteers to ≤1000 ppm TTE, six hours/day, five days/week, for two weeks did not cause treatment-related changes in performance in psychophysiological tests or in physical examinations.

The U.S. EPA (1990) discusses several studies which report that accidental and voluntary human exposures to TTE vapors caused sudden death generally attributed to cardiac arrest. This hypothesis is supported by animal studies in which acute exposure to moderate to high TTE concentrations caused adverse cardiac effects (EPA, 1990).

No data regarding the systemic toxicity of TTE via the oral and dermal routes were available in the literature reviewed.

Teratogenic and Other Developmental Effects

No teratogenic effects were reported in rats exposed to air containing ≤25,000 ppm on days 6-15 of gestation (Ward, 1983).

Mutagenic Effects

TTE was not mutagenic in assays for dominant lethal mutations in mice (Epstein et al., 1972), nor in assays for reverse mutations in *Salmonella typhimurium* (Simmon et al., 1977; Longstaff, 1988; Mahurin and Bernstein, 1988).

Carcinogenic Effects

Trochimowicz et al. (1988) exposed rats to concentrations of 0, 2,000, 10,000, or 20,000 ppm for two years. No treatment related increases in tumor incidences were reported.

Ecotoxicity

No data regarding the ecotoxicity of TTE were available in the literature reviewed.

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity

Oral slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	3.0×10^{-1} mg/kg/day
Chronic Inhal. RfD:	7.72×10^{0} mg/kg/day
Subchronic Oral RfD:	3.0×10^{0} mg/kg/day
Subchronic Inhalation RfD:	7.72×10^{0} mg/kg/day
MCL:	NA
AWQC:	NA

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TRIMETHYLBENZENES

Use

Trimethylbenzenes (TMBs) are classified as alkylbenzenes. They are used as solvents and in the manufacture of dyes and perfumes. 1,2,4-TMB is used as the raw material for the manufacture of trimellitic anhydride. TMBs are found in the exhaust fumes of diesel engines (Sittig, 1991).

Physical and Chemical Properties

Chemical Formula: $C_6H_3(CH_3)_3$

MW:	120.19		BP:	1,3,5-TMB:	165°C
VP:	1,3,5-TMB:	1.5 mm Hg at 25°C		1,2,4-TMB:	169°C
	1,2,4-TMB:	2.03 mm Hg at 25°C		1,2,3-TMB:	176°C
	1,2,3-TMB:	2.5 mm Hg at 25°C			
Sol. (water):		1,3,5-TMB: 75 mg/l			
		1,2,4-TMB: 52-57 mg/l			
		1,2,3-TMB: 48-97 mg/l			

Fate and Transport

Wakeham et al. (1983) reports that volatilization and biodegradation may be important fate processes for TMBs in aquatic systems. Lyman et al. (1982) and U.S. EPA (1986) calculated the volatilization half-lives for TMBs in a body of water 1 m deep flowing at 1 m/sec with a wind speed of 3 m/sec to be approximately 3.5 hrs. U.S. EPA (1987) reports that bioaccumulation in aquatic organisms would be insignificant and that moderate adsorption to suspended solids and sediments may occur. Atmospherically, TMBs are expected to exist in the vapor phase (U.S. EPA, 1987). U.S. EPA (1987) reports that vaporization and biodegradation are expected to be important fate processes for TMBS in soils and that TMBs should be moderately mobile in soils.

Pharmacokinetics

U.S. EPA (1987) reports that absorption of TMBs following oral administration appears to be significant. Gerade (1959) reports that alkylbenzenes are "absorbed into the blood from the various portals of entry at a rate and to an extent that appears to be proportional to the water solubility of the alkylbenzenes." Mikulski and Wiglusz (1975) administered a single oral dose of 1.2 g/kg of 1,3,5-TMB, 1,2,4-TMB, or 1,2,3-TMB to male wistar rats. 93.7 percent of the 1,3,5-TMB, 62.6 percent of the 1,2,4-TMB, and 56.6 percent of the 1,2,3-TMB was excreted as metabolites in the urine over a period of 3 days. Similarly, Gerarde (1959) reports that, in general, alkylbenzenes are excreted as biotransformation products in the urine or unchanged from the lung.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

In a study of 27 people occupationally exposed to 10-60 ppm hydrocarbon vapor of "Fleet-x-DDV-99", a solvent containing 30 percent 1,3,5-TMB and 50 percent 1,2,4-TMB, for several years, Battig et al. (1957) reports that "a significant number" of people exhibited symptoms of nervousness, tension, anxiety, and asthmatic bronchitis. These individuals also evidenced tendencies toward hyperchromic anemia and blood coagulation. Gerarde (1960) suggested that the hematologic effects evidenced by individuals may have been the result of a small proportion of benzene present in the hydrocarbon vapor.

Rats exposed to 1000 mg/m^3 TMBs for 4 hours/day, 6 days/week for 6 months exhibited an inhibition in the phagocytic activity of leukocytes (Bernshtein, 1972). In a similar study, rats exposed to 300 mg/m^3 1,3,5-TMB, 6 hours/day, 6 days/week for 5 weeks exhibited "slight" alterations in differential white blood cell counts and elevated SGOT levels in male rats (Wiglusz et al, 1975a, b).

Teratogenic and Other Development Effects

No data regarding the teratogenicity of TMBs were available in the literature reviewed.

Mutagenic Effects

No data regarding the mutagenicity of TMBs were available in the literature reviewed.

Carcinogenic Effects

No data regarding the carcinogenicity of TMBs were available in the literature reviewed.

Ecotoxicity

No data regarding the ecotoxicity of TMBs were available in the literature reviewed.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slop Factor:	NA	
Inhalation Slope Factor:	NA	
Chronic Oral RfD:	1,2,4-TMB:	6×10^{-4} mg/kg/day
	1,3,5-TMB:	4×10^{-4} mg/kg/day
Chronic Inhalation RfD:	1,2,4-TMB:	5.71×10^{-4} mg/kg/day
	1,3,5-TMB:	5.71×10^{-4} mg/kg/day
Subchronic Oral RfD:	1,2,4-TMB:	6×10^{-4} mg/kg/day
	1,3,5-TMB:	4×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	1,2,4-TMB:	NA
	1,3,5-TMB:	NA
MCL:	NA	
AWQC:	NA	

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1,1,1-TRICHLOROETHANE

Use

1,1,1-trichloroethane is a colorless, nonflammable liquid with an odor similar to chloroform. It is sometimes referred to as methyl chloroform.

Relatively recently, it has become widely substituted for carbon tetrachloride. It is used as a degreaser, and for dip-cleaning, bucket cleaning, and cold-cleaning of metals. 1,1,1-trichloroethane's solvent properties compel its use as a dry-cleaning agent, a vapor-degreasing agent and as a propellant (Sittig, 1991).

Physical and Chemical Properties

Chemical Formula: CH_3CCl_3
M.W.: 133.41
Sol. (water): insoluble
Sol. (organics): alcohol, ether, chloroform

BP: 74°C
MP: -30.4°C
VP: 123 mm Hg at 25°C

Fate and Transport

The half-life of 1,1,1-trichloroethane in water was reported by Callahan et al. (1979) to be 20-25 minutes. Volatilization to the atmosphere is the most likely route of escape. Singh et al. (1981) and Makide and Rowland (1981) reported the half-life of 1,1,1-trichloroethane in air to be 2.2 - 4.8 years. This suggests that the compound may move up into the stratosphere where it could contribute to ozone depletion.

Evaporation is expected to be the major fate of 1,1,1-trichloroethane from surface soil (Bouwer et al., 1981). Tabak et al. (1981) concluded that biodegradation of 1,1,1-trichloroethane is a slow process in subsurface soils. Coupled with low water solubility and a relatively low octanol/water partition coefficient, this suggests that the compound will remain substantially undegraded in subsurface soils creating the potential for leaching into ground water. In fact, Page (1981) detected the presence of this compound in ground water at a frequency of 78 percent.

Pharmacokinetics

It was determined by Stewart (1971) that 1,1,1-trichloroethane is "rapidly and completely" absorbed from the GI tract of humans and distributed preferentially and rapidly to the CNS. The U.S. EPA investigated the possibility of using 1,1,1-trichloroethane as an anesthetic and considered it to be more potent than trichloroethylene and safer than chloroform (U.S. EPA 1984).

Pulmonary absorption of inhaled 1,1,1-trichloroethane is initially rapid, but then slows dramatically until equilibrium is reached (U.S. EPA, 1984) Monster et al. (1979) and Humbert and Fernandez (1977) exposed volunteers to 70 or 140 ppm of 1,1,1-trichloroethane for 4 and 8 hours. Equilibrium was reached in 4 hours and, at that time, Monster et al. (1979) reported a retention of 30 percent of the inhaled dose. This is 40 percent less than that reported by Humbert and Fernandez (1977). These data led the EPA to classify the compound as a poorly absorbed, partially soluble vapor (U.S. EPA, 1984).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Stewart et al. (1975) exposed 20 human subjects to 1,1,1-trichloroethane for 3 weeks, 5 days/week, 7.5 hours/day to a 500 pm concentration. The only effects detected were complaints of fatigue, irritation and headache by the subjects.

Similarly, Seki et al. (1975) found no dose-related effects in 196 male workers exposed to varying concentrations of 1,1,1-trichloroethane for greater than 5 years. Maroni et al. (1977), as well, found no signs of neurotoxicity when comparing 21 women exposed to 110-345 ppm 1,1,1-trichloroethane for 6.5 years to 7 unexposed control subjects.

Torkelson et al. (1958), however, detected statistically significant increased liver weights in female guinea pigs exposed to 1,1,1-trichloroethane vapor at a concentration of 1,000 ppm for 3 hours/day, 5 days/week for 3 months. Females exposed to 500 ppm showed no adverse effects after exposure for 7 hours/day, 5 days/week, for 6 months. Adams et al. (1950), as well, reported a slight depression in weight gain when guinea pigs were exposed to 1,1,1-trichloroethane at a concentration of 650 ppm for 7 hours/day, 5 days/week for 2 to 3 months.

Teratogenic and Other Developmental Effects

In studies by Leong et al. (1975) and Schwetz et al. (1975), "no remarkable malformations were observed" in the fetuses of mice or rats exposed to 1,1,1-trichloroethane. A similar lack of adverse effect was reported when Charles River albino rats were exposed to 300 ppm 1,1,1-trichloroethane (U.S. EPA, 1984).

Mutagenic Effects

Both positive and negative results have been reported in the literature. Farber (1977) and Nestmann et al. (1980) found 1,1,1-trichloroethane to be mutagenic in *S. typhimurium* strain TA1535 and Simmon et al. (1977) produced positive results in *S. typhimurium* strain TA100.

Farber (1977) and Simmon et al. (1977) found no gene conversion of mitotic recombination in *Saccharomyces cerevisiae* upon exposure to 1,1,1-trichloroethane and the chemical also failed to produce chromosomal aberrations in the bone marrow of cats (Rampy et al., 1977).

Carcinogenic Effects

Quast et al. (1978) exposed 96 Sprague-Dawley rats of both sexes to 875 of 1,750 ppm vapor concentrations of 1,1,1-trichloroethane for 6 hours/day, 5 days/week for 12 months, followed by a 19-month observation period. There were no signs of carcinogenicity other than a significant increased incidence of focal hepatocellular alterations in female rats at the highest dosage.

Neither Quost et al. (1978) nor NCI (1977) found significant dose-related incidences of neoplasms. In the NCI study, Osborne-Mendel rats and B6C3F1 hybrid mice were treated with 750 or 1,500 mg/kg and 2,807 or 5,615 mg/kg of 1,1,1-trichloroethane, respectively, five times/week for 78 weeks. Although a variety of neoplasms were observed in the treated animals, they were not dose-related, nor were they statistically different from the occurrence of neoplasms in untreated animals.

Ecotoxicity

The available data on the toxicity of chlorinated ethanes in freshwater systems indicate that acute toxicity for trichloroethanes occurs at concentrations as low as 18,000 µg/liter. chronic toxicity occurs at concentrations as low as 9,400 µg/liter for 1,1,2-trichloroethane, a similar chlorinated ethane. In saltwater systems, acute toxicity to fish and invertebrate species occurs at 31,200 µg/liter for 1,1,1-trichloroethane. Both acute and chronic toxicity values are expected to be lower for species more sensitive to chlorinated ethanes than those tested (U.S. EPA, 1986).

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	9×10^{-2} mg/kg/day
Chronic Inhalation RfD:	3.0×10^{-1} mg/kg/day
Subchronic Oral RfD:	9.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	3.0×10^{0} mg/kg/day
MCL:	0.2 mg/l
AWQC:	Water and Fish Consumption - 18.4 mg/l
	Fish Consumption - 1030 mg/l

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TRICHLOROETHYLENE

Use

Trichloroethylene (TCE) is a synthetic chlorinated hydrocarbon that is colorless, nonflammable, and noncorrosive. Its odor is similar to that of other chlorinated solvents used commercially (Sittig, 1991). TCE is mainly used as a metal degreaser but is also used to decaffeinate coffee, as a dry cleaning agent, and as an intermediate in the production of pesticides, paints and varnishes. TCE is moderately volatile and is used nationwide. As a result, TCE is present at a large number of hazardous waste sites. Approximately 3 percent of drinking water supplies derived from well water contain TCE at levels higher than 0.5 µg/L (EPA, 1985).

Chemical and Physical Properties

Chemical Formula: C_2HCl_3

MW: 131.5
SG: 1.464 at 20°C
FP: none
Sol. (water): 1000 mg/l
BP: 87°C
MP: -73°C
VP: 4.53 mmHg at 25°C
Sol. (organics): soluble in alcohol, ether, acetone and chloroform

Fate and Transport

The main avenues of TCE release to the environment are through the metal degreasing industry. The majority of the releases occur through volatilization, with a smaller percentage released as a result of accidental spills (EPA, 1985). Large quantities of spent TCE that were regularly landfilled are now reclaimed, eliminating that avenue of release.

TCE volatilizes from surface waters and soils and is rapidly degraded in the air. In moist soil and ground water, TCE is known to be stable, often remaining therein for a period of months to years (EPA, 1985). TCE usually degrades to either 1,2 dichloroethylene, or vinyl chloride and is a degradation product of tetrachloroethylene.

The major avenue of TCE contamination to humans is through the ground water. TCE does not bioaccumulate in animals or food chains (EPA, 1985).

Pharmacokinetics

When 200 mg/kg of ^{14}C-TCE in corn oil was administered to rats in their food, 97 percent of the dose was recovered during the 72 hours after dosing (EPA, 1985). Rats exposed to TCE by gavage at doses of 0, 10, 100 or 1,000 mg/kg/day, 5 days per week for six weeks, showed marginal increases in TCE tissue levels at the 10 mg/kg/day and 100 mg/kg/day dose groups.

Compared to controls, a marked increase in TCE levels in most tissues was observed in the highest dose group. TCE was distributed throughout all tissues examined with the highest concentrations in the fat, kidney, lung, adrenal, vas deferens, epididymis, brain and liver (EPA, 1985). Studies indicate that TCE is metabolized to trichloroethylene oxide, trichloroacetaldehye, trichloroacetic acid, monochloroacetic acid, trichloroethanol, and trichloroethanol glucuronide (EPA, 1985). Trichloroethylene and its metabolites are excreted in urine, by exhalation, and to a lesser degree in sweat, feces, and saliva (EPA, 1985).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The National Cancer Institute (NCI) exposed Osborne-Mendel rats and B6C3F1 mice to TCE by gavage. Doses ranged between 300 and 550 mg/kg/day for mice and between 471 and 949 mg/kg/day for rats. Toxic nephropathy was observed at all dose levels.

Oral exposure of humans to 15 to 25 ml TCE resulted in vomiting and abdominal pain followed by transient unconsciousness (EPA, 1985). In a study done in 1971 by Lachnit, humans showed symptoms indicating damage to the liver parenchyma.

Teratogenic and Other Developmental Effects

The U.S. EPA reports a high rate of miscarriages among women exposed to TCE in the workplace, as noted in one case study. Swiss-Webster mice and Sprague-Dawley rats exposed to TCE vapors at a concentration of 300 ppm for 7 hours per day on days 6-15 of gestation showed no treatment-related increases in malformations. However, slightly reduced fetal body weight, delayed skeletal development, and an increase in the incidence of undescended testes were observed in mice (EPA, 1988). The offspring of pregnant rabbits exposed to TCE vapors at 500 ppm for 7 hours per day, 5 days per week beginning three weeks prior to mating on days 0-21 of gestation, or on days 6-21 of gestation, were all reported to have an increased incidence of external hydrocephalus (EPA, 1984).

Mutagenic Effects

TCE is mutagenic in *Salmonella typhimurium* and in the *E. coli* K-12 strain when liver microsomes were used for activation (EPA, 1985).

Carcinogenic Effects

TCE has been shown to be carcinogenic in different strains of mice via inhalation as well as oral exposure. The National Cancer Institute (1976) and the National Toxicology Program (1982) conducted two separate studies with TCE contaminated with epichlorohydrin and with TCE free of epichlorohydrin. In these studies, B6C3F1 mice displayed a significant increase in liver neoplasms. Technical TCE (with epichlorohydrin and other compounds) was found to induce a hepatocellular carcinogenic response in mice. In this study, "time-weighted" average doses of 1,169 and 2,339 mg/kg for males and 869 and 1,783 mg/kg for females were administered (EPA, 1985).

Only one human study is available that shows a causative effect between TCE and human cancers. Workers were exposed to tetrachloroethylene and carbon tetrachloride in conjunction with the TCE. All other studies were inconclusive (EPA, 1984).

Ecotoxicity

Fathead minnows (*Pimephales promelas)* exposed to TCE in flow-through tests with measured concentrations and in static tests without measured exposure concentrations yielded LC_{50} (median lethal concentrations) values of 40,700 and 66,800 µg/liter, respectively. Also examined in static tests for 96 hours was the bluegill (*Lepomis macrochirus*), with an LC_{50} value of 44,700 µg/liter (EPA, 1980). The 48-hour EC_{50} (median effective concentration) value for *Daphnia magna* and TCE is 85,200 µg/L. Comparisons made among three laboratories show the 50 percent effect concentrations for *Daphnia magna* ranged from 41,000 to 100,000 µg/liter. At one laboratory, *Daphnia pulex* was also tested to determine any sensitivity, and the results were 39,000 and 51,000 µg/liter indicating no difference in sensitivity between species (EPA, 1980). TCE is practically nontoxic for freshwater aquatic organisms under these acute exposure conditions.

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	1.1×10^{-2} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	1.7×10^{-2} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	6.0×10^{-3} mg/kg/day
Chronic Inhalation RFD:	currently under review by EPA
Subchronic Oral RfD:	7.0×10^{-3} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.005 mg/l
AWQC:	Water and Fish Consumption - 2.7 µg/l
	Fish Consumption - 80.7 µg/l

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TRICHLOROFLUOROMETHANE

Use

Trichlorofluoromethane, commonly known as Freon, is used as a refrigerant, aerosol propellant and foaming agent (Sittig, 1991). It is also used in the extraction of oil and grease from waters and soils for analytical purposes.

Chemical and Physical Properties

Chemical Formula: CCl_3F	BP: 23.82°C
MW: 137.37	MP: -111°C
Sol. (water): 1,100 mg/liter	VP: 667.4 mmHg at 20°C

Sol. (organics): soluble in alcohol, ether and other organic solvents

Fate and Transport

The low solubility, high vapor pressure, and low boiling point of trichlorofluoromethane make volatilization the likely transport process for removal of the compound from aqueous systems (ICF, 1985). Once in the troposphere, trichlorofluoromethane remains stable and is eventually transported to the stratosphere or is carried back to earth by precipitation (ICF, 1985).

Trichlorofluoromethane that reaches the stratosphere is broken down by high energy, short wavelength ultraviolet light, producing chlorine atoms which are theorized to serve as a catalyst in destruction of the stratospheric ozone layer (ICF, 1985). This pathway is considered the major environmental fate of trichlorofluoromethane (ICF, 1985).

Absorption of trichlorofluoromethane onto sediments may occur, as suggested by a log octanol/water partition coefficient of 2.53 (ICF, 1985).

Pharmacokinetics

In a human exposure study by Stewart et al. (1975) the rate of excretion of trichlorofluoromethane in the expired air was found to be a function of the duration of exposure. These was no significant accumulation of trichlorofluoromethane in the body following 8-hour exposures to 1000 ppm, repeated every 24 hours, however.

Using radioactive tracer techniques, Morgan et al. (1972) concluded that, as a group, fluorocarbons have low lipid solubility compared to aliphatic chlorinated hydrocarbons. Much of inhaled chlorine-38-labeled fluorocarbon vapors were exhaled indicating poor absorption in the lung (Clayton and Clayton, 1981). After 30 minutes, only 23 percent of the total unexpired trichlorofluoromethane was retained in the lung. Given that only a small fraction of the retained material was found in the lung after 5 minutes, it appears that the fluorocarbon remained in the lung tissue (Clayton and Clayton, 1981).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The National Cancer Institute (NCI, 1978) performed a bioassay on rats and mice exposed to various doses of trichlorofluoromethane by gavage for a period of 78 weeks. The Tarone test revealed a significant positive association between increased dosage and accelerated mortality in male and female rats and female mice. Rats of both sexes illustrated treatment-related elevated incidences of pleuritis and pericarditis.

Several studies with guinea pigs have revealed toxicity. Scholz (1962) reported that a 1 hour exposure to a 20 percent concentration of trichlorofluoromethane was lethal and Caujolle (1964) found that inhalation of a 25 percent concentration for 30 minutes was lethal to half the guinea pigs tested. Nuckolls (1959) found that a 1 hour exposure to a 10 percent concentration resulted in coma with a lower concentration (2.5 percent for 30 minutes) producing irregular breathing, occasional tremors, and bruxus.

Teratogenic and Other Developmental Effects

Paulet et al. (1974) exposed rats and rabbits during gestation to a 20 percent concentration of a propellant mixture of 10 percent trichlorofluoromethane and 90 percent fluorocarbon 12. The rats were exposed during days 4-16, the rabbits during days 5-20, for 2 hours/day. No adverse affects on the offspring of the exposed pregnant animals was reported.

Mutagenic Effects

Pertinent data regarding the mutagenicity of trichlorofluoromethane was not found in the literature reviewed.

Carcinogenic Effects

The NCI (1978) bioassay on rats and mice found no signs of carcinogenicity in either sex of either species at any dose level.

Other relevant data regarding the carcinogenicity of trichlorofluoromethane was not found in the literature reviewed. The US EPA has not evaluated trichlorofluoromethane for human carcinogenic potential.

Ecotoxicity

Pertinent data regarding the toxicity of trichlorofluoromethane to domestic and wildlife, both terrestrial and aquatic, was not found in the literature reviewed. However, trichlorofluoromethane in suspected of being the major contributor to the depletion of the stratospheric ozone layer (ICF, 1985). This layer filters out ultraviolet rays from the sun which are known to be harmful to biota. Therefore, trichlorofluoromethane could potentially have adverse effects for all forms of life by eliminating the global protection system against the harmful ultraviolet rays.

Standards, Criteria and Guidelines

Unclassified as to Carcinogenicity

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	3×10^{-1} mg/kg/day
Chronic Inhalation RfD:	2×10^{-1} mg/kg/day
Subchronic Oral RfD:	7×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	2.0×10^{0} mg/kg/day
MCL:	NA
AWQC:	Water and Fish Consumption - 0.19 µg/l
	Fish Consumption - 15.7 µg/l

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VINYL CHLORIDE

Use

Vinyl chloride is used in the manufacture of polyvinyl chloride (PVC), rubber, glass, electrical wire, and automotive parts (EPA, 1985). Generally used in its gaseous state, vinyl chloride and PVC copolymers are distributed and processed as dry resins, plastisol and latex (EPA, 1985). Environmental releases of vinyl chloride generally occur where it is produced because the industrial processes in which vinyl chloride is used are wastefull (EPA, 1985).

Chemical and Physical Properties

Chemical Formula:	CH_2CHCl		
MW: 62.5		MP:	-13.37°C
SG: 0.9106 at 20°C		BP:	-153.8°C
Sol.(Water):	1,100 mg/l at 25°C	VP:	2,660 mmHg at 25°C
Sol.(Organics):	Alcohol, ether, carbon tetrachloride		

Fate and Transport

When released to surface waters, vinyl chloride volatilizes to the atmosphere within a few hours where it chemically degrades. In the atmosphere, vinyl chloride degrades within one or two days of its release. Degradation products include hydrogen chloride, formyl chloride and carbon monoxide (ICF, 1985). When released to the ground, vinyl chloride does not adsorb to soils and leaches readily to the ground water.

In the ground water, vinyl chloride may degrade to carbon dioxide and the chloride ion (EPA, 1985). Ground water is considered to be the major source of human exposure to vinyl chloride (EPA, 1985).

Pharmacokinetics

Vinyl chloride is absorbed rapidly in rats exposed via inhalation and ingestion. The greatest concentration of absorbed vinyl chloride was found in the liver, kidneys, muscle, lungs, fat, spleen, and brain of rats following exposure via inhalation and ingestion (EPA, 1985). Hefner et al. (1975) noted that vinyl chloride is metabolized primarily by alcohol dehydrogenase. After absorption into the body, vinyl chloride and its metabolites are excreted in the urine.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Vinyl chloride is known to be hepatotoxic to workers exposed in the PVC manufacturing business (ACGIH 1984). One study reported that acute exposure to high levels of vinyl chloride causes central nervous system dysfunction such as euphoria, dizziness, and incoordination (ACGIH, 1984). Chronic exposure to high concentrations of vinyl chloride is known to cause bronchitis, headache, irritability, and severe systemic disorders such as sclerotic syndrome, acro-osteolysis, thrombocytopenia, and liver damage (ICF, 1985). Liver damage appeared to be the most abundant systemic effect of chronic exposure in laboratory animals. In another study, researchers exposed rats to 30,000 ppm vinyl chloride for 4 hours/day, 5 days/week in an attempt to induce acro-osteolysis, a condition described as a combination of thrombopenia, liver damage, circulatory obstruction, and bone alterations. Metaplastic bone changes, similar to those noted in cases of acro-osteolysis in humans, were noted in this study (ACGIH, 1984).

Teratogenic and Developmental Effects

Minor skeletal abnormalities and an increased fetal death rate was noted in experimental animals exposed to vinyl chloride via inhalation (ICF, 1985). In humans, a significant increase in fetal deaths was noted in women whose husbands were occupationally exposed to vinyl chloride (ICF, 1985).

Mutagenic Effects

Vinyl chloride appears to be mutagenic to bacteria and fruit flies (EPA, 1985). Abundant chromosomal aberrations were noted in occupationally exposed workers (ICF, 1985).

Carcinogenic Effects

Vinyl chloride is classified as a known human and animal carcinogen (Class A) by the International Agency for Research and Cancer (IARC). IARC found that chronic, occupational exposure to vinyl chloride causes an increase in the number of liver angiosarcomas, brain tumors, lung tumors, hemopoietic tumors, and lymphopoietic tumors (EPA, 1985).

Feron et al. (1981) administered, via ingestion, 1.7 mg vinyl chloride/kg bw/day, to rats over their lifespan. The treatment induced an increase in angiosarcomas,

hepatocellular carcinomas, and adverse hepatic effects. Maltoni (1981) noted that chronic inhalation of vinyl chloride by rats and mice induced liver cancer and tumors in various other bodily tissues.

Ecotoxicity

Pertinent information regarding the ecotoxic effects of vinyl chloride were not located in the available literature although, it can be inferred from the effects on laboratory animals, that vinyl chloride is highly toxic to most organisms.

Standards, Criteria and Guidelines

EPA Class A Carcinogen

Oral Slope Factor:	1.9×10^{0} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	2.94×10^{-1} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	0.002 mg/l
AWQC:	Water and Fish Consumption - 2 µg/l
	Fish Consumption - 525 µg/l

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XYLENES

Use

Xylenes refer to a mixture of orth-, meta-, and para-xylenes. Xylenes are components of petroleum and gasoline products; are used in iron and steel manufacturing, foundries, pulp and paper mills; and are used as solvents for paints, inks, and adhesives (ICF, 1985; EPA, 1987). Xylenes occur naturally as a constituent of petroleum oil, and are produced in large volumes during gasoline refinement. The release of xylenes to the environment occurs mostly to air as a result of volatilization. Releases of xylenes to water and soil are primarily attributed to spills and leaks of gasoline and other petroleum products, with lesser releases due to disposal of waste paints, inks, and other industrial products (EPA, 1987).

Chemical and Physical Properties

Chemical Formula: $C_6H_4(CH_3)_2$

MW: 106.17
SG: 0.860 at 25°C
FP: 25°C
Sol. (water): 160 mg/l at 25°C
Sol. (organics): alcohol, ether, and numerous other organic solvents

BP: 137 - 140°C (mixed)
MP: meta - 48°C
ortho - 25°C
para - 13°C

VP: 10 mmHg at 25°C

Fate and Transport

Volatilization is the most important means by which xylene is removed from soils and surface water. Xylene is adsorbed by organics in moist soils and transport to ground water is unlikely (EPA, 1985).

In the atmosphere, xylene is photohydroxylated to produce carbon dioxide, cresol, and peroxyacetylnitrate.

Pharmacokinetics

Inhalation of mixed xylenes by humans showed that xylenes are absorbed readily to an extent of 64 percent. Animal studies indicate that xylenes are rapidly distributed to the brain and adipose tissue and reach maximum tissue levels within one hour after inhalation; xylenes are also distributed to the kidneys, subcutaneous fat, sciatic nerve, blood, liver, lungs, spleen, and muscles. Metabolism of xylenes is generally accomplished by oxidation of methyl groups and ring hydroxylation, thus producing methyl hippuric acid (95 percent) and xylenols (1-2 percent) as metabolites (EPA, 1985).

Synergistic effects result from metabolic interactions when xylenes are coadministered with other chemicals. Ethanol potentiates the effect of xylenes by delaying its metabolism and elevating blood xylene levels (EPA, 1985). Depressed metabolism was also observed when xylene were coadministered with any of the following compounds: 1,1,1-trichloroethane (EPA, 1984), benzene, ethylbenzene, or toluene (EPA, 1985). Xylenes induce enzyme activity and thus potentiate the hepatoxicity of carbon tetrachloride by elevating the levels of toxic metabolites (EPA, 1985).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The lowest oral dose to cause death in humans was reported at 50 mg/kg. Xylenes produce central nervous system disturbances in humans that include alterations in numerative ability, short-term memory, and electroencephalographic patterns. No adverse effects were observed in males following inhalation of xylene for 70 minutes at concentrations of 435 and 1,300 mg/m^3. However, inhalation of xylene at 1,300 mg/m^3 after 30 minutes of strenuous exercise caused decreased mental abilities. Psychophysiological functions were impaired in humans following exposure to xylene at 391 mg/m^3 for five consecutive days. The effects included depression of manual coordination and impairment of body balance (EPA, 1985).

NTP (1986) studies of rats and mice administered gavage doses of 0, 250, or 500 mg/kg/day and 0, 500, or 1000 mg/kg/day for 5 days/week for 103 weeks revealed the following: increased dose-related mortality observed in rats; and hyperactivity in mice given the high dose.

Chronic inhalation of 770, 2,200 and 3,500 mg/m^3 of mixed xylenes by rodents for six days/week or five days/weeks for 13 weeks did not produce any effects at the lower two doses. The higher dose produced renal tubular degeneration. Inhalation of 337 and 3,358 mg/m^3 of o-xylene for thirty and ninety days continuously by rats, guinea pigs, monkeys, and dogs did not produce significant effects with respect to body weight, hematology, and histopathology. The only observed effect was tremors produced in dogs. Oral exposure to o-xylene at 200 mg/kg diet for six months produced hepatotoxicity in rats. Inhalation of 4,750 mg/m^3 of xylene for eight hours/day, seven days/week for one year produced hepatotoxicity in rats (EPA, 1985).

Teratogenic and Other Developmental Effects

Increased incidence of fused sternebrae and extra ribs were observed in rats inhaling 1,000 mg/m^3 of mixed xylenes for 24 hours/day during days 9 to 14 of pregnancy. No maternotoxic effects were observed in the rats. In a study which exposed pregnant rats to 0, 434, and 1,730 mg/m^3 of xylenes during days 6 to 15 of pregnancy, no teratogenic effects were observed (EPA, 1985). Maternotoxicity appears to occur in mice exposed to xylenes (EPA, 1985).

Mutagenic Effects

Short-term *in vitro* assays and the Ames test indicate that xylenes are not mutagenic (EPA, 1985).

Carcinogenic Effects

Xylene is designated by EPA as a Group D -- not classified Weight-of-Evidence category for potential carcinogens (EPA, 1985). Several studies including one by NTP (1986) report no increase in the incidence of cancer in laboratory animals exposed to xylenes.

Ecotoxicity

Xylenes adversely affect trout at concentrations as low as 3.6 mg/L and have a LC_{50} value of 13.5 mg/L. LC_{50} values for other fresh water fish average 13.5 mg/L. Information regarding the toxicity of xylenes to terrestrial animals was not available (EPA, 1980).

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2.0 x 10^0 mg/kg/day (mixed xylenes)
Chronic Inhalation RfD:	8.57 x 10^{-2} mg/kg/day (mixed xylenes)
Subchronic Oral RfD:	4.0 x 10^0 mg/kg/day
Subchronic Inhalation RfD:	8.57 x 10^{-2} mg/kg/day
MCL:	10 mg/l
AWQC:	NA

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BASE-NEUTRAL/ACID EXTRACTABLE (SEMI-VOLATILES)

BENZOIC ACID

Use

Benzoic acid is used as a food preservative, in pharmaceutical and cosmetics preparation, in the manufacture of resins and as an intermediate in the synthesis of other industrial chemicals (Verscheuren, 1977).

Chemical and Physical Properties

Chemical Formula: C_6H_5COOH

MW: 122.1	MP: 121.7°C
SG: 1.27	BP: 249°C
Sol. (water): 2,900 mg/L	VD: 4.21

Fate and Transport

Benzoic acid is known to biodegrade in soils within 1 day of release (Verscheuren, 1977). No other information regarding the fate and transport of benzoic acid was located in the available literature.

Pharmacokinetics

Benzoic acid is absorbed rapidly in the stomach and gastrointestinal tracts of humans (IRIS). No other information regarding the fate and transport of benzoic acid was located in the available literature.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The Food and Drug Administration (FDA) (1983) reports that daily per capita intakes of 0.9-34 mg of benzoic acid have resulted in no toxic effects on humans. The FDA has given benzoic acid the status of Generally Recognized As Safe (GRAS).

Chronic oral doses of 40 mg benzoic acid/kg/day administered to mice and rats for 17 months resulted in a decreased resistance to stress and a possible reduction in food and water intake (Shtenberg and Ignat'ev, 1970). Another study resulted in the same effects in rats exposed orally to 750 mg benzoic acid/kg/day for a lifetime (IRIS).

Levels between 0.9 and 34 mg benzoic acid do not appear to induce toxic effects in humans when exposed via ingestion (IRIS).

Gerlach (1909) reported no visible ill effects in humans ingesting benzoic acid at 0.5 - 1.0 g/day for 44 days or 82 of 86 days. Wiley and Bigelow (1908), however, noted irritation and discomfort in humans given oral doses less than 1.75 g/day.

Teratogenic and Other Developmental Effects

No information regarding the teratogenic or developmental effects of benzoic acid were located in the available material.

Mutagenic Effects

Benzoic acid has been tested for mutagenicity and genotoxicity in prokaryotes, eukaryotes and mammalian cell cultures. Reports have all been negative (IRIS).

Carcinogenic Effects

Toth (1984) administered 3502 and 3367 mg benzoic acid/kg/day to male and female Swiss mice, respectively. After a lifetime, examinations revealed no toxic or carcinogenic effects.

Shtenberg and Ignat'ev (1970) reported an 8 percent incidence of malignant tumors in mice exposed to 40 mg benzoic acid/kg/day for 17 months. Tumor incidences of the controls were not reported. In a separate study, Dinerman and Ignat'ev (1966) reported that mice exposed to 0.2 percent benzoic acid for 3 months displayed increased susceptibility to carcinomas induced by inoculations with Erlich ascites carcinoma cells. The incidence of tumors more than doubled when benzoic acid was co-administered.

Ecotoxicity

Benzoic acids LD_{50} for rats was found to be 1.7 g/kg. *Daphnia magna* are immobilized by 146 mg benzoic acid/L water when exposed for a prolonged period of time (Verscheuren, 1977).

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	4 x 10^0 mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	4 x 10^0 mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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BIS(2-ETHYLHEXYL)PHTHALATE

Use

The phthalate esters, such as bis(2-ethylhexyl)phthalate (BEHP), are widely used in PVC resins and vinyl copolymer resins to impart flexibility to the finished product. Other reported uses include as an inert ingredient in pesticides, a component in dielectric fluids (replacing PCBs) in electric capacitors, a solvent for erasable ink, acarid in orchids, in vacuum pump oils, and as a testing agent for air filtration systems. Consumer products using BEHP include vinyl upholstery, table cloths, shower curtains, raincoats, and food wrap. Annual consumption of BEHP is approximately 130 million kg.

Chemical and Physical Properties

Chemical Formula: C_6H_{40} ($COOCH_2CH$ (C_2H_5)$C_4Hg)_2$

MW:	3,190	BP:	386.9 C at 5 mmHg
SG:	0.985 at 20 C	MP:	-50 C
FP:	218.33 C	VP:	2 x 10^{-7} mmHg at 20 C

Sol. (water): 0.4 mg/L at 25 C
Sol. (organics): mineral oil and hexane

Fate and Transport

In aquatic media, BEHP does not volatilize or photo-oxidize readily. Apparently, adsorption to suspended solid and particular matter are probably the most important of BEHPs fate processes (ICF, 1985). Bioaccumulation is another important fate process for BEHP. Several unicellular and multicellular aquatic organisms are known to accumulate BEHP (ICF, 1985).

In soils, BEHP would be expected to sorb to organic matter. Very little volatilization and leaching would be expected (ICF, 1985).

Pharmacokinetics

Studies indicate that, following an oral dose, BEHP is initially hydrorolyzed by a nonspecific lipase in the gastrointestinal tract to produce mono(ethylhexyl)phthalate (MEHP) (and 2-ethylhexanol) which is readily absorbed from the gastrointestinal tract. One study indicated that BEHP is poorly absorbed following dermal application. In acute inhalation toxicity studies in rats, it has been demonstrated that BEHP is absorbed by the lung. Information on the oral absorption of BEHP in humans is limited, and data is not available on the absorption of BEHP by humans exposed via inhalation or through dermal exposure (ATSDR, 1989).

Absorbed BEHP and is metabolites are distributed rapidly to tissues and organs with only a slight cumulative potential. The liver appears to be the major, initial repository organ. BEHP is eliminated from the body mainly through urinary excretion. Urinary metabolites appear to differ amongst species (ATSDR, 1989).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Various rodent studies revealed LD_{50}s ranging from 26,000 to 49,000 mg/kg following oral exposure. No data are available on the effects of oral, ingestion, or dermal exposure to BEHP on lethality in humans (ATSDR, 1989).

The liver and the testes have been shown to be the primary target organs of BEHP. Morphological and biochemical changes in the liver of exposed rodents have been observed following exposure to high doses of BEHP. No data are available on the hepatic toxicity of BEHP in humans via inhalation, oral, or dermal exposure. Testicular effects, including a decrease in relative organ weight and histological changes in the seminiferous tubules have been observed in the rat and mouse, but not in the hamster, ferret, or marmoset following exposure to BEHP and MEHP (ATSDR, 1989).

Teratogenic and Other Developmental Effects

BEHP is a reproductive toxicant in male and female mice; reduced fertility and both production of fewer litters by breeding pairs and decreased litter size has been observed (ATSDR, 1989). Available data suggests that BEHP is developmentally toxic in rats and mice. One study indicated that, following administration of 0.05, 0.1, 1.0, 2.5, 5.0, or 10.0 Ml/kg BEHP by gavage on day 7 of gestation, a decrease in body weight of live fetuses occurred at the 0.05 mL/kg dose. At doses administered at or above 0.1, mL/kg, a decrease in fetal body weight was observed, and the fetuses were deformed or dead. In a study in which pregnant Fisher 344 rats were exposed to BEHP in their diets during 0 to 20 of gestation, the number and percentage or resorptions, non-live fetuses, and malformed fetuses were increased in a dose-related manner; with a statistically significant increase in the high-dose group (20,000 ppm/1,055 mg/kg/day) (ATSDR, 1989). The NOAEL for teratogenic effects or maternal toxicity in a study of pregnant CD-1 mice exposed to BEHP in their diets was 250 ppm. BEHP was found to be developmentally toxic in ICR mice when administered orally (at 1,000 mg/kg and 2,000 mg/kg), but not when

administered by intraperitoneal injection. One hundred percent of live fetuses were malformed when pregnant mice were given 1 mL/kg MEHP on day 8 of gestation (ATSDR, 1989).

Mutagenic Effects

BEHP has not been shown to be mutagenic in most microbial and mammalian assay systems. Most of the data also suggest that MEHP and 2-ethylhexanol are not mutagenic (ATSDR, 1989).

Carcinogenic Effects

EPA has evaluated the weight of evidence on the carcinogenicity of BEHP and has concluded that it is a probable human carcinogen (Group B2). Evidence on potential carcinogenicity from animal studies is "sufficient", while there is no inadequate human data. Data from a bioassay using Fisher 344 rats and B6C3F1 mice have been used by EPA to calculate the upper-bound incremental unit carcinogenic risk to humans (the unit risk value is estimated to be 4.0×10^{-7} for drinking water containing 1 µg/L BEHP). These rodents were fed diets containing 0, 6,000 or 12,000 ppm for 103 weeks. A statistically significant increase in hepatocellular carcinomas and neoplastic nodules was observed in the high dose groups (NTP, 1982).

Ecotoxicity

The LC_{50} values for the midge, scud, and bluegill all exceeded the highest concentrations tested, which were 18,000 32,000 and 770,000 µg/liter, respectively. Because these values are greater than the water solubility of the chemical, it is unlikely that BEHP will be acutely toxic to organisms in natural waters. In a chronic toxicity test with *Daphnia magna,* significant reproductive impairment was found at the lowest concentration tested, 3µg/liter. These data imply that some chronic toxicity will be observed in freshwater aquatic life subsequent to long-term exposure to BEHP (ICF, 1985).

BEHP is removed from water primarily through uptake by suspended matter, sediments, and biota. BEHP is absorbed by both single- and multi-cellular organisms. The tendency for BEHP to undergo bioaccumulation is lessened because it is degraded by microorganisms and metabolized by invertebrates, fish and other animals. Very rapid bioaccumulation and concentration factors ranging from several hundred to several thousand times the concentration of BEHP in water, however, have been observed for various aquatic organisms, seen mostly in smaller aquatic invertebrates (ATSDR, 1989).

Acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 940 and 3 µg/L, respectively, according to available data; more sensitive species than those tested would be expected to be affected by even lower concentrations. For saltwater aquatic life, acute toxicity occurs at concentrations as low as 2,944 µg/L. No data are available to enumerate the chronic toxicity of phthalate esters to saltwater aquatic life; however, toxicity of one species of algae occurs at concentrations as low as 3.4 µg/L (EPA, 1986).

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	1.4×10^{-2} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2×10^{-2}mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	2×10^{-2} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.004 mg/l
AWQC:	Water and Fish Consumption - 15 mg/l (for phthalate esters) Fish Consumption - 50 mg/l

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BUTYL BENZYL PHTHALATE

Use

No information regarding the use of butyl benzyl phthalate (BBP) was located in the available literature.

Chemical and Physical Properties

Chemical Formula: $C_{19}H_{20}O_2$

MW: 312
Sol (water): 2.9 mg/L

BP: 377°C at 760 torr
MP: -35°C

Fate and Transport

Volatilization, photolysis and hydrolysis appear to be insignificant as fate processes of BBP. It is thought that, because phthalate esters, the class in which BBP is grouped, generally sorb to organic material and bioaccumulate, BBP would to do the same (EPA, 1979).

Pharmacokinetics

BBP appears to effect the kidneys, liver and testes of male rats (IRIS, 1990).

Human Toxicity

Noncarcinogenic

Systemic Effects

The National Toxicology Program (NTP) administered doses between 17 and 1417 mg BBP/kg/day in the diet of rats. Rats in the highest dose groups displayed depressed body weight and shrunken testes. Mean organ weight decreased for the heart, kidneys, lungs and seminal vesicles in the highest dose group only. An increase in liver weight was observed at the next to highest dose administration. Only the highest dose group showed evidence of abnormal morphology in any organ. In an addendum to this study, NTP noted a significant reduction in the total marrow cell count in the high dose group.

Two 14-day studies support the NTP results. In the first, significant increases in the liver and kidney weights of rats administered 5.0 percent doses of BBP per day for 14 days were noted. In the other, it was reported that only liver weights increased in rats fed 500,750 or 1000 mg BBP/kg/day (IRIS).

Teratogenic and Other Developmental Effects

No information regarding the teratogenic or developmental effects of BBP were located in the available literature.

Mutagenic Effects

Studies indicate that BBP is not mutagenic to *Salmonella typhimurium, E. Coli* of Chinese hamster ovary cells (IRIS).

Carcinogenic Effects

NTP (1985) noted that, among female rats given 12,000 ppm BBP daily for 103 weeks, the number of mononuclear cell leukemia or lymphoma significantly increased after 83 weeks. When given similar doses, male rats exhibited high mortality rates within the first 30 weeks due to internal hemorrhaging (IRIS). In the same study, however, mice displayed no toxic or carcinogenic effects when exposed to the same dosage. Theiss et al. (1977) noticed no induction of lung adenomas in Strain A mice administered intraperitoneal injections of 160, 400 or 800 BBP mg/kg.

Ecotoxicity

No information regarding the ecotoxic effects of BBP were located in the available literature.

Standards, Criteria and Guidelines

EPA Class C Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2×10^{-1} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	2×10^{0} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.1 mg/L
AWQC:	Water and Fish Consumption - 3000 µg/L
	Fish Consumption - 5200 µg/L

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4-CHLOROANILINE

Use

4-Chloroaniline is used in the synthesis of dyestuffs, chemical products, insecticides and many other industrial products.

Chemical and Physical Properties

Chemical Formula: $C_6H_4NH_2Cl$

MW:	127.57	BP:	231°C
FP:	>104°C	MP:	70°C
Sol. (water):	Insoluble		
Sol. (organics):	Ether		

Fate and Transport

No information on this topic was found in the literature.

Pharmacokinetics

Routes of entry include inhalation of vapor, skin contact and ingestion (Sittig, 1991).

Human Toxicity

Noncarcinogenic

Systemic Effects

Exposure to 4-chloroaniline may decrease the blood's ability to carry oxygen. This can cause headaches, breathing difficulty, weakness, a bluish color to the nose and lips, collapse and death. Contact can severely irritate and burn the eyes (Sittig, 1991).

Rats fed 250 and 500 ppm 4-chloroaniline for 78 weeks showed increased mortality and decreased body weight in the 500 ppm group. Lesions of the spleen were observed in most of the treated rats (NCI, 1979).

Teratogenic and Development Effects

No information on this subject was found in the literature.

Mutagenic Effects

No information on this subject was found in the literature.

Carcinogenic Effects

No information on this subject was found in the literature.

Ecotoxicity

No information on this subject was found in the literature.

Standards, Criteria, and Guidelines

Unclassified by EPA as to carcinogenicity.

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	4.0×10^{-3} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	4.0×10^{-3} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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DIBENZOFURAN

Use

Dibenzofuran has been used as an insecticide and as a laboratory chemical.

Chemical and Physical Properties

Chemical Formula: $(C_6H_4)_2O$

MW:	168.2	BP:	288°C
VD:	5.8	MP:	87°C

Fate and Transport

No information regarding the fate and transport of dibenzofuran was located in the available literature.

Pharmacokinetics

No information regarding the pharmacokinetics of dibenzofuran was located in the available literature.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

No information regarding the toxic effects of dibenzofuran was located in the available literature.

Teratogenic and Other Developmental Effects

No information regarding the teratogenic and developmental effects was located in the available literature.

Mutagenic Effects

Schoeny (1982) reported that dibenzofuran was non-mutagenic in several strains of *Salmonella typhimurium* with or without external metabolic activation.

Carcinogenic Effects

Dibenzofuran has not been widely studied. Polychlorinated dibenzofurans have been reported to be carcinogenic to humans, but these compounds are not adequately similar to dibenzofuran to propose any correlations (IRIS, 1990).

Ecotoxicity

No information regarding the ecotoxic effects of dibenzofuran were located in the available literature.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	4.0×10^{-3} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	4.0×10^{-3} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

DIETHYL PHTHALATE

Use

Diethyl phthalate (DEP) is used as a solvent for cellulose esters, as a vehicle in pesticidal sprays, as a fixitive and solvent in perfumery, as an alcohol denaturant, and as a plasticizer in solid rocket propellants (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: $C_6H_4 (OCOC_2H_5)_2$

MW:	222.24	BP:	298°C
SG:	1.1175 at 20°C	MP:	-40.5°C
Sol.(water):	896 mg/liter	VP:	0.05 mm Hg at 70°C
Sol.(organics):	Acetone, benzene		

Fate and Transport

Much of the information on the fate and transport of DEP is for phthalate esters in general. ICF (1985) evaluated this general information in relation to DEP. ICF reports that hydrolysis of DEP in surface waters is likely although this process occurs at such a slow rate that it is not environmentally significant. Photolysis and oxidation are not significant fate processes either. Although volatilization is not a significant fate in natural waters, it may occur slowly from DEP-containing materials at relatively high temperatures.

The most important environmental fate for DEP is absorption onto suspended solids and particulate matter, and complexation with natural organic substances. ICF (1985) reports that the octanol/water partition coefficient for DEP suggests that it would be absorbed onto particulates high in organic matter and, in fact, phthalate esters are often found in sediment samples. DEP readily forms water-soluble complexes with humic substances which may lead to its dispersal is aquatic and terrestial systems.

Bioaccumulation is considered an important fate process. Many unicellular and multicellular organisms take up and accumulate phthalate esters (ICF, 1985). Phthalate esters are bio-degraded under most conditions, however, making long-term bioaccumulation unlikely.

Pharmacokinetics

EPA (1980) reports that phthalate esters and their metabolites are readily absorbed from the intestinal tract, the intraperitoneal cavity, and the lungs. Shaffer et al. (1945) reports that a single oral dose of 10g of di-2-ethylhexyl phthalate (DEHP) in a human subject resulted in the recovery of a phthalate equivalent equal to 4.5 percent of the original dose in the urine after 24 hours, 5g of DEHP resulted in a 2 percent recovery. A study by Dillingham and Pesh-Imam (unref.)

indicated that dermal absorption may also occur. 24 hours after labeled DEHP had been applied to rabbit skin, 9 percent was detected in urine. After 48 hours, this level increased to 14 percent and after 72 hours, it reached 16-20 percent.

Absorbed phthalate esters and/or their metabolites are distributed quite rapidly to various organs and tissues in humans depending upon the route and physical form of the ester (EPA, 1980). Jaeger and Rubin (1970) reported the presence of DEHP in the spleen, liver, lung, and abdominal fat of 2 deceased patients who had received large volumes of blood stored in PVC blood bags. It also appears that, although distribution is rapid there is no apparent accumulation. Waddell, et al. (1977) found that DEHP accumulated in the kidney and liver was rapidly excreted into urine, bile, and the intestine.

However, EPA (1980) reports that patients having received large volume blood or blood products may have phthalate ester residues in their tissues and organs. Jacobson, et al. (1977) found trace amounts of DEHP 14 months after a transfusion in nonhuman primates. Dillingham and Pesh-Imam (unref.) report that dermal application of DEP resulted in its distribution to the lungs, heart, liver, kidneys, gonads, spleen, and brain after 3 days. Interestingly, no DEP was detected on the skin or subdermal fatty tissue at the site of application.

EPA (1980) states that significant biotransformation of phthalate esters in the gut is likely based on the Albro, et al. (1973) study with DEHP and rats. In their study, DEHP was converted to a monoester which is then further metabolized in the liver. Phthalate esters are, for the most part, readily excreted in urine and feces in humans (EPA, 1980). Lake, et al. (1975) found nearly all of a single oral dose of DEHP excreted in urine and feces within a 4 day period.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The phthalate esters may be considered as having a relatively low order of toxicity. In fact, it is now thought that the toxic effects of the esters are due to a metabolite, particularly the monoester (EPA, 1980). The low volatility of most of the esters makes acute toxicity from inhalation unlikely (EPA, 1980).

Several studies have indicated that chronic exposure to DEP affects weight gain and food consumption by laboratory animals. Brown et al. (1978) exposed groups of male and female CD rats to diets containing 0, 0.2, 1.0, or 5.0 percent DEP for 16 weeks. No changes in behavior or other clinical signs of toxicity were observed. Those rats exposed to 5.0 percent DEP exhibited significantly less weight gain throughout the 16 weeks, as did the females exposed to 1.0 percent DEP. These rats also exhibited reduced food consumption. Urinalysis, histologic

and hematologic examinations did not reveal any dose- or time-related trends. Although the absolute weights of the brain, heart, spleen, and kidneys were decreased in rats exposed to 5.0 percent DEP, the relative weights of the brain, liver, kidneys, stomach, small intestines, and full caecum were significantly greater in these rats.

In another study by Brown et al. (1978), rats exposed to 5.0 percent DEP in the diet exhibited increased food consumption (although not statistically significant) and a 7-10 percent reduction in weight gain. Food Research Laboratories, Inc. (1955) also reports reduced weight gain in rats exposed to 5.0 percent DEP in the diet. They also report a significant decrease in the efficiency of food utilization in this group.

Teratogenic and Other Developmental Effects

Singh et al. (1972) exposed pregnant Sprague-Dawley rats to 0.506, 1.012, and 1.686 ml/kg DEP intraperitoneally on days 5, 10, and 15 of gestation. Offspring exhibited skeletal malformations and reduced fetal size. NTP (1984) reports that oral exposure to 0.25, 1.25, and 2.5 percent DEP does not effect the reproductive performance of mice, however, second generation breeding pairs exposed to 2.5 percent DEP exhibited increased right epididymis and prostrate weights in males and decreased pituitary weights in females.

Mutagenic Effects

U.S. EPA (IRIS) reports that several studies have found DEP to be a weak direct-acting mutagen in forward and reverse mutation assays in S. typhimurium. Several other studies reported by U.S. EPA indicate that DEP produced negative results in mammalian cell chromosomal aberration assays.

Carcinogenic Effects

No data on the carcinogenicity of DEP were available in the literature reviewed.

Ecotoxicity

EPA (1986) reports that the available data for phthalate esters indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 940 and 3 µg/l respectively. Acute toxicity to saltwater species occurs at concentrations as low as 2,944 µg/l and chronic toxicity to one species of algae occurs at 3.4 µg/l. It should be noted that these concentrations would be lower in species more sensitive than those tested.

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	8×10^{-1} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	8×10^{0} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption: 350 mg/L
	Fish Consumption: 1800 mg/L

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2,4-DIMETHYLPHENOL

Use

2,4-Dimethylphenol is used in the manufacture of disinfectants, solvents, pharmaceuticals, insecticides, fungicides, rubber chemicals and dyestuffs. It is also an additive to, or constituent of, lubricants and gasolines.

Chemical and Physical Properties

Chemical Formula: $(CH_3)_2C_6H_3OH$

MW: 122.2
SG: .956 at 20°C

BP: 210°C
MP: 27°C
VP: 0.06 mm Hg at 20°C

Sol. (water): 17 g/liter
Sol. (organics): Freely soluble in alcohol, chloroform, ether and benzene

Fate and Transport

Photooxidation is probably the primary mechanism for removing 2,4-dimethylphenol from clear surface waters. Absorption and biodegradation may also play a role and are probably the main processes in murky stagnant waters. The high water solubility of this compound makes it highly mobile in soils, however, biodegradation limits its accessibility to ground waters (ICF, 1985).

Pharmacokinetics

2,4 Dimethylphenol was found to be distributed rapidly to the brain, liver and fatty tissue in rats receiving a 6 hour constant i.v. infusion (Somani et al., 1981; Kaka et al., 1982). Accumulation of this compound was not detectable in fat and the liver within 60 minutes of dosing. However, it did persist in brain tissue (Somani et al., 1981). Kaka et al. (1982) reported that this compound was rapidly metabolized to its conjugates, glucuronide being the main one.

Human Toxicity

Noncarcinogenic

Systemic Effects

2,4-Dimethylphenol causes severe skin and eye irritation. It may cause headaches, dizziness, nausea, vomiting, stomach pain and exhaustion (Sittig, 1991).

U.S. EPA (1989) reported squinting, lethargy, prostration and lack of muscle coordination in albino mice administered 250 mg 2,4-dimethylphenol for 90 days. It also reported lower mean hemoglobin concentration in females.

Teratogenic and Developmental Effects

No information on this topic found in literature.

Mutagenic Effects

Negative results for dimethylphenols were reported in reverse notation assays in *Salmonella typhimurium* and in *Escherichia coli* (Pool and Lin, 1982; Florin, et al., 1980; Szybalski, 1958).

Carcinogenic Effects

2,4-Dimethylphenol has been shown to promote cancer in rat skin painting studies (ICF, 1985).

Boutwell and Bosch (1959) reported that dimethylphenol isomers may be carcinogenic to the skin of mice.

Ecotoxicity

2,4-Dimethylphenol does not appear to be acutely toxic to freshwater species (ICF, 1985).

Fathead minnows exposed to 2,4-dimethylphenol for 32 days experienced growth inhibition. Embryos were more resistant to this compound than larval and juveniles (Holcombe et al., 1982).

Standards, Criteria, and Guidelines

Unclassified by EPA as to carcinogenicity.

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2.0×10^{-2} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	2.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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DI-N-BUTYL PHTHALATE

Use

Di-n-butyl phthalate, also known as dibutyl phthalate and dibutyl-1,2-benzenedicarbozylate, is used in plasticizing vinyl acetate emulsion systems and in plasticizing cellulose esters. It is also used as an insect repellant (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: C_6H_4 $(COOC_4H_9)_2$ BP: 340°C
MW: 278.34 FP: 171°C
Sol. (water): 0.4 mg/liter SG: 1.0484 at 20°C
Sol. (organics): very soluble in alcohol, ether, acetone, and benzene.

Fate and Transport

The US EPA (1981) reports the dominant transport process for di-n-butyl-phthalate is probably that of phthalate esters as a group, that is, sorption onto particulates and complexation with organics. Direct photolysis does not occur and indirect photolysis appears to be too slow to be environmentally important. Oxidation is considered unimportant and hydrolysis of phthalate esters as a group is believed to be too slow to be significant. Volatilization, as well, does no appear to be an important transport process. Phthalate esters in general have been found bioaccumulated in many organisms, are known to biodegrade rapidly in natural soil, and undergo some biotransformation. In fact, all biological processes are considered important fates for phthalate esters (US EPA, 1981).

Pharmacokinetics

The US EPA (1980) reports that phthalic acid esters and/or their metabolites are readily absorbed from the lungs, intraperitoneal cavity, the intestinal tract, and possibly through the skin. Shaffer et al. (1945) reported that 4.5 percent of a single dose of 10 g of DEHP (di-2-ethylhexyl phthalate) in a human subject was recovered in the urine after 24 hours. In another subject, 2 percent of a 5 g dose was recovered in the urine after 24 hours. Wallen et al. (1974) found that a significant amount of orally administered DEHP is absorbed in the gastrointestinal tract as the intact compound. The US EPA (1980) reports that Dillingham and Pesh-Imam detected 9 percent of a labeled dose of DEHP that had been applied to a rabbit's skin in the urine after 24 hours. In 48 hours the level had risen to 14 percent and in 72 hours it had risen to 16-20 percent of the original dose.

The US EPA (1980) reports that absorbed esters of phthalate acid esters or their metabolites distribute quite rapidly to various organs and tissues in animals and humans but accumulation apparently does not occur. Jaeger and Rubin (1970) reported on the distribution of DEHP in

human tissues of 2 deceased patients who had received large volumes of blood stored in PVC blood bags. They detected the presence of DEHP in the spleen, liver, lung, and abdominal fat. Intravenously administered DEHP was found to disappear rapidly from the blood and within 2 hours 60-70 percent of the total dose was detected in the liver and lungs. Results from a study by Waddell et al. (1977) reveal a rapid accumulation of DEHP in the kidney and liver followed by a rapid excretion into urine, bile, and the intestine. Albro et al. (1973) concluded that the first step in the metabolism of DEHP in rats is conversion of the diester to the monoester mono-2-ethylhexyl phthalate followed by a series of oxidations. The US EPA (1980) concluded from this that the same metabolism is possible for other diesters and it is possible in other animals, including man.

Illustrative of several studies, Lake et al. (1975) found that a single oral dose of DEHP was practically all excreted in the urine and feces within a 4-day period, leaving less than 0.1 percent of the DEHP in the organs and tissues.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Smith (1953) fed diets containing 0, 0.01, 0.05, 0.25, and 1.25 percent dibutyl phthalate to male Sprague-Dawley rats in groups of 10 for 1 year. One-half of all the rats receiving the highest dose died during the first week. The remaining animals all lived and exhibited no treatment-related adverse effects. Men'shikova (1971) exposed rats continuously for 93 days to chamber concentrations of 0.098, 0.256, and 0.98 mg/m^3. The only detected adverse effect was a dose-related increase in gamma globulin.

Men'shikova (1971) reported that atmospheric concentrations of 0.12 and 0.15 mg/m^3 resulted in abnormal encephalographic responses in 3 human subjects in the study. A reduced level of 0.093 mg/m^3 appeared to have no effect. In a study of 147 industrial workers (87 women and 60 men) exposed primarily to dibutyl phthalate with other esters present in lower concentration, Milkov et al. (1973) found varying degrees of toxic polyneuritis.

Teratogenic and Other Developmental Effect

In a rat teratogenic study, Singh et al. (1975) administered 0.305 ml/kg, 0.610 ml/kg, and 1.01 ml/kg dibutyl phthalate intraperitoneally to pregnant female rats on days 5, 10, and 15 of gestation. Dose-related gross and skeletal abnormalities were observed in the fetuses that included absence of tail, anophthalmia, twisted hands and legs, hematomas, elongated and fused ribs, absence of tail bones,

abnormal or incomplete skull bones, and incomplete or missing leg bones. Reduced fetal weight was also observed.

Mutagenic Effects

Dibutyl phthalate did not induce mutations in *Salmonella* strains TA100 and TA98 in a modified reverse mutation plate incorporation assay at concentrations up to 1000 µg/plate in the presence or absence of 59 hepatic homogenate (Kozumbo et al., 1982). It was also negative for clastogenic activity in human leukocytes (Tsuchiya and Hattori, 1977).

However, Seed (1982) found that it was a weak direct-acting mutagen in a forward mutation assay in *S. typhimurium.* CMA (1986) also found it to be mutagenic in the mouse lymphoma forward mutation assay, but only in the presence of metabolic activation. Additionally, Ishidate and Odashima (1977) found some evidence of clastogenic activity in Chinese hamster fibroblasts.

Carcinogenic Effects

Pertinent data regarding the carcinogenicity of di-n-butyl phthalate were not found in the literature reviewed. Di-n-butyl phthalate is designated Group D (not-classified) for the weight-of-evidence category for potential carcinogens.

Ecotoxicity

LC_{50} values for 4 fish and 2 invertebrates with di-n-butyl phthalate were conducted. Values ranged from 730-6,470 µg/liter. Bluegills were the most sensitive fish and scuds the most sensitive invertebrate tested (US EPA, 1980) For phthalate esters in general, acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 940 and 3 µg/liter, respectively, and is expected to be lower among more sensitive species (US EPA, 1986).

The US EPA (1986) reports that acute toxicity to saltwater aquatic life for phthalate esters occurs at concentrations as low as 2,944 µg/liter and is expected to be lower among more sensitive species than those tested. No data concerning the chronic toxicity of phthalate esters to saltwater aquatic life was found in the literature reviewed.

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	$1x10^{-1}$ mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	1×10^{0} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 34 mg/l Fish Consumption - 154 mg/l

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3-3'-DICHLOROBENZIDINE

Use

3,3'-Dichlorobenzidine (DCB) is used in the production of yellow, red, and orange paint pigments, and in the textile, paper, rubber and plastic industries. DCB is also used as a compounding ingredient for rubber and plastics, as a test for the presence of gold, and in the formulation of fire protective equipment (ATSDR, 1989). DCB is produced primarily through the reduction of o-nitrochlorobenzene.

Chemical and Physical Properties

MF: $C_{12}H_{10}Cl_2N_2$
MW: 253.13
Sol.(water): 3.11 mg/l
Sol.(organics): alcohol, benzene

BP: 368°C
MP: 132°C
VP: 4.5E-09 at 20°C

Fate and Transport

In the atmosphere, DCB adsorbs to particulate material. Wet and dry deposition are thought to be the primary means of removal of DCB from the air (ATSDR, 1989). In aquatic media, DCB's solubility is relatively low. In waters with high organic content, DCB will adsorb to silt, clays, and sediments as well as the organics. In soils, DCB is not readily biodegraded but is known to bioaccumulate (ATSDR, 1989). DCB is not expected to desorb or leach from soils.

Pharmacokinetics

DCB is readily absorbed through the lungs via inhalation and through the lining of the digestive tract after oral exposure routes due to the size of the DCB particle. Dermal absorption is not considered to be significant. Handke et al. (1986) noted DCB's presence in the urine of exposed workers following inhalation.

Hsu and Sikka (1982) reported that rats absorbed over 90 percent of a radiologically marked quantity of DCB exposed orally. Dermal absorption in rats of DCB was shown to be relatively low. Shah and Guthrie (1983) noted that 6 percent, 23 percent and 49 percent of radiologically marked DCB was absorbed through the skin over 1-, 8- and 24-hour periods, respectively.

Once absorbed, DCB is distributed to the kidney, lungs, spleen, heart, pancreas and testes (Hsu and Sikka, 1982).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Inhalation of DCB is thought to cause sore throats, upper respiratory infections, and gastrointestinal upset in workers, however, no adverse effects were noted in rats exposed to DCB via inhalation (Gerarde and Gerarde, 1970).

Dermatitis is frequently noted in workers exposed dermally to DCB. No laboratory studies reported the occurrence of any adverse effects in animals exposed to DCB (ATSDR, 1989).

Teratogenic and Other Developmental Effects

DCB is thought to induce chromosomal aberrations in pregnant mice. Cihak and Vontorvoka (1987) reported that micronuclei were induced in polychromatic erythrocytes in the bone marrow of dams after oral exposure to 1000 mg/kg-bw DCB. Abnormal growth of kidneys was noted in the fetuses of pregnant mice treated with 257/kg-bw daily (ATSDR, 1989).

Mutagenic Effects

DCB is though to be genotoxic *in vitro* and *in vivo* to rat embryo cells, hamster kidney cells, and *S. typhimurium*. In all cases, however, an activation system was required for the effects to be noted (ATSDR, 1989). Shiraishi (1986) reported that DCB induced sister-chromatid-exchange in Bloom Syndrome ß-lymphablastoid cell lines.

Carcinogenic Effects

DCB may be carcinogenic to human bladder tissues, however, disputing evidence exists so the data is considered inadequate (IRIS). Pliss (1959) exposed white rats to 10 - 20 mg DCB per day, 6 days per week for 12 months. Of the 29 surviving rats, 22 had developed tumors. Tumor sites included (in order of decreasing frequency) the zymbal gland, skin, mammary gland, ileum, bladder, hemopoietic, connective tissue, salivary glands, liver and thyroids.

In a separate study, Stula et al. (1975) fed 1000 ppm DCB to ChR-DC rats in the diet for 2 years. At the end of the study, the researchers noted a significant increase in granulocytic leukemia, mammary adenocarcinoma, and zymbal gland carcinoma.

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Oral Slope Factor:	4.5×10^{-1} $(mg/kg/day)^{-1}$
Inhalation Slope Factor;	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 0.0103 µg/l
	Fish Consumption - 0.0204 µg/l

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DIMETHYL PHTHALATE

Use

Dimethyl phthalate (DMP) is a colorless oily liquid which is used as a plasticizer for cellulose ester plastics and as an insect repellant (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: $C_6H_4(COOCH_3)_2$

MW:	194.19	BP:	283.7°C
SG:	1.196 at 15.6°C	MP:	5.5°C
Sol.(water):	0.43 g/100ml	VP:	<.01 mm Hg 20°C

Fate and Transport

Much of the information on the fate and transport of DMP is for phthalate esters in general. ICF (1985) evaluated this general information in relation to diethyl phthalate (DEP), a phthalate ester similar to DMP. ICF reports that hydrolysis of DEP in surface waters is likely although this process occurs at such a slow rate that it is not environmentally significant. Photolysis and oxidation are not significant fate processes either. Although volatilization is not a significant fate in natural waters, it may occur slowly from DEP-containing materials at relatively high temperatures.

The most important environmental fate for DEP is absorption onto suspended solids and particulate matter, and complexation with natural organic substances. ICF (1985) reports that the octanol/water partition coefficient for DEP suggests that it would be absorbed onto particulates high in organic matter and, in fact, phthalate esters are often found in sediment samples. DEP readily forms water-soluble complexes with humic substances which may lead to its dispersal is aquatic and terrestial systems.

Bioaccumulation is considered an important fate process. Many unicellular and multicellular organisms take up and accumulate phthalate esters (ICF, 1985). Phthalate esters are bio-degraded under most conditions, however, making long-term bioaccumulation unlikely.

Pharmacokinetics

EPA (1980) reports that phthalate esters and their metabolites are readily absorbed from the intestinal tract, the intraperitoneal cavity, and the lungs. Shaffer et al. (1945) reports that a single oral dose of 10g of di-2-ethylhexyl phthalate (DEHP) in a human subject resulted in the recovery of a phthalate equivalent equal to 4.5 percent of the original dose in the urine

after 24 hours, 5g of DEHP resulted in a 2 percent recovery. A study by Dillingham and Pesh-Imam (unref.) indicated that dermal absorption may also occur. 24 hours after labeled DEHP had been applied to rabbit skin, 9 percent was detected in urine. After 48 hours, this level increased to 14 percent and after 72 hours, it reached 16-20 percent.

Absorbed phthalate eaters and/or their metabolites are distributed quite rapidly to various organs and tissues in humans depending upon the route and physical form of the ester (EPA, 1980). Jaeger and Rubin (1970) reported the presence of DEHP in the spleen, liver, lung, and abdominal fat of 2 deceased patients who had received large volumes of blood stored in PVC blood bags. It also appears that, although distribution is rapid there is no apparent accumulation. Waddell, et al. (1977) found that DEHP accumulated in the kidney and liver was rapidly excreted into urine, bile, and the intestine.

However, EPA (1980) reports that patients having received large volume blood or blood products may have phthalate ester residues in their tissues and organs. Jacobson, et al. (1977) found trace amounts of DEHP 14 months after a transfusion in nonhuman primates. Dillingham and Pesh-Imam (unref.) report that dermal application of DEP resulted in its distribution to the lungs, heart, liver, kidneys, gonads, spleen, and brain after 3 days. Interestingly, no DEP was detected on the skin or subdermal fatty tissue at the site of application.

EPA (1980) states that significant biotransformation of phthalate esters in the gut is likely based on the Albro, et al. (1973) study with DEHP and rats. In their study, DEHP was converted to a monoester which is then further metabolized in the liver. Phthalate esters are, for the most part, readily excreted in urine and feces in humans (EPA, 1980). Lake, et al. (1975) found nearly all of a single oral dose of DEHP excreted in urine and feces within a 4 day period.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The phthalate esters may be considered as having a relatively low order of toxicity. In fact, it is now though that the toxic effects of the esters are due to a metabolite, particularly the monoester (EPA, 1980). The low volatility of most of the esters makes acute toxicity from inhalation unlikely (EPA, 1980).

Dimethyl phthalate has produced few toxic effects in humans. Draize, et al. (1948) conducted two-year feeding studies in female rats with DMP concentrations ranging from 2 to 8 percent in the diet. Levels between 4 and 8 percent produced only a minor growth effect. Some indication of nephritic involvement was detected at the 8 percent level. EPA (1980) reports that a

dermal application study with rabbits resulted in an LD_{50} (the dost at which ½ the study population died) of greater than 4 ml/kg. EPA (1980) also reports that DMP does not produce primary irritation of the skin nor has it been found to act as a sensitizing agent.

Teratogenic and Other Developmental Effects

Singh, et al. (1975) exposed pregnant female rats intraperitoneally to 1/10, 1/5, and 1,3 of the acute LD_{50} of DMP. Treatments occurred on days 5, 10, and 15 of gestation and the fats were sacrifices on day 20. DMP produced dose-related gross and skeletal abnormalities. Gross abnormalities included absence of tail anophthalmia, twisted hands and legs, and hematomas. Skeletal abnormalities included elongated and fused ribs (bilateral and unilateral), absence of tail bones, abnormal or incomplete skull bones, an incomplete or missing leg bones. Dead fetuses were also detected. EPA (1980) states that the results of this intraperitoneal study should no be extrapolated to possible teratogenic effects if the compounds had been administered orally or by other routes.

Mutagenic Effects

EPA (IRIS) reports that several studies found DMP to be a weak direct-acting mutagen in forward and reverse mutation assays in S. typhimurium. CMA (1986) reported that DMP was active in the mouse lymphoma forward mutation assay only in the presence of metabolic activation. Yurchenko and Gleiberman (1980) reported negative results in a mouse dominant lethal test.

EPA (IRIS) also reports that several studies indicate that DMP is hydrolyzed to the monoester which Kozumbo et al., (1982) has shown to be nonmutagenic in Salmonella assays.

Carcinogenic Effects

Data on the carcinogenicity of DMP was not available in the literature reviewed. An in vitro mutagenic assay, performed by Rubin et al. (1979), produced positive results for DMP, however, suggesting but not proving carcinogenic potential (EPA, 1980).

Ecotoxicity

EPA (1986) reports that the available data for phthalate esters indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 940 and 3 µg/l respectively. Acute toxicity to saltwater species occurs at concentrations as low as 2,944 µg/l and chronic toxicity to one species of algae occurs at 3.4 µg/l. It should be noted that these concentrations would be lower in species more sensitive than those tested.

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	1.0×10^0 mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	1.0×10^0 mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption: 313 mg/L
	Fish Consumption: 2900 mg/L

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2,6-DINITROTOLUENE

Use

Dinitrotoluene is used in the formulation of dyes, in the munitions industry and in the waterproofing of explosive compositions.

Chemical and Physical Properties

Chemical Formula: $C_7H_6N_2O_4$

MW:	182.15	BP:	300°C
SG:	1.32 at 71°C	MP:	71°C
FP:	206.7°C	VP:	0.018 torr at 20°C

Sol.(water): 0.3 g/kg at 20°C
Sol.(organics): alcohol, ether, acetone, benzene

Fate and Transport

Photochemical degradation is expected to be an important removal mechanism (Spanggord et al., 1980; Zepp et al., 1984). 2,6-Dinitrotoluene (2,6-DNT) could potentially absorb to humus and clay particulate matter and sediments, thereby reducing the probability of leaching (Callahan et al, 1979). 2,6-DNT biodegrades under aerobic anareobic conditions in water, therefore it is also expected to undergo microbial biodegradation in soil (HEA, 1987). Bioaccumulation of dinitrotoluene in animal tissues is expected to be negligible (Toxicological, Profile, 1989).

Pharmacokinetics

Routes of absorption include inhalation of vapor, percutaneous absorption of liquid, ingestion and eye and skin contact (Sittig, 1991).

The main metabolites, excreted by workers exposed to technical grade dinitrotoluene, were dinitrobenzoic acid, 2-amino-4-nitrobenzoic acid and dinitrobenzol glucuronide. The predominant excretion route in humans appears to be through urine (Levine et al., 1985).

Human Toxicity

Noncarcinogenic

Systemic Effects

Acute toxic effects result from the ability of this compound to reduce the oxygen carrying capacity of the blood. These effects include headache, irritability, dizziness, weakness, unconsciousness, and death (NIOSH, 1985).

A mild dermal response was observed in guinea pigs in response to 2,6-DNT. In feeding studies of rats and mice, this compound reduced the rate of weight gain in both sexes. Toxic effects on the liver by this compound have been shown. Rats fed 14 or 35 mg/kg/day technical-grade dinitrotoluene showed degeneration and alteration of the hepatocytes and bile duct epithelium. These lesions were shown to be dose-dependent (Rickert et al., 1984).

Teratogenic and Develompent Effects

No teratogenic effects were observed in the offspring of pregnant rats gavaged with up to 100 mg/kg/day of technical-grade DNT from days 7 to 20 of gestation (NIOSH, 1985). Testicular atrophy and decreased spermatogenesis or aspermatogenesis were induced in rats, mice and dogs administered 2,6-DNT through gavage or feeding (NIOSH, 1985; Bloch et al., 1988).

Mutagenic Effects

Bacterial assays have shown DNT to be weakly mutagenic (NIOSH, 1985). Furthermore, a metabolite of 2,6-dinitrotoluene, 2,6-dinitrobenzaldehyde, has also been found to be a mutagen (Sayama et al., 1989).

Purified 2,6-DNT is a potent inducer of DNA repair in rat mutagenicity tests (NIOSH, 1985; Rickert et al., 1984; Bermundez et al., 1979).

Carcinogenic Effects

In rats given 7 mg/kg/day of this compound, 18 of 20 developed liver cell carcinomas and 19 of 19, given 14 mg/kg/day, showed the same effect after 52 weeks. The conclusion that 2,6-DNT is a potent liver carcinogen was reached by these authors (Leonard et al., 1987).

Schut et al. (1983), however, showed that 2,6-DNT did not seem to induce pulmonary tumors in mice.

Ecotoxicity

No information on this subject was found in the literature.

Standards, Criteria, and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	6.8×10^{-1} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	NA
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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DI-N-OCTYL PHTHALATE

Use

Di-n-octyl phthalate (DOP) is used as a plasticizer in the manufacture of plastics (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: C_6H_4 $(COOC_8H_{17})_2$

MW: 391.0
SG: 0.978
Sol. (water): 3 mg/L at 25°C

BP: 220°C at 5 mmHg
MP: 25°C
VP: <0.2 at 150°C

Fate and Transport

The fate and transport of DOP can only be inferred from data on other phthalate esters. Photolysis, oxidation, and hydrolysis are probably not significant fate processes for DOP. Volatilization does not appear to be an important fate process except during the manufacture of plastics (ICF, 1985). DOP is expected to adsorb to organic material in both soils and aquatic media. Bioaccumulation is known to occur in a variety of organisms (ICF, 1985).

Pharmacokinetics

Information regarding the pharmacokinetics of DOP was not located in the available literature.

Human Toxicity

Noncarcinogenic

Systemic Effects

DOP does not appear to be highly toxic to mice but is known to be a severe eye irritant and a mild skin irritant to rabbits (ICF, 1985). The LD_{50} value for mice was determined to be 1.3 mg DOP/kg when injected intraperitoneally for 5 days/week for 10 weeks (ICF, 1985).

Teratogenic and Other Developmental Effects

DOP was shown to be fetotoxic and teratogenic to rats. The same study indicated that rats, when intraperitoneally administered 5 g DOP/kg on days 5 to 15 of gestation, showed an increase in fetal resorption and fetal toxicity.

Mutagenic Effects

No evidence is available indicating that DOP is mutagenic.

Carcinogenic Effects

Information regarding the carcinogenicity of DOP was not located in the available literature.

Ecotoxicity

Freshwater species display seven to eight day LC_{50} values between 690 and 42,000 ug/L for DOP (ICF, 1985).

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2.00×10^{-2} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	2×10^{-2} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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2-METHYLPHENOL

Use

2-Methylphenol is used as a disinfectant; a solvent; a fiber treatment agent, tanning agent and a metal degreasing agent.

Chemical and Physical Properties

Chemical Formula: CH_3C_6OH

MW:	108.14	MP:	30.9°C
SG:	1.048	VP:	0.2453 mm Hg at 25°C
FP:	81°C	BP:	191°C

Sol. (water): 31 g/liter at 40°C
Sol. (organics): Miscible with alcohol, benzene, ether and glycerol

Fate and Transport

2-Methylphenol or O-Cresol is one of the isomers of Cresol. From the physical data, Cresol is not expected to volatize. The main transport mechanism in the environment is movement in water. In aerated surface waters it is photooxidized. It also seems that biodegradation by water and soil organisms is an important fate process. Because of these competing degradation processes, cresol is not very persistent in the environment (ICF, 1985).

Pharmacokinetics

Possible routes of entry include inhalation or percutaneous absorption of liquid or vapor, ingestion, eye and skin contact (Sittig, 1991). The skin absorption rate is more strongly dependent on the size of the area exposed than on the concentration. The major route of excretion of the Cresols is in the urine and the bile. Trace amounts may also be exhaled. The body metabolism of Cresol includes oxidation and conjugation with sulfuric and glucuronic acids. It is important to note that the Cresols are normally present in human urine (Patty, 1978).

Human Toxicity

Noncarcinogenic

Systemic Effects

Acute exposure by all routes may cause muscular weakness, gastroenteric disturbances, depression, collapse and death. The primary effects are manifested on the central nervous system but kidney, liver, pancreas and spleen injury may

also occur. Cresol is very corrosive to tissues, producing burns and dermatitis. Certain individuals are hypersensitive to Cresol (Patty, 1978). Children exposed to this compound have shown convulsions (Sittig, 1991).

Teratogenic and Developmental Effects

No information on this topic was found in the literature.

Mutagenic Effects

Ortho-Cresol has been shown to be negative in the induction of unscheduled DNA synthesis in rat hepatocytes (Litton Bionetics, 1981c). Ortho-Cresol was also negative in BALB/3T3 cell transformation assays (Litton Bionetics, 1980d). The Cresols have not been found to be mutagenic for various strains of *Salmonella typhimurium* (Haworth et al., 1983).

Carcinogenic

The Cresols have been shown to be tumor promoters (Proctor et al., 1988). Garrett (1978) reported bladder cell carcinoma after chronic exposure to Cresol and Creosote.

Ecotoxicity

Waterborne Cresol isomers are toxic to aquatic life. Trout embryos seem to be one of the most sensitive species. There is no evidence available that the Cresols bioaccumulated in the tissues of wildlife species. No alterations in reproductive activity or other changes have been attributed to these compounds (ICF, 1985).

Standard, Criteria, and Guidelines

EPA Class C Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	5.0×10^{-2} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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4-METHYLPHENOL

Use

4-Methylphenol, more commonly known as p-cresol, is generally used commercially as a mixture with o- and m-cresol. Cresols are produced by distilling coal tar, cracking petroleum, and by synthesis from other chemicals (EPA, 1985). Cresols are often used as solvents and disinfectants. 4-Methylphenol, in particular, has been used primarily as an antioxidant, a resin, and a wire enamel solvent (EPA, 1985).

Chemical and Physical Properties

MF: C_7H_8O	BP: 202°C
MW: 108.13	MP: 35.3°C
Sol.(water): 24,000 mg/l at 40°C	VP: 0.11 at 25°C
Sol.(organics): alcohol, glycol	

Fate and Transport

The half-life of 4-methylphenol exposed to direct sunlight in aquatic media is estimated to be 70 days. Photolysis and photodegradation are thought to be the primary breakdown processes in aquatic media (Smith et al., 1978). In natural water bodies, the half-life of 4-methylphenol ranges from 100-400 days. The presence of humic acid has been shown to retard the photodegradation of 4-methylphenol. Biodegradation is also considered to be a significant fate of cresols in aquatic media.

The atmospheric fate of 4-methylphenol appears to be chemical reactivity. 4-methylphenol reacts with oxygen, ozone, and nitrates in the atmosphere. A half-life of less than 1 minute has been estimated (Carter, et al., 1981). Wet and dry deposition of 4-methylamine appears to be negligible.

Biodegradation is expected to be the fate determining process for 4-methylphenol in soils. Cresols are not expected to volatilize but are expected to leach from soils (EPA, 1985).

Pharmacokinetics

Cresols are readily absorbed across the epithelial tissues of the respiratory and gastrointestinal tracts and through the skin. Eighty percent of cresols administered to rabbits by gavage was recovered in the urine within 24 hours (Bray, 1950). In a fatal case of dermal contact by an infant with coal tar fluid (90 percent cresols), cresol was identified in the blood, liver, urine and brain (Green, 1975). Cresols are metabolized into sulfate and glucoronate conjugates through oxidation.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

In a study conducted by EPA (1986), Sprague-Dawley rats were exposed by gavage to 0, 50, 175 or 600 mg/kg/day of 4-methylamine once daily for ninety days. At the end of the study, the high dose rats (600 mg/kg/day) showed a significant reduction in weight gain and a significant increase in central nervous system (CNS) depression. The CNS effects noted include lethargy, excess salivation, tremors and diarrhea. In the mid-dose groups, a reduction in body weight was noted.

Pertinent data regarding the chronic toxicity of 4-methylphenol were not located in the available literature.

Teratogenic and Other Developmental Effects

Several human case studies indicate that cresol-containing solutions have been used illegally as abortifacients (Presley and Brown, 1956).

Mutagenic Effects

4-Methylphenol has been shown to cause unscheduled DNA synthesis in human lung fibroblast cells and primary rat hepatocytes (Litton, 1980). Cresols are not mutagenic to various strains of *S. typhimurium* (EPA, 1991).

Carcinogenic Effects

Data regarding the carcinogenic effects of 4-methylphenol in humans and laboratory animals is limited. Cresols have been proven to be co-carcinogens when exposed dermally with polycyclic aromatic hydrocarbons (Boutwell and Bosch, 1959).

Standards, Criteria and Guidelines

EPA Class C Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	5×10^{-2} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - NA Fish Consumption - NA

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3-NITROANILINE

Use

3-Nitroaniline, also known as m-nitroaniline, is used predominantly as a dye intermediate (U.S. EPA, 1985). Hall et al. (1980) report that 3-nitroaniline is also used to manufacture photographic anti-fogging agents, coccidostatics, interior paint pigments, and artificial sweeteners.

Chemical and Physical Properties

Chemical Formula: $C_6H_6N_2O_2$

MW:	138.12	MP:	111.8°C
SG:	1,430 at 20°C	BP:	305-307°C
VP:	1 mmℓHg at 119.3°C		
Sol. (water):	890 mg/l at 25°C		

Fate and Transport

The U.S. EPA (1985) speculates that atmospherically nitroanilines in general, may be susceptible to oxidation of the amino group via photochemical reactions and through interactions with hydroxy radicals and molecular oxygen. The U.S. EPA (1985) also reports that transport of nitroanilines from the atmosphere to surface water and soil through wet deposition seems significant because their reasonably high aqueous solubility.

In aquatic media, nitroanilines could react with the available free radicals in the aquatic media and/or undergo direct photochemical reactions (U.S. EPA, 1985). Several studies have indicated that 2- and 4-nitroaniline, at least, may undergo some photoreaction in aquatic media, although the significance of this fate process is unclear (U.S. EPA, 1985). A study by Liang (1963) indicates that nitroanilines will not undergo significant hydrolysis under natural aquatic conditions. Based on a study conducted by Endyus'kin and Fidippov (1980), the oxidation of residual nitroaniline in drinking water during the chlorination step of the treatment process seems possible. The results of a study conducted by Challis et al. (1978) indicate that nitrosation will not be a significant fate process in natural aquatic systems. The U.S. EPA (1985) reports that significant volatilization of nitroanilines from aquatic media is unlikely given the high aqueous solubilities and expected vapor pressures of these compounds. In addition, the U.S. EPA (1985) reports that adsorption and subsequent precipitation in bottom sediments probably plays only a moderate role in the removal of nitroanilines from aquatic media.

Information regarding the biodegradability of nitroanilines is conflicting. While many studies indicate that nitroanilines are rapidly biodegraded by microorganisms in various media, others have shown nitroanilines to be virtually nonbiodegradable. Therefore, it is not possible to determine to significance of biodegradation as a fate process in aquatic media.

Based on a bioconcentration factor of 8, calculated by Howard et al. (1976) for 3-nitroaniline, bioaccumulation of 3-nitroaniline in aquatic species should not be significant.

In soils, the U.S. EPA (1985) states that biodegradation of nitroanilines in soils would occur slowly. In contrast, a study by Briggs (1981) indicates that 3-nitroaniline should be moderately mobile in soil.

Pharmacokinetics

Although no data concerning the adsorption of 3-nitroaniline were available, 4-nitroaniline was found to be readily adsorbed by the gastrointestinal tract and peritoneal cavity. Mate et al. (1967) recovered 85.7% of a gavage administered dose in the urine within 24 hours following exposure, and 80.9% of an intraperitoneally-injected dose.

No data regarding the distribution of nitroaniline were available. A study conducted by Mate et al. (1967) indicates that 4-nitroaniline is metabolized to p-phenylenediamine (43%) and 2-amino-5-nitrophenol (26%). Five hours after 13.8 mg/kg/bw of 3-nitroaniline were administered intraperitoneally to 5 male rats, unspecified diazopositive metabolites of the nitroaniline isomers were identified in the urine by Watanabe et al. (1976).

Human Toxicity

Non-Carcinogenic Effects

Systemic Effects

Data regarding the toxicity of 3-nitroaniline are limited. Several Russian studies cited in U.S. EPA (1985) report that acute oral exposure to the three isomers of nitroaniline resulted in an increased number of red blood cells, white blood cells (except for lymphocytes), and reticulocytes in experimental animals. Walter and Israel (1974) report that Heinz bodies and clumps of denatured hemoglobin are noted in the red blood cells of animals exposed to nitroanilines.

Data regarding the toxicity of 4-nitroaniline are available. Houser et al. (1983) reports that no differences were observed in body weight gain, food consumption, or clinical chemistry parameters between control rats and rats treated by gavage with 0, 3, 10, or 30 mg/kg bw/day of 4-nitroaniline for 90 days. However, exposed rats did exhibit a dose-related increase in the blood level of methemoglobin, as well as decreased hematocrit and hemoglobin levels, and increased reticulocyte counts and mean corpuscular volumes. At all dose levels, microscopic changes in the spleen, including excessive extramedullary hemopoiesis, hemosiderosis, splenic congestion, and vacuolization of the red pulp were observed. Similar results were reported by Chhabra et al. (1983) and Nair

et al. (1983). Studies conducted by Anderson (1946) and Belknap (1957) reveal that the adverse effects on the oxygen transporting capacity of the blood are consistent with cases of human poisoning in which onset of cyanosis and dyspnea occurred several hours following exposure to 4-nitroaniline (U.S. EPA, 1985).

Teratogenic and Other Developmental Effects

No data regarding the teratogenicity of nitroanilines were available in the literature reviewed.

Mutagenic Effects

3-Nitroaniline did not induce reverse mutations in *Salmonella typhimurium* strains TA100 or TA98 at concentrations of 0.1, 1.0, and 10.0 µmol (probably per plate) in the absence of metabolic activation (Chiu et al., 1978). Garner and Nutman (1977), however, found that, in the presence of metabolic activation (rat liver microsomal preparations), there was a significant increase in the number of *S. typhimurium* strain TA1538 mutants when they were incubated in the presence of 50 and 100 µg/plate of 3-nitroaniline. As in Chiu et al. (1978), mutagenicity did not occur in the absence of metabolic activation. As in Garner and Nutman (1977), Thompson et al. (1983) reports that 3-nitroaniline was mutagenic to *S. typhimurium* strains TA1535, TA100, TA1538, and TA98 at concentrations ranging from 30-100 µg/ml agar in the presence of metabolic activation. In addition, Thompson et al. (1983) reports that no nitroanilines caused unscheduled DNA synthesis in primary cultures of adult rat hepatocytes when tested up to 500 nmoles/ml.

Carcinogenic Effects

No data regarding the carcinogenicity of nitroanilines were available in the literature reviewed.

Ecotoxicity

No data regarding the ecotoxicity of 3-nitroaniline were available in the literature reviewed. Data on the ecotoxicity of 4-nitroaniline, however, are available. In acute toxicity studies, Curtis and Ward (1981) reported a 96 hour LC_{50} of 106.1 ppm for the fathead minnow, *Pimephales promelas*; Wellens (1982) reports a 96 hour LC_{50} of 87.6 ppm for the Zebra fish, *Brachydanio rerio*; and Juhnke and Luedemann (1978) report an LC_{50} of 35 ppm, an LC_{100} of 80 ppm and an LC_0 of 10 ppm for the golden orfe, *Leucisucs idus melanotus*. In the himedaka fish, *Oryzias latipes*, Tonogai et al. (1982) determined the 48-hour median threshold limit (TLm) of 4-nitroaniline to be 50 ppm. Lysak and Marcinek (1972)

report the results of exposure of rainbow trout to 4-nitroaniline for 48 hours as follows: 100% mortality occurred at 85 ppm; 0% mortality at 18.3 ppm; and <100% mortality at 28-56 ppm.

Bringmann and Kuehn (1977) report an LC_{50} of 24 ppm for 4-nitroaniline in the invertebrate, *Daphnia magna*. In a later study, *Daphnia magna* were immobilized with a median effective concentration (EC_{50}) of 2.5 ppm (Bringmann and Kuehn, 1982). In the protozoans, *Entosiphon sulcatum* and *Uronema parduczi*, the "toxic threshold", (the concentration that decreases test populations by $\geq$ 5%), was 6.9 ppm (Bringmann, 1978) and 3.1 ppm (Bringmann and Kuehn, 1980a), respectively.

In the bluegreen and green algae, *Microcystis aeruginosa* and *Scenedesmus quadricauda*, Bringmann and Kuehn (1978) established toxic thresholds (the concentration that reduces population growth relative to controls after 8 days) of 0.35 ppm and 11 ppm 4-nitroaniline, respectively. Bringmann and Kuehn (1980b) also obtained a toxic threshold of 4 ppm in the bacteria, *Pseudomonas putida*.

Standards, Criteria and Guidelines

EPA Class C Carcinogen

Oral Slope Factor:	4×10^{-2} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	NA
Chronic Oral RfD:	3.0×10^{-4} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	3.0×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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N-NITROSODIPHENYLAMINE

Use

N-Nitrosodiphenylamine (NDPA) is a man-made compound used to retard the premature vulcanization of rubber materials in rubber producing processes. Production of NDPA in the United States was stopped in the early 1980s because substitute chemicals were found that were more efficient and less hazardous (ABDR, 1988). Currently, exposure to NDPA, either occupationally or in day-to-day life, is unlikely to occur. Occupational exposure in rubber-producing industries and in chemical-manufacturing industries prior to 1980 may have been significant (ATSDR, 1988).

Chemical and Physical Properties

MF:	$C_{12}H_{10}N_2O$	BP:	268.2°C
MW:	198.22	MP:	66.5°C
Sol.(water):	40 mg/l	VP:	0.1 mmHg at 25°C

Sol.(organics): acetone, ethanol, benzene and ethylene dichloride

Fate and Transport

NDPA is expected to be rapidly photolyzed in the atmosphere. The half-life of this compound is less than 1 day (reportedly 5-30 minutes) (Cupitt, 1980). In aquatic environments, NDPA is expected to either volatilize or transform photochemically (EPA, 1987). The calculated half-life of NDPA is estimated to be 11 hours (Lyman et al, 1982). NDPA may also adsorb to solids and sediments in aquatic media but bioaccumulation is thought to be negligible.

NDPA is not though to be highly mobile in soils. A half-life of 22 days has been noted, the study however, was conducted in the absence of sunlight so photolysis was not a factor (Mallik and Tesfai, 1981).

Pharmacokinetics

The pharmacokinetics of NDPA are unknown. Studies regarding absorption, inhalation, or ingestion of NDPA are considered to be insufficient (ATSDR, 1988).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Subchronic exposure in the diets of rats and mice to NDPA resulted in a 10 percent depression in body weight (NCI, 1979). Necroscopies of the test subjects revealed no significant systemic effects resulted from the exposure. In the same study, NCI reported that male rats fed a diet containing 1000 or 4000 ppm NDPA for 101 weeks exhibited a significant incidence of corneal opacity in the high dose group (15 of 50) Females exposed to the same concentrations exhibited similar effects. In addition, increased incidence of submucosal inflammation of the urinary bladder and epithelial hyperplasia in the bladder were also noted (NCI, 1979).

Teratogenic and Other Developmental Effects

Pertinent data regarding the developmental effects of NDPA were not located in the available literature.

Mutagenic Effects

Available studies do not indicate that NDPA is mutagenic to bacteria or *in vitro* hamster cells (Metsushima, 1981; Mishra et al., 1978).

Carcinogenic Effects

NCI (1979) noted that rats administered 1000 or 4000 ppm NDPA in their diets over a 101-week period exhibited a significant increase in bladder transitional cell carcinoma. The effects appeared to be dose-related.

Other studies of rats and mice exposed to NDPA have resulted in contradictory evidence of carcinogenicity. Administration of similar concentrations of NDPA by gavage in mice produced negative results (BRL, 1968). Dermal exposure of rats to NDPA also produced negative results (Iverson, 1980).

Standards, Criteria and Guidelines

EPA Class B2 carcinogen

Oral Slope Factor:	4.9×10^{-3} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	NA
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 4.9 µg/L
	Fish Consumption - 16.1 µg/L

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NITROPHENOLS

Use

There are three nitrophenol isomers: 2-nitrophenol, 3-nitrophenol, and 4-nitrophenol.

The main use of 2-nitrophenol is in the production of 2-aminophenol, 2-nitroanisole, and other dyestuffs (Matsuguma, 1967). 2-Aminophenol, the major single derivative of 2-nitrophenol, is used as an intermediate in a variety of industrial dye syntheses and as a commercial developer in photography (Farris, 1978). U.S. EPA (1985) reports that 2-nitrophenol is also used in the synthesis of other chemicals and as an analytical indicator.

3-Nitrophenol is used as an analytical indicator (Hawley, 1977) and as an intermediate in organic synthesis (U.S. EPA, 1985).

4-Nitrophenol is used mainly in the production of the pesticides, ethyl and methyl parathions (CMR, 1984). Other uses of 4-nitrophenol include intermediate applications in dye production, fungicides, photochemicals, pharmaceuticals, and leather preservatives (CMR, 1984; Matsuguma, 1967).

Chemical and Physical Properties

Chemical Formula: $NO_2C_6H_4OH$

MW: 139.11

VP: 2-nitrophenol: 1 mm Hg at 49.3°C
3-nitrophenol: 0.75 mm Hg at 20°C
4-nitrophenol: 0.75 mm Hg at 20°C

SG: 2-nitrophenol: 1.295 at 45°C
3-nitrophenol: 1.485 at 20°C
4-nitrophenol: 1.479 at 20°C

BP: 2-nitrophenol: 214-217°C
3-nitrophenol: 194°C
4-nitrophenol: 179°C

MP: 2-nitrophenol: 45°C
3-nitrophenol: 96°C
4-nitrophenol: 114°C

Sol. (water): 2-nitrophenol: 2100 ppm at 20°C
3-nitrophenol: 1.35/100 parts at 20°C
4-nitrophenol: 16,000 ppm at 25°C

Sol. (organics): alcohol, ether

Fate and Transport

U.S. EPA (1988) reports that much of the information on the fate and transport of nitrophenols is for 4-nitrophenol. It is assumed, however, that the environmental fate for all 3 isomers is similar (U.S. EPA, 1988). 4-Nitrophenol appears to be removed from the atmosphere by wet and

dry deposition although direct photolysis or reaction with photochemically generated hydroxyl radicals may also occur (U.S. EPA, 1985 and 1988). U.S. EPA (1985) reports the estimated half-life of nitrophenols due to rainfall as 3 weeks and the estimated half-life of the hydroxyl reaction of 2-nitrophenol as 9 days.

In aquatic systems, photolysis and biodegradation appear to be significant fate processes (U.S. EPA, 1985 and 1988). The observed photolysis half-life of 4-nitrophenol in aqueous solution is reported to vary from 16 hours to 5.7 days at pH 5, 6.7 days at pH 7, and 13.7 days at pH 11.5 (U.S. EPA, 1985). The relatively low vapor pressures of nitrophenols suggest that volatilization is not a significant fate process (U.S. EPA, 1988).

In soils, biodegradation and leaching appear to be major fate processes, although biodegradation may occur at such a rate as to prevent extensive leaching (U.S. EPA, 1985).

Pharmacokinetics

Data regarding the absorption of nitrophenols after oral administration or inhalation were not available in the literature reviewed. However, Snodgrass (1983) studied the absorption of 4-nitrophenol following dermal application. Dermal application of 357 µg of radioactive 4-nitrophenol to 3 male New Zealand rabbits over a 7-day period resulted in 53 percent of the dose remaining unabsorbed at the site of application at the end of the 7 days, 35 percent was recovered in the urine, and 0.53 percent in the feces. In a similar study with 3 male beagle dogs, 86 percent of the 714 µg applied remained unabsorbed at the site of application, 11 percent was recovered in the urine, and less than 0.5 percent in the feces.

Although studies focusing directly on the distribution of nitrophenols following absorption were not available in the literature reviewed, Snodgrass (1983) did not detect radioactivity in the liver, lung, kidney, spleen, heart, brain, adrenal gland, urinary bladder, muscle, bone, fat, thyroid gland, testes, bone marrow, or bile 7 days after dermal application of radioactive 4-nitrophenol in rabbits and dogs. No radioactivity was detected in these tissues following intravenous injection of 714 µg of radioactive 4-nitrophenol either.

Numerous studies cited in U.S. EPA (1988) indicate that 4-nitrophenol is metabolized into glucuronide and sulfate conjugates and Machida et al. (1982) reports detecting these metabolites within 1 minute after dosing with 1.6 - 8.0 mg/kg bw of 4-nitrophenol.

Numerous studies cited in U.S. EPA (1988) indicate that excretion of nitrophenols and their metabolites occurs mainly in the urine (75-99 percent) with less than 1 percent being excreted in the feces. Methods of administration of the nitrophenols included oral administration, interperitoneal injection, and intravenous injection.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

von Oettingen (1941) reports that acute administration of 4-nitrophenol in cats resulted in methemoglobin formation. Smith et al. (1967) reports the same result in mice. Grant (1959), however, did not observe methemoglobin formation following oral administration of 3- or 4-nitrophenol to rats. The formation of methemoglobin may be dependent on the nitroreductase activity of the organism which is not extensive in most species (U.S. EPA, 1980 and 1985). Vitamin C-deficient guinea pigs developed cataracts when treated with 4-nitrophenol (Ogino and Yasukura, 1957) although it is unclear whether the nitrophenol or the lack of vitamin C produced the cataracts (U.S. EPA, 1980).

U.S. EPA (1988) states that several investigators have studied the possibility that the different isomers of nitrophenol act similarly. However, Cameron (1958) found substantial differences in the effect of the isomers on the metabolic activity of rats. Exposure to 4-nitrophenol increased carbon dioxide output, exposure to 3-nitrophenol depressed oxygen consumption, and exposure to 2-nitrophenol had no effect on either of these parameters.

U.S. EPA (1988) reports that results from several studies indicate that nitrophenols may act directly on cell membranes.

Makhinyas (1969) reports that administration of 2-, 3-, or 4-nitrophenol produced gastritis, enteritis, colitis, hepatitis, neuritis, splenic hyperplasia, and inhibited oxidation processes in an unspecified species.

Teratogenic and Development Effects

Plasterer et al. (1985) reports that oral administration of 400 mg/kg 4-nitrophenol, from days 7-14 of gestation, to groups of 10 mice had no adverse effects on indices of fetal survival, birth weights, or gross malformations. However, maternal survival and weight gain were significantly decreased.

Mutagenic Effects

The U.S. EPA (1985, 1988) reports that several studies have found that nitrophenols do not increase the frequency of reverse mutations in either *S. typhimurium* or *E. coli.* Fabrig (1974) reports that 4-nitrophenol did not increase the frequency of forward mutations in *E. coli* or *Serratia marcescens*, or recessive

lethal mutations in *Drosophila melanogaster*. However, Fabrig (1974) also reports an increase in the frequency of mitotic gene conversion in *saccharomyces cerevisiae* with 4-nitrophenol. This is most likely indicative of DNA single strand breaks (U.S. EPA, 1988). Poirier et al. (1975) reports that 4-nitrophenol inhibited both repair and replicative DNA synthesis in confluent human fibroblast cultures.

Carcinogenic Effects

Data regarding the carcinogenicity of nitrophenols is limited. Boutwell and Bosch (1959) found no increase in the incidence of any tumor type following dermal application of 25 µl of 2- or 4-nitrophenol in dioxane, twice weekly for 12 weeks on female mice. The U.S. EPA (1985), however, considered this study inadequate to assess the carcinogenicity of nitrophenols.

Ecotoxicity

The U.S. EPA (1980) reports that the available data indicate that acute toxicity of nitrophenols to fresh water aquatic life occurs at concentrations as low as 230 µg/l. Toxicity to one species of algae occurs at concentrations as low as 150 µg/l. In salt water, acute toxicity to aquatic life occurs at concentrations as low as 4850 µg/l. It should be noted that toxic concentrations may be even lower in species more sensitive than those tested (U.S. EPA, 1980).

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption: 13.4 µg/l
	Fish Consumption: 765 µg/l

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PENTACHLOROPHENOL

Use

Pentachlorophenol (PCP) is a chlorinated hydrocarbon used primarily as a fungicide, bactericide and slimicide. It's primary use as such is in wood preservatives and stains (Sittig, 1991). It has been recently been used as a insecticide molluscicide, herbicide because of its biologically toxic properties (ACGIH, 1984).

Chemical and Physical Properties

Chemical Formula: C_6Cl_5OH

MW: 266.32
SG: 1.978 at 20°C
FP: not combustible
Sol. (water): 14 mg/l at 20°C
Sol. (organics): alcohols, ether, benzene

BP: 309°C (decomposes only)
MP: 190°C
VP: 1.1×10^{-4} mmHg at 20°C

Fate and Transport

PCP is rapidly degraded in air, soil and water, usually through photolysis. PCP absorption in soils is dependent on the pH of the soil. At high pHs, PCP will leach into the soil whereas at low pHs, PCP will be absorbed by the soil. It has been detected at approximately 4.2 percent of the 2,783 hazardous waste sites that have been sampled (ATSDR, 1989). Volatilization of PCP does not ostensibly occur under normal environmental conditions.

Pharmacokinetics

The most significant routes of exposure to PCP are probably through inhalation and dermal contact. PCP is readily absorbed by into bronchial tissues. Cassarett et al. (1969) exposed 2 workers in an enclosed processing room for 45 minutes. Levels of 230 and 432 ng/l PCP were found in the worker's urine. Absorption rates of 88 and 76 percent were calculated for the two subjects (Cassarett et al., 1969).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The majority of deaths caused by exposure to pentachlorophenol have occurred through dermal contact. Acute contact for periods as short as 3 days have been

known to cause death (Menon). It was noted that the organs most affected were generally the liver, kidney, skin, blood, lungs, central nervous system, and gastrointestinal tract.

Superficial exposure to PCP can cause local irritation and, in a limited number of people, allergic reactions. Poisoning is characterized by profuse swearing, fever, weight loss and complaints of abdominal cramps (EPA 1985).

Chronic exposure to PCP has not been proven to cause any toxic effects in humans. Many of the toxic effects may be caused by other compounds, such as polychlorinated dibenzo dioxin, contained in technical grade PCP. Studies performed with pure grade PCP result in less severe effects.

Teratogenic and Other Developmental Effects

No data regarding the teratogenicity of pentachlorophenol were available in the literature reviewed.

Mutagenic Effects

No data regarding the mutagenicity of pentachlorophenol were available in the literature reviewed.

Carcinogenic Effects

No data regarding the carcinogenicity of pentachlorophenol were available in the literature reviewed.

Ecotoxicity

Acute exposure of experimental mammals to PCP causes an elevations in the respiration and body temperature of the animal and eventually death. No ill effects were noted as a result of chronic exposure to PCP in laboratory animals. Carcinogen and mutagen studies performed on rats revealed negative results, even when given doses large enough to cause immediate toxic effects (EPA 1985).

High levels of PCP have been found to be acutely toxic to freshwater aquatic organisms, including some plants. This has not been studied in detail.

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	1.2×10^{-1} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	NA
Chronic Oral RfD:	3.0×10^{-2} mg/kg/day
Chronic Inhalation RfD:	currently under review by EPA
Subchronic Oral RfD:	3×10^{-2} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	1.0 µg/l
AWQC:	Water and Fish Consumption - 1.01 mg/l
	Fish Consumption - NA

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PHENOL

Use

Phenol is used in the production of fertilizer, explosives, coke, paints, paint remover, rubber, asbestos, wood preservatives, resins, and pharmaceutical products. It is also widely used as a disinfectant in the petroleum, leather, paper, soap, toymaking, and tanning industries (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: C_6H_5OH

MW:	94.11	BP:	181.75°C
SG:	1.057 or 20°C	MP:	43°C
FP:	85°C	VP:	0.3513 mmHg at 25°C

Sol. (water): 93,000 µg/L at 25°C

Sol. (organics): alcohol, chloroform, ether, carbon disulfide, hot benzene, carbon tetrachloride.

Fate and Transport

In surface waters, phenol is expected to photo-oxidize and biodegrade. Volatilization may be a feasible fate but, since phenol would be rapidly photo-oxidized, atmospheric transport is expected to be limited (ICF, 1985). Microorganisms and at least one fish species are known to biotransform phenol.

In soils, biodegradation would be phenol's predominant fate. Phenol does not sorb to soils and is not highly water soluble, so it is expected to be mobile in soils. However, transport to ground water is not expected to occur because of the speed with which phenol biodegrades. Exceptions occur when large quantity spills kill off the degrading microbial populations (EPA, 1984).

Pharmacokinetics

Pitrowski (1971) reported that humans absorb 60 - 80 percent of an inhaled dose of phenol over the course of exposure. Approximately 99 percent of the inhaled dose was excreted within 16 hours post-treatment. Absorption of phenol appears to occur in the stomach and bowel (Gosselin, 1976). Phenol is metabolized primarily to sulfuric and glucuronic acids. Small quantities are metabolized to hydroquinone. Phenol vapors can be absorbed through the skin and alveoli (Clayton and Clayton, 1981).

Human Toxicity

Noncarcinogenic

Systemic Effects

Phenol is known to have caused fatality to humans upon acute oral exposure (Clayton and Clayton, 1981). Roughly 50 percent of accidental ingestions of greater than 1 gram of phenol have resulted in death in humans (Clayton and Clayton, 1981). The central nervous system is effected rapidly upon ingestion of phenolic compounds. Hypothermia, vasoconstriction, and respiratory arrest, often followed by coma and death, are common symptoms of phenol ingestion (Gosselin, 1976).

Dow Chemical (1976) reported that liver and kidney damage occurred in rats exposed to greater than 50 mg phenol/kg/day.

Much data exists regarding the chronic effects of phenol exposure to humans because of its use as an anaesthetic (as carbolic acid) in the late 19th and early 20th centuries. One study reported that common symptoms of chronic phenol poisoning include vomiting, digestive disorders, anorexia, eruptions on the skin, and nervous disorders (Clayton and Clayton, 1981). To the contrary, Kobert (1906) reported that many doctors exposed daily to phenol exhibited no toxic symptoms (Clayton and Clayton, 1981). This can most likely be attributed to the fact that phenol is completely removed from the body within 48 hours.

Teratogenic and Other Developmental Effects

The principle study used to derive the reference dose (RfD) for phenol was a teratological evaluation study of phenol in rats and mice. This study revealed a highly significant reduction in fetal body weights in the high dose group (120 mg/kg/day); the NOAEL was 60 mg/kg/day (NTP, 1983).

Heller and Pursell (1938) reported that rats exposed via drinking water, to levels of phenol between 100 and 12,000 ppm for various time periods, exhibited adverse developmental effects. At levels greater than 7,000 ppm, stunted growth was noted; levels greater than 8,000 ppm resulted in the death of numerous young; and at levels of 12,000 ppm, no reproduction occurred.

Mutagenic Effects

Mutagenic activity has been noted to occur in *E. coli* exposed to concentrations between 0.1 and 0.2 percent. It has also induced mutations in the gonads on *Drosophila melanogaster* (EPA, 1984).

Carcinogenic Effects

Phenol appears to induce local tumors in mice exposed cutaneously. In one strain of mice known for its sensitivity to carcinogens, however, the tumors reported to be cancerous, all others were benign (ICF, 1985). NCI reported that the ingestion of phenolic compounds does not induce carcinogenic effects in rats and mice (ICF, 1985).

No reports of phenol's carcinogenic effects to humans were located in the available literature.

Ecotoxicity

Phenol has acutely toxic effects on freshwater species at levels between 5,000 µg/L (juvenile rainbow trout) and 67,500 µg/L (fathead minnow) (ICF, 1985). Phenol does not appear to bioaccumulate in fish.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	6×10^{-1} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	6×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 3.5 mg/l Fish Consumption - 4600 mg/L (recalculated)

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POLYCYCLIC AROMATIC HYDROCARBONS

Background

Polycyclic Aromatic Hydrocarbons (PAHs) constitute a class of materials which are characterized as containing more than one benzene ring. Because of the similarities between them, PAHs have been summarized in one toxicity profile. Discussions of specific PAHs are provided in cases where available literature exists. Otherwise, PAHs are discussed in general terms. The most common and the most hazardous PAH is benzo (a) pyrene (BaP). As a result, most of the information located in the available literature deals with BaP.

Use

As a class, PAHs are used industrially in the production of automobile tires, rubber stoppers, dyes, and glass and can be found in yeasts, whiskeys, dried prunes, and cigarette smoke (ICF, 1985). PAHs are often found as byproducts to the refining processes of petroleum, shale, coal, and coke.

Chemical and Physical Properties

Summarized in Table 1.

Fate and Transport

In general, PAHs are expected to exist as vapor and particulates in the atmosphere. Once in the atmosphere, PAHs may be removed through photochemical reactions, chemical reactions, or by wet and dry deposition. In aquatic media, PAHs are expected to volatilize, react photochemically, and be degraded microbially. In high water and wind flow conditions, volatilization would occur readily. In water, PAHs would adsorb to organic matter and would most likely fall out of the water column (EPA, 1984a).

In soils, PAHs are subject to microbial degradation and adsorption. Because of their affinity to organic matter, PAHs are not expected to be highly mobile in soils, therefore, leaching to ground water is not considered to be a significant fate process.

Pharmacokinetics

Although few studies have been performed on human ingestion of PAHs, it is thought that they would be absorbed readily in the gastrointestinal tract. Benzo (a) pyrene (BaP), chrysene, and benzo (a) anthracene (BaA), three of the more common PAHs, are reported to transport passively across the gastrointestinal mucosa (EPA, 1984a). Chang (1943) noted that rats given BaP by

gavage absorbed approximately 50 percent of the administered dose. Certain PAHs require metabolic activation by specific enzymatic systems in order to acquire carcinogenic properties. PAHs and their metabolites are excreted through the feces and through the hepatobiliary system. There is little evidence that PAHs bioaccumulate extensively (EPA, 1984a).

Human Toxicity

Acenapthene

Knobloch, et al. (1969) reported that, when administered orally to rats, acenapthene causes changes in renal function, lowers body weight and causes unspecified changes in the peripheral vascular system (EPA, 1984f). Mild morphological damage to the kidneys and liver were also noted. U.S. EPA (1989a) reports liver weight changes accompanied by microscopic alterations in mid- to high-dose mice exposed to acenapthene ranging in concentration form 0 to 700 mg/kg/day. Nonspecific pneumonia was noted by Reshetyuk et al. (1970) in rats exposed to 12 mg/m^3 acenapthene by inhalation for 4 hours/day, 6 days/week for 5 months (EPA, 1984f).

Acenapthene has not been shown to be mutagenic; however, it is known to cause changes in the DNA content of a number of plant and microbial species. These changes are a result of disruptions of the spindle mechanisms during mitosis (ICF, 1985).

At high exposure levels, acenapthene is known to cause liver and kidney damage, but is not known to be carcinogenic (ICF, 1985).

Acenaphthylene

The U.S. EPA is currently reviewing the noncarcinogenic risk assessment of this substance (IRIS). No data relative to the toxicity of this chemical were found in the literature reviewed.

Kaden et al. (1979) found that acenaphthylene (1mM) yielded positive results in a *Salmonella typhimurium* forward mutation assay. However, Bos et al. (1988) reported negative results in *S. typhimurium* strains TA98 and TA100 in the presence of hepatic homogenates.

Cook (1932) observed no carcinogenic effect in a lifetime study of the effect of dermally introduced 0.25 percent acenaphthylene in mice. Survival, however, was only 65 percent at 6 months and 35 percent at 12 months.

Anthracene

Chronic exposure to anthracene is thought to cause dermatitis, hyperkeratoses, and other skin disorders in workers (ICF, 1985). Numerous studies of chronic and acute exposure

of laboratory animals to anthracene have suggested that it does not cause any systemic toxic effects (i.e., U.S. EPA, 1989b).

Anthracene has been shown to cause reproductive effects in mice given a single oral dose during the last week of gestation. A lower survival rate was seen in experimental mice than in controls (IARC, 1983).

In twenty experiments on the mutagenicity of anthracene, very few have resulted in positive effects (IARC, 1983); therefore, not enough evidence is available to consider anthracene a mutagen. There are no epidemiologic studies available that suggest anthracene is carcinogenic to humans. In studies of the effects of subcutaneous injections of anthracene to laboratory animals, it was noted to cause local tumors. The carcinogenic effects of subcutaneous injections of anthracene appear to be enhanced by ultra-violet light. Schmahl (1955), however, found no incidence of tumors following exposure of rats to 4.5 g anthracene/rat over 78 weeks.

However, this evidence was determined to be inadequate in proving anthracene's carcinogenicity (IARC, 1983).

Benzo (a) Anthracene

Benzo (a) anthracene (BaA) is known to cause skin disorders, such as hyperplasia and hyperkeratosis, in workers exposed occupationally (ICF, 1985). Cutaneous exposure to BaA causes destruction of the sebaceous glands of laboratory mice and, when injected repeatedly, BaA produces gross changes in the lymphoid tissues of mice and rats (ICF, 1985).

It is also known that many carcinogenic PAHs, such as BaA, cause immunosuppressive effects, although studies on BaA have not been conclusive (ICF, 1985).

BaA is known to be mutagenic in *Salmonella typhimurium* and *Drosophila melanogaster.* It is also known to cause sister-chromatid-exchange in cultured mammalian cells.

Several studies indicate that BaA is carcinogenic to laboratory animals. Oral administrations and sub-cutaneous injections have resulted in statistically significant increases in tumors and adenomas (Klein, 1963; IARC, 1983).

Benzo (a) Pyrene

From laboratory studies performed on mice, it appears as though BaP's toxicity to organisms is dependent upon the constitution of a specific gene locus. The particular locus determines whether or not aryl-hydrocarbon-hydroxylase, an enzyme which alters the chemical makeup of aromatic hydrocarbons, is easily released (induced) into the body (EPA, 1985). Those animals that cannot easily induce the release of aryl hydrocarbon

hydroxylase are more susceptible to BaP's toxic effects. Robinson, et al. (1975) administered 120 mg/kg-bw BaP in food to "poorly inducible" and "easily inducible" mice. The "poorly inducible" mice developed aplastic anemia and died within 4 weeks whereas the "easily inducible" mice remained healthy for at least 6 months.

In a study a carried out by Rigdon and Rennels (1964), only one of seven pregnant female rats carried viable fetuses to term, after having been fed a diet containing BaP at a level of 50 mg/kg/day for up to 3.5 months. Of four pups delivered, two were stillborn, one of which was grossly malformed. A third was killed for observational purposes, while the fourth died of starvation because it did not appear to be lactating.

In a teratogenicity and reproduction study, Rigdon and Neal (1965) fed diets containing BaP at a level of 0, 250, 500, or 1,000 mg/kg to male and female mice over various time spans during mating, gestation, and lactation. No apparent reproductive, teratogenic, embryotoxic or fetotoxic effects were observed.

MacKenzie and Angevine (1981) administered BaP orally at a level of 10 mg/kg/bw to CD-1 mice during pregnancy. There was no effect on fetal body weight; however, reduced fertility and reproductive capacity were observed in the offspring.
BaP has been used as a positive control in a variety of short-term tests. It has yielded positive results in assays for bacterial mutation, mutation in *Drosophila melanogaster*; DNA binding, DNA repair, sister chromatid exchange (SCE), chromosomal aberration, point mutation and transformation in mammalian cells in culture; and *in vivo*, including DNA binding, SCE, chromosomal aberration, sperm abnormality and the specific locus (spot) test (IARC, 1982; deSerres and Ashby, 1981; Hollstein and McCann, 1979).

PAH mixtures containing BaP have been shown to induce lung cancer in humans as a result of chronic exposure to cigarette smoke, roofing tar, and coke oven emissions (IRIS). It is impossible to conclude from these studies however, that BaP is the responsible agent.

Cottini and Mazzone (1939) applied a 1 percent solution of BaP to the skin of 26 patients. The skin of the patients developed regressive verrucae, reversible and apparently benign cysts that are thought to represent the early stages of neoplasia.

BaP is known to be carcinogenic to mice when exposed subcutaneously. Neal and Rigdon (1967) noted a dose-response relationship in the incidence of stomach tumors in male and female CFW-Swiss mice treated orally with 1-250 ppm BaP for 197 days. Individuals treated with greater than 20 ppm doses exhibited a significant increase in stomach carcinomas and papillomas. Mice treated with 250 ppm BaP exhibited an increase in the incidence of lung adenoma and leukemia.

In an inhalation study, Thyssen, et al. (1981) exposed hamsters to 2.2, 9.5 or 45 mg/m^3 BaP for 4.5 hours/day for 10 weeks and 3 hours/day 7 days/week for up to 675 days. Animals exposed to 9.5 mg/m^3 developed tumors of the nasal cavity, larynx trachea and pharynx. Animals exposed to 45 mg/m^3 BaP developed a significant number of tumors in the respiratory tract and upper digestive tract.

Benzo (b) Fluoranthene

No data concerning the systemic effects of benzo (b) fluoranthene (BbF) on humans or laboratory animals were located in the available literature.

One study has demonstrated that BbF caused chromosomal aberrations in the bone-marrow cells of Chinese hamsters (IARC, 1983). In this study, hamsters were given two doses of 450 mg BbF/kg-bw. In separate studies, unspecified mutations in *Salmonella typhimurium* cultures were noted when exposed to 100 µg BbF (IARC, 1983).

BbF is known to be carcinogenic to laboratory mice and rats. 3-month old female Osborne-Mendel rats exposed to BbF through lung implants illustrated a dose-related increase in the incidence of epidermoid carcinoma and pleomorphic sarcomas in the lung and thorax (Deutsch-Wenzel et al., 1983). A 0.5 percent solution of BbF produced papilloma in 100 percent of laboratory mice that were painted three times per week (IARC, 1983). In one study, researchers were able to induce local sarcoma in 18 of 24 mice that were subcutaneously injected with 0.6 mg BbF. The lowest carcinogenic dose of BbF painted on mice was noted to be a 0.01 percent solution (IARC, 1983).

No experiments concerning the carcinogenic effects of BbF on humans were located in the available literature.

Benzo (k) Fluoranthene

No data concerning the systemic effects of benzo (k) fluoranthene (BkF) on humans or laboratory animals were located in the available literature. BkF was reported to be mutagenic in bacteria such as *Salmonella typhimurium* (IARC, 1983).

The International Agency for Research on Cancer (IARC) has determined that there is sufficient evidence to prove that BkF is carcinogenic to laboratory animals. Tumors were noted in 69 percent of NMRI mice treated with 9.2 mg BkF/kg-bw. In this study, 3.4, 5.6 or 9.2 mg BkF were applied to the mice's skin. In the lowest test group, 8 of 34 individuals exhibited local tumors (IARC, 1983).

When injected into the pulmonary tissues of rats, BkF caused squamous cell carcinomas (IARC, 1983). Female Osborne-Mendel rats exposed to BkF through lung implants illustrated a dose-related increase in the incidence of epidermoid carcinomas in the lung and thorax (Deutsch-Wenzel, et. al., 1983).

Benzo (g,h,i) Perylene

No data concerning the systemic effects of benzo (g,h,i) perylene (B(g,h,i)P) on humans or laboratory animals were located in the available literature. IARC states that there is inadequate evidence to prove that B(g,h,i)P is toxic when exposure is short-term.

B(g,h,i)P was shown to be mutagenic to *Salmonella typhimurium* when administered in various doses (IARC, 1983).

In seven studies evaluated by IARC, B(g,h,i)P caused no visible carcinogenic effects. The tests included five skin application assays, one intrapulmonary injection study, and one co-administration study. In the latter study, B(g,h,i)P was administered with BaP. A higher number of skin tumors was noted in the test group than in the group administered BaP alone (IARC, 1983).

There is not sufficient evidence to classify B(g,h,i)P as carcinogenic to humans or laboratory animals (IARC, 1983).

Chrysene

Chrysene's toxic effects to humans and animals have not been studied extensively. It is expected that chrysene causes damage to epidermal tissues in workers exposed daily. Although not specific to chrysene, numerous studies indicate that PAHs cause immunorepressive effects (IARC, 1983).

Chrysene was shown to be mutagenic to *Salmonella typhimurium* when administered at doses of 10 mg/plate. Another study concluded that chrysene causes embryonic cell transformations in Syrian hamsters. Chrysene is known to cause sister-chromatid-exchange in Chinese hamsters and aberrations in the oocyte development of laboratory mice (IARC, 1983).

Chrysene is thought to be weakly carcinogenic to laboratory animals. It does not appear to be locally or systemically carcinogenic to laboratory animals when exposed epidermally although some studies provide evidence to the contrary. A number of these studies were ignored due to contamination to stock by methylchrysenes (IARC, 1983). Perinatal and subcutaneous administrations have resulted in similar effects and conclusions.

Although some studies have indicated chrysene to be carcinogenic to laboratory animals (Wislocki et al., 1986), IARC has determined that only limited evidence of chrysene's carcinogenicity exists.

Dibenzo (a,h) Anthracene

Researchers reported a decreased growth rate in young rats when exposed to between 3 and 90 mg/kg bw dibenzo (a,h) anthracene (D(a,h)A), (IARC, 1983). No other evidence of systemic or local noncarcinogenic toxic effects were located in the available literature.D(a,h)A was found to be mutagenic to a number of cultured and *in vivo* cells. D(a,h)A was highly mutagenic to *Salmonella typhimurium.* It also induced unscheduled DNA syntheses in the presence of an exogenous metabolic system in cultured mammalian cells. D(a,h)A was found to be embryotoxic to rats when administered in high doses (IARC, 1983).

D(a,h)A has produced tumors in rats, guinea pigs, mice, frogs, pigeons, and chickens (Snell and Stewart; 1962, 1963). Carcinogenic affects, both local and systemic, have been noted as a result of oral, intratracheal, and cutaneous applications (IARC, 1983).

Fluoranthene

Male and female CD-1 mice (20/sex/group) were exposed to 0, 125, 250, or 500 mg/kg/day fluoranthene by gavage for 13 weeks (U.S. EPA, 1988a). Mice exhibited increased food consumption and body weight gain at the highest dose. Increased SGPT values and increased absolute and relative liver weights occurred at 250 and 500 mg/kg/day. Compound-related microscopic liver lesions were observed in 65 and 87.5 percent of the mid- and high-dose mice, respectively.

Fluoranthene was found to be mutagenic in *Salmonella typhimurium* and *in vitro* human lymphoblastoid cells in the presence of an exogenous metabolic system (IARC, 1983). There have been no studies done that indicate fluoranthene to be carcinogenic to humans or laboratory animals. Of eight studies reviewed by IARC, none provided sufficient evidence to conclude that fluoranthene is carcinogenic. However, one study noted twice as many tumors in mice administered fluoranthene in conjunction with BaP than in mice administered BaP alone (IARC, 1983).

Fluorene

CD-1 mice (25/sex/group) were exposed to 0, 125, 250, or 500 mg/kg/day fluorene by gavage for 13 weeks (U.S. EPA, 1989c). Increased spleen, liver, and kidney weights were observed at the high doses. Other systemic effects included a decreasing trend in BUN and an increasing trend in serum bilirubin.

Fluorene does not appear to be mutagenic, teratogenic, or embryotoxic to laboratory animals. Three studies were reviewed by IARC, all were inconclusive as to the reproductive effects of fluorene (IARC, 1983).

Fluorene did not cause cancer in laboratory animals from skin applications, subcutaneous injections, or oral administrations (IARC, 1983). Due to insufficient studies, there is inadequate evidence to evaluate the carcinogenicity of fluorene (IARC, 1983).

Indeno (1,2,3-cd) Pyrene

No data regarding the systemic, mutagenic, teratogenic, or developmental effects of indeno (1,2,3-cd) pyrene (IP) were located in the available literature.

IP is carcinogenic to laboratory mice when administered by skin painting at a dose of 250 µg. Researchers noted that doses of 0.01 and 0.05 percent IP produced no tumors. A dose of 0.1 percent IP produced a total of 6 papillomas and 3 carcinomas in 20 mice. Seven papillomas and five carcinomas were noted in 20 mice painted with 0.5 percent IP. The same study demonstrated that 10 paintings at two-day intervals, resulting in a total dose of 250 mg initiated skin carcinogenesis (IARC, 1983).

When administered subcutaneously to mice, 0.6 mg IP given at one-month intervals, produced 10 sarcomas in 14 male mice and 1 sarcoma in 14 female mice (IARC, 1983).

In a lung implantation study (Deutsch-Wenzel et al., 1983), IP produced epidermoid carcinomas.

2-Methylnapthalene

No data were located in the available literature.

Naphthalene

Naphthalene appears to effect ocular function in humans, rats, and rabbits. Ghetti and Mariani (1956) reported that 8 of 21 workers exposed to an unspecified concentration of naphthalene in a dye-manufacturing process developed cataracts. All of these workers were less than 50 years of age. Fitzhugh and Buschke (1949) observed cataracts in young rats exposed to 2 percent naphthalene by ingestion for 60 days (approximately 1 g/kg bw/day). Ghetti and Mariani (1956) noted similar effects in rabbits.

Naphthalene is known to be fetotoxic because of its ability to cross the placental wall (EPA, 1984). It is also known to cause DNA damage in mice (ICF, 1985). The offspring of rats injected with unspecified amounts of naphthalene displayed retarded cranial and heart development (ICF, 1985).

Wolf (1976) reported that 6 of 15 workers exposed, via inhalation, to naphthalene develop laryngeal carcinomas and neoplasms of the pylorus and cecum. A study on the effects to rats of subcutaneously injected naphthalene concluded with negative results (Schmahl,

1955). The rats were injected with either 10 or 0.82 g naphthalene for an unspecified amount of time. No tumors were noted. (EPA, 1984b).

Phenanthrene

No data regarding the systemic effects of phenanthrene to humans or laboratory animals were located in the available literature.

The majority of the studies concerning the developmental effects of phenanthrene concluded with negative results. One study reported that *Salmonella typhimurium* mutated when exposed to 12 mg phenanthrene (IARC, 1983). Abnormally high concentrations of exogenous metabolites were introduced into the culture before mutations were seen. In two other experiments, phenanthrene was reported to induce mutations *in vitro* human cells and *in vivo* hamster cells. These studies do not provide enough evidence to classify phenanthrene as a mutagenic compound (IARC, 1983).

Experiments indicate that phenanthrene is not carcinogenic to laboratory animals (Higgins and Yang, 1962). No case studies of human exposure to phenanthrene were located. Mice and rats were exposed to phenanthrene via painting, subcutaneous injections, intraperitoneal injections, and ingestion. None of the studies resulted in the induction of tumors (IARC, 1983).

Pyrene

Cd-1 mice were exposed to 0, 75, 125, or 250 mg/kg/day pyrene by gavage for 13 weeks (U.S. EPA, 1989d). Nephropathy and reduced relative and absolute kidney weights were observed in the high dose groups.

It was noted in one study of the effects of pyrene exposure that the growth of young rats was inhibited when fed 2000 mg pyrene/kg/day for 100 days. In the same study, it was noted that the rats' livers were enlarged after prolonged exposure (IARC, 1983). No toxic effects to humans or animals resulting from pyrene exposure were noted in the available studies, although one researcher reported an LD_{50} for mice of 678 mg pyrene/kg-bw for 4 days (IARC, 1983). Pyrene induced unscheduled DNA synthesis in cultured rat hepatocytes (EPA, 1984d) and cultured human fibroblast cells (IARC, 1983). It induced sister-chromatid-exchange in Syrian hamster embryonic cells in one instance and, in another, it mutated *Salmonella typhimurium* cultures (IARC, 1983).

The carcinogenic effects of pyrene on laboratory animals have been studied extensively. Oral, inhalation, injection, and topical studies have all been performed and have all concluded that pyrene is noncarcinogenic (EPA, 1984d). Pyrene did not initiate tumors in mouse skin, although it did enhance the carcinogenic effects of benzo (a) pyrene when co-applied. Evidence regarding the carcinogenicity of intratracheal administration were considered inadequate for evaluation (IARC, 1983).

Ecotoxicity

The ecotoxic effects of PAHs have not been widely studied. It appears as though the effects of PAHs on aquatic organisms are more variable than the effects on humans.

Acenapthene resulted in 96-hour LC_{50} values of 970 and 2,230 mg/l for mysid shrimp and sheepshead minnows, respectively. Two freshwater species subjected to acenapthene exposure displayed EC_{50} values of 41,200 and 1,700 mg/l (*Daphnia magna* and bluegill, respectively) (ICF, 1985).

Fluoranthene appears to be less toxic to freshwater species than does acenapthene. The 96-hour LC_{50} value for bluegill was 3,970 mg/l and the 48-hour EC_{50} value for *Daphnia magna* was 325,000 mg/l. The 96-hour LC_{50} value for mysid shrimp, a saltwater species, was 40 mg/l, significantly lower than the value for acenapthene (ICF, 1985). Fluoranthene is known to bioaccumulate but, to what extent is unknown.

The medium effect concentration of naphthalene for freshwater species was reported to be greater than 2,300 mg/l. Acute values for saltwater species (polychaetes, oysters, shrimp) are reported to be greater than 2,350 mg/l (ICF, 1985).

Standards Criteria and Guidelines

Summarized in Table 2.

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2,4,5 TRICHLOROPHENOL

Use

2,4,5 Trichlorophenol is used to produce defoliants and other related products. It is also used as a fungicide, antimildew and preservative agent, algicide and bactericide.

Chemical and Physical Properties

Chemical Formula: $C_6H_2Cl_3OH$

MW: 197.5
SG: 1.678 at 25°C

BP: 253°C
MP: 68-70.5°C
VP: 1 mmHg at 72.0°C

Sol. (water): 12 g/liter at 25°C
Sol. (organics): alcohol, organic solvents, ligroin

Fate and Transport

The dominant fate of this compound is expected to be photodecomposition in the atmosphere (HEA, 1984). Photodecomposition is also expected to play a major role in aquatic media; this compound may be resistant to microbial degradation (EPA, 1980a).

2,4,5 Trichlorophenol may have significant mobility in sandy soils. The biodegradation and solubility of this compound facilitates leaching (HEA, 1984).

Pharmacokinetics

Routes of absorption include inhalation, skin absorption, ingestion and eye contact (Sittig, 1991).

Dougherty and Piotrowska (1976) suggested that chlorophenols as a group tend to be eliminated rapidly through the urine.

Human Toxicity

NonCarcinogenic

Systemic Effects

Symptoms include skin, eye and nose irritation; sweating; thirst; nausea; diarrhea; abdominal pain; increase followed by decrease in respiratory rate and urinary output; and fever. Lung; liver and kidney damage; collapse; and coma are possible toxic effects (Sittig, 1991).

Rats treated orally 18 times with doses ranging from 30 to 1,000 mg/kg over 24 days did not show any adverse health effects. Rabbits, on the other hand, did show slight renal lesions when oral dosages of 10 to 500 mg/kg were administered for 28 days. Liver lesions were also observed in the rabbits receiving the 500 mg/kg dosage. However, when the daily dose was 300 and 1,000 mg/kg and extended to 98 days, the rats did show dose related liver and kidney effects (McCollister et al., 1961).

It is important to note that the technical grade form of this compound is contaminated with polychlorinated dibenzo-p-dioxins, some of which have been shown to induce adverse health effects in animals (ICF, 1985).

Teratogenic and Developmental Effects

No information on this topic was found in the literature.

Mutagenic Effects

2,4,5 Trichlorophenol has given negative results in Ames mutagenicity assays, but it has been found to promote the formation of benign tumors on the skin of mice (ICF, 1985).

Carcinogenic Effects

No information on this topic was found in the literature.

Ecotoxicity

2,4,5 Trichlorophenol is acutely toxic to mysid shrimp and sheepshead minnow after 96 hour exposures to 3,830 and 1,660 µg/liter concentrations, respectively (ICF, 1985).

Standards, Criteria, and Guidelines

Unclassified by EPA as to carcinogenicity.

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	1.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	1.0×10^{0} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 2600 µg/L Fish Consumption - NA

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TABLE 1. CHEMICAL AND PHYSICAL PROPERTIES OF PAHs (U.S. EPA, 1986)

Compound	Chemical Formula	MW	SP. GR. (at 20°C)	BP (°C)	MP (°C)	VP (mmHg at 20°C)	Solubility (at 25°C)
Acenapthene	$C_{12}H_{10}$	154.21	1.225 at 0°C	279	96.2	$1.55x10^{-3}$	water: 3.42 mg/l organics: ethanol, toluene, chloroform, benzene
Acenaphthylene	$C_{12}H_8$	152.21			92.0*	$1x\ 10^{-3}$	water: 3.93 mg/1
Anthracene	$C_{14}H_{10}$	178.2	1.25	342	218	$1.95x10^{-4}$	water: 0.0446 mg/kg organics: benzene, chloroform, methanol
Benzo(a)anthracene	$C_{18}H_{12}$	228.30		435	167.0	2.2x10 at 20°C	water: 9.4 µg/kg organics: alcohol, ether, acetone, benzene,
Benzo(a)pyrene	$C_{20}H_{12}$	252.30		311	179.15	$5.6x10^{-9}$	water: 1.2 µg/kg organics: most
Benzo(b)fluoranthene	$C_{20}H_{12}$	252.3			168.3	$5.0x10^{-7}$	water: 0.014 mg/l organics: benzene, acetone
Benzo(k)fluoranthene	$C_{20}H_{12}$	252.3		480	215.7	$5.1x10^{-7}$	water: 0.0043 mg/l organics: acetic acid, benzene, ethanol
Benzo(g,h,i)perylene	$C_{22}H_{12}$	276.30			278.3	$1.03x10^{-10}$	water: 0.7 µg/kg organics: benzene, acetone
Dibenzo(a,h)anthracene	$C_{22}H_{14}$	278.4			266.6	$1.1x10^{-10}$	water: insoluble organics: benzene, toluene, xylene, oils

(Continued)

TABLE 1. (Continued)

Compound	Chemical Formula	MW	SP. GR. (at 20°C)	BP (°C)	MP (°C)	VP (mmHg at 20°C)	Solubility (at 25°C)
Chrysene	$C_{18}H12$	228.20	1.274	448	255.5	$6.3x10^{-9}$	water: 1.8 µg/kg organics: benzene, ether, alcohol
Fluoranthene	$C_{16}H_{10}$	202.24	1.252	250.5	111.1	$5.0x10^{-6}$	water: 0.206 mg/kg organics: acetic acid, benzene, chloroform, ethanol
Fluorene	$C_{13}H_{10}$	166.2		295	116.5	10 at 146°C	water: insoluble organics: most
Indeno(1,2,3-c,d)pyrene	$C_{22}H_{12}$	276.3			163.6	10^{-10} torr	water: insoluble organics:
2-Methylnapthalene	$C_{11}H_{10}$	142.20		241.05	34.58		water: insoluble organics: most
Naphthalene	$C_{10}H_8$	128.16	1.15	217.9	80.55	0.082	water: 31.7 mg/l organics:
Phenanthrene	$C_{14}H_{10}$	178.22	1.025	340.0	100	6.8x10	water: 1 mg/kg organics: ethanol, toluene, benzene
Pyrene	$C_{16}H_{10}$	202.24		385	149.5	$2.5x10^{-6}$	water: 0.132 mg/kg organics: benzene, diethyl ether, ethanol, toluene, acetone

TABLE 2. STANDARDS, CRITERIA AND GUIDELINES FOR PAHs(a)

Compound	EPA Carc. Class	Slope Factor Inh/Oral (mg/kg/day)$^{-1}$*	Chronic Oral RfD (mg/kg/day)	Chronic Inhalation RfD (mg/kg/day)	Subchronic Oral RfD(b) (mg/kg/day)	Subchronic Inhalation RfD (mg/kg/day)	MCL(c) (mg/l)	Ambient Water Quality Criteria (d) Fish & Water Consumption	Fish Consumption
2									
3	8							2.8 ng/l**	31.1
ng/l**									
Acenapthene	--	--	$6.0x10^{-2}$	--	$6.0x10^{-1}$	--	--		
Acenapthylene	D	--	p	--	--	--	NA		
Anthracene	D	--	$3.0x10^{-1}$	--	$3.0x10^{0}$	--	NA		
Benzo(a)anthracene	B2	--/5.79 x 10^{-1}	--	--	--	--	0.0001		
Benzo(a)pyrene	B2	--/5.79 x 10^{0}	--	--	--	--	0.0002		
Benzo(b)fluroanthene	B2	--/5.79 x 10^{-1}	--	--	--	--	0.0002		
Benzo(k)fluroanthene	B2	--/5.79 x 10^{-1}	--	--	--	--	0.0002		
Benzo(g,h,i)perylene	D	ND/ND	--	--	--	--	NA		
Chrysene	B2	--/5.79 x 10^{-2}	--	--	--	--	0.0002		
Dibenz(a,h)anthracene	B2	--/5.79 x 10^{0}	--	--	--	--	0.0003		
Fluoranthene	D	--	$4.0x10^{-2}$	--	$4.0x10^{-1}$	--	--	42 µg/l	54 µg/l
Fluorene	D	--	$4.0x10^{-2}$	--	$4.0x10^{-1}$	--	NA		
Indeno(1,2,3-c,d)pyrene	B2	--/5.79 x 10^{-1}	--	--	--	--	0.0004		
2-Methylnapthalene	--	--	--	--	--	--	--		
Naphthalene	D	--	$4.0x10^{-3}$(b)	--	$4.0x10^{-2}$	--	NA		
Phenanthrene	D	--	--	--	--	--	NA		
Pyrene	D	--	$3.0x10^{-2}$	--	$3.0x10^{-1}$	--	NA		

*Values derived from B(a)P slope factor listed on IRIS by applying Toxic Equivalency Factors (TEFs).
-- = no data
NA = not available
ND = not determined
** = PAHs, in general (from U.S. EPA, Quality Criteria for Water, May 1986)
p = pending; currently under review by EPA.
References:
(a) U.S. EPA, Integrated Risk Information System (IRIS).
(b) U.S. EPA, Health Effects Assessment Summary Tables (HEAST).
(c) U.S. EPA, Drinking Water Regulations and Health Advisories.
(d) U.S. EPA, OERR, CERCLA Compliance With Other Laws Manual, Interim Final, August 1988.

2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD)

Use

2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) belongs to the class of halogenated dibenzo-*p*-dioxins. TCDD has no commercial use. It is a synthetic chemical formed during the production and use of certain herbicides, 2,4,5-trichlorophenol, 2,4,5-T, and chlorophenols. It may also be formed during the incineration of municipal and industrial wastes or the bleaching process in the pulp/paper industry, or a result of accidental fires in capacitors or transformers containing chlorobenzene.

Chemical and Physical Properties

MF: $C_{12}H_4Cl_4O_2$
MW: 321.97
PD: Colorless needles
MP: 305°C
BP: 412.2 °C (estimated)
VP: 1.5 x 10-9 @ 25°C
SG: N/A
Sol. in water: 7.91 ng/l @ 20-22°C
Sol. in organics: Soluble in dichloro- and chlorobenzene, benzene, chloroform, methanol, and acetone.

Fate and Transport

The majority of atmospheric TCDD is probably particulate-bound. The likely fate for particulate-bound TCDD in air is photolysis followed by removal by wet or dry deposition, with ultimate fate in surface soils (U.S. EPA, 1984). The half-life of this particulate-bound atmospheric TCDD is probably a few days, long enough for transport over considerable distances (ATSDR, 1989).

Due to its low solubility, TCDD does not often appear in water samples except at low levels in surface water samples, where it is bound to suspended material. Some photolysis or vaporization may occur. The half-life of aquatic TCDD is greater than one year. The ultimate sink for TCDD is in sediments (ATSDR, 1989).

Vaporization and photodegradation are likely processes for TCDD in soils. The half-life in surface soils is 1-3 years, while for deeper soil levels the half-life increases to 10-12 years (ATSDR, 1989). TCDD is immobile in most soils but may move horizontally in runoff water during flooding. Minimal vertical movement may occur in soil with low organic content (U.S. EPA, 1984).

TCDD can bioaccumulate in the fatty tissues of fish and mammals. It has also been found in rice grown with phenolic herbicides and in the fat of animals that graze on pastures treated with these herbicides (U.S. EPA, 1987).

Pharmacokinetics

No studies were found in which the method of exposure to TCDD was inhalation. Major routes of exposure are considered to be ingestion and dermal contact.

In one study, a human male ingested 1.14 ng/kg TCDD in oil in a single dose. Over 87 percent TCDD was absorbed and the half-life to elimination was calculated at 2,120 days (Poiger and Schlatter, 1986, as cited in ATSDR, 1989). In a study of Vietnam veterans exposed to Agent Orange, the Center for Disease Control (1987, as cited in ATSDR, 1989) found a half-life of 2,595 days.

No studies were found on dermal absorption of TCDD in humans, but several studies show TCDD to be well absorbed through the skin of mice and rats. However, the vehicle in which TCDD is administered had a great effect: administered in methanol, 40 percent TCDD was absorbed by rats while in a soil paste, less than 2 percent was absorbed (ATSDR, 1989).

Since TCDD readily binds to soil particles, adsorption to particulate matter may reduce absorption of ingested or contacted TCDD below laboratory levels, which are often based on a dose administered in an oily vehicle. Lucier (1986, as cited in ATSDR, 1989) and others showed oral absorption from TCDD adsorbed to soil to be only 50 percent of that from TCDD in corn oil. Soil type and the presence of competing binding agents can further affect the bioavailability of TCDD.

Once in the system, Roberts (1985, as cited in ATSDR, 1989) suggests a receptor-mediated mechanism in which TCDD binds to a soluble, cytoplasmic receptor protein. The complex then migrates to the cell nucleus where it may affect DNA transcription and/or translation. TCDD seems to distribute to organs according to their lipid content rather than their sensitivity to TCDD's toxicity (Ryan, 1985, as cited in ATSDR, 1989). Adipose tissue and mother's milk are therefore particularly expected to accumulate TCDD concentrations.

Metabolism of TCDD is thought to result in the detoxification and relatively rapid removal of metabolites through excretion in bile or urine. Unmetabolized TCDD is eliminated in feces and by lactation (ATSDR, 1989).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

There is no evidence of death in humans as a result of oral or dermal exposure to TCDD.

Sensitivity to TCDD seems to vary greatly among species: Schwetz et. al. (1973, as cited in ATSDR, 1989) reported a single-dose oral LD50 value of .6 ug/kg for guinea pigs, while Henck et. al. (1981, as cited in ATSDR, 1989) reported a corresponding value of 5500 ug/kg for hamsters. Schwetz also found a 50 percent death rate in guinea pigs that had received oral doses of .008 ug/kg/day for 90 days but no deaths in a group receiving .0006 ug/kg/day. In rabbits, he reported an LD50 value of 275 ug/kg for dermal exposure. Allen (1977, as cited in ATSDR, 1989) recorded deaths in 5 out of 8 monkeys given .01 ug/day orally for 9 months.

The only definitively identified effect of TCDD in humans is the condition chloracne, which results in persistent, deforming face and upper-body lesions (ATSDR, 1989). In hairless mice a single oral dose of 70 ug/kg caused the condition (Grieg, 1984, as cited in ATSDR, 1989), as did dermal applications of 1.4 ug/kg 3 times a week for 4 weeks (Puhvel, 1982, as cited in ATSDR, 1989). In monkeys, oral doses of .01 ug/day for 9 months resulted in lesions (Allen, 1977, as cited in ATSDR, 1989).

TCDD has been shown to result in the wasting syndrome (progressive weight loss and degeneration) in many species. DeCaprio (1986, as cited in ATSDR, 1989) found a dose-response relationship for guinea pigs given TCDD orally for 90 days. He reported a frank-effects level (FEL) of 31 ng/kg/day (40 percent weight decrease, mortalitites), a lowest-observable-adverse-effects level (LOAEL) of 4.86 ng/kg/day (13 percent weight decrease, no mortalities), and a no-observable-adverse-effects level (NOAEL) of .68 ng/kg/day.

Several studies also classify TCDD as hepatotoxic for many species. Turner and Collins (1983, as cited in ATSDR, 1989) could not find a NOAEL for guinea pigs given TCDD orally. DeCaprio (1986, as cited in ATSDR, 1989) again reported a LOAEL of 4.86 ng/kg/day and a NOAEL of .68 ng/kg/day for guinea pigs. In rats, Kociba et. al. (1978a,b as cited in ATSDR, 1989) reported "toxic hepatitis and degenerative liver changes" at a LOAEL of .001 ug/kg/day after 2 years. They could not find a NOAEL. Stevens (1981, as cited in ATSDR, 1989) estimated a minimum toxic dose for humans as .1 ug/kg by extrapolating from existing data and considering the minimum toxic dosage for nonhuman primates. There are data relating hepatotoxicity in humans to populations exposed to TCDD, but due to the presence of confounding variables there is no definitive conclusion (U.S. EPA 1985a, as cited in ATSDR, 1989).

Research on the immunotoxic effects of TCDD includes Vos's (1973, as cited in ATSDR, 1989) study of guinea pigs given oral doses weekly for 8 weeks. Effects included decreases in thymus weight, total lymphocyte number, and leukocyte number. The study indicated a FEL of .2 ug/kg, a LOAEL of .04 ug/kg, and a NOAEL of .008 ug/kg.

Symptoms exhibited by humans who have been exposed to TCDD-contaminated chemicals include chloracne, decreased immunoefficiency, hyperpigmentation, hyperkeratosis, hirsutism of the skin, possible hepatoxicity, hypertriglyceridemia, hypercholesterolemia, muscle aches, appetite loss, weight loss, digestive disorders, headache, neuropathy, insomnia, sensory loss, and loss of libido (ATSDR, 1989).

Teratogenic and Other Developmental Effects

There is no well-substantiated evidence of teratogenic effects of TCDD in humans.

Anomalies in rat fetuses were observed with a FEL of .125 TCDD ug administered orally on days 6-15 of gestation and a NOAEL of .03 ug (Sparschu, 1971a,b as cited in ATSDR, 1989). Similar results occurred in mice, with a FEL of approximately 1 ug and a NOAEL of approximately .3 ug (Neubert and Dillman, 1972, as cited in ATSDR, 1989).

In a study of monkeys fed .0015 ug/kg/day for 7 months, 67 percent experienced spontaneous abortions (Allen 1979, as cited in ATSDR, 1989). Murray (1979, as cited in ATSDR, 1989) found decreased fetal survival in rats with a LOAEL of .01 ug/kg/day orally and a NOAEL of .001 ug/kg/day. EPA (1988b, as cited in ATSDR, 1989) upheld this finding despite a statistical reevaluation by Nisbet and Paxton (1982, as cited in ATSDR, 1989), but considering the low dosage in the Allen study, it is probably prudent to accept .001 ug/kg/day as a LOAEL for calculating a minimum risk level.

EPA reviews of studies involving dermal exposure to TCDD found data on reproductive and developmental effects too limited to prove direct causation.

Mutagenic Effects

No studies were found on mutagenic effects of TCDD in humans or animals. Although there is some conflicting data, a large number of negative results in assays usually predictive of genotoxic agents suggests TCDD is not genotoxic (ATSDR, 1989).

Carcinogenic Effects

In reviews by EPA (1985b, 1988b) and Hiremath (1986) of literature reporting associations between TCDD exposure and incidence of soft-tissue sarcomas and lymphomas, EPA concluded that despite confounding factors in the studies, there is *limited* evidence that exposure to phenoxyacetic acid herbicides and chlorophenols is causally related to soft-tissue sarcomas. Evidence is insufficient to make a conclusion about TCDD *alone*.

An NTP study of mice (1982b, as cited in ATSDR, 1989), found that only one strain of mice (Swiss females) developed skin tumors after receiving dermal applications of .01 ug 3 times a week. Kociba (1978a,b, as cited in ATSDR, 1989) found tumors in 9/85, 3/48, 18/48, and 34/40 female rats receiving oral doses of 0, .001, .01, and .1 ug/kg/day respectively. EPA

(1985a) used this study and a reevaluation by Squire (which found tumors in 16/85, 8/48, 27/48, and 34/40 for the same dosages) to calculate a q1* value of 1.56 x 10+5/ug/kg/day. Later EPA concluded a human cancer risk of 1/10,000 from a dose of .6 pg/kg/day and a risk of 1/10,000,000 from a dose of .0006 pg/kg/day (U.S. EPA, 1988a, as cited in ATSDR, 1989).

Ecotoxicity

Due to its long half-life, low solubility in water, and high lipophilicity, TCDD is expected to bioaccumulate in fatty animal tissues.

Standards, Criteria and Guidelines

TCDD is listed as an EPA Class B2 Carcinogen on its own, but as a Class B1 Carcinogen when considered in association with phenoxyherbicides and/or chlorophenols (U.S. EPA, 1985b, as cited in ATSDR, 1989).

TCDD is listed as an IARC Class 2B Carcinogen (IARC, 1982, as cited in ATSDR, 1989).

Oral Slope Factor:	1.5 x 10+5 mg/kg/day
Inhalation Slope Factor:	1.5 x 10+5 mg/kg/day (HEAST, 1992)
Chronic Oral RfD:	1 x 10-6 ug/kg/day (ATSDR, 1989)
AWQC:	to obtain 10-4 - 10-7 excess cancer risk = 1.3 x 10-9 - 1.3 x 10-12 mg/l (U.S. EPA, 1984, as cited in ATSDR, 1989).

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PESTICIDES

ALDRIN

Use

Aldrin is used as an insecticide agent.

Chemical and Physical Properties

Chemical Formula: $C_{12}H_8Cl_6$

MW: 365
SG: 1.70 at 20°C
FP: 150°F
Sol. (water): 20 µg/liter at 25°C
Sol. (organics): Soluble in most organic solvents

BP: Decomposes at 145°C and 2 mm Hg
MP: 104°C
VP: 2.31×10^{-5} mm Hg at 20°C

Fate and Transport

In the environment, aldrin evaporates readily from water environments. However, aldrin is strongly bioconcentrated by aquatic organisms.

Important fate processes include biotransformation and biodegradation by aquatic organisms. The biodegradation of aldrin produces the equally toxic and persistent product of dieldrin. Therefore, biodegradation of aldrin represents a change of state and not detoxification (ICF, 1985).

Pharmacokinetics

Routes of entry include inhalation, skin absorption, ingestion and eye and skin contact. The target organs are the central nervous system, liver, kidneys and skin (Sittig, 1991).

Aldrin is readily biotransformed to dieldrin and stored primarily in fatty tissue (Bann et al, 1956).

Human Toxicity

Noncarcinogenic

Systemic Effects

Aldrin is an extremely toxic substance. The probable lethal dose for humans is between 7 drops and 1 ounce for a 150 pound man. Aldrin often produces convulsions before other less serious symptoms appear. Some victims have

reported headache, nausea, vomiting, dizziness and mild clonic jerking, while others suffer convulsions without warning (IRIS).

Rats fed aldrin in the diet at levels between 0.5 ppm and 150 ppm for two years showed liver lesions. An increase in liver to body weight ratio was also observed at all dose levels (Fitzhugh et al., 1964).

Lesions similar to the ones found in rats were observed in dogs after a 15-month exposure in the diet. Similar effects have been observed in other subchronic to chronic rat and dog studies (IRIS).

Teratogenic and Development Effects

Aldrin is toxic to the reproductive system and teratogenic. Reproductive effects include decreased fertility, increased fetal death and effects on gestation. Teratogenic effects include cleft palate, webbed foot and skeletal anomalies (ICF, 1985).

Mutagenic Effects

Probst et al. (1981) reported unscheduled DNA synthesis in rats and Georgian (1975), chromosomal aberrations in human cells.

Carcinogenic Effects

Human carcinogenicity data is inconclusive. Davis and Fitzhugh (1962) fed mice a diet containing 10 ppm Aldrin for up to two years. An increase in liver lesions was found in the treated animals. An independent re-evaluation (Epstein, 1975) of the liver lesions in the treated animals found that most of those liver lesions were liver carcinomas.

In another study, Aldrin was administered in doses between 3 ppm and 8 ppm in the diet of mice over a period of 80 weeks. An increase in dose-related liver carcinomas was found in male mice (NCI, 1978).

However, other studies (Deichmann et al., 1970; NCI, 1978) found no evidence of carcinogenic response in response to Aldrin in rats.

Ecotoxicity

Aldrin is acutely toxic to freshwater species at low concentrations. LC_{50} values in fish range from 1 to 46 µg/liter depending on the species. Saltwater fish also show acute sensitivity to Aldrin, with LC_{50} values ranging from 2 to 100 µg/liter.

Aldrin and its metabolite dieldrin are both toxic to terrestrial wildlife and domestic animals at low levels (ICF, 1985).

The freshwater and marine acute values are 3.0 and 1.3 µg/L respectively (EPA, 1991).

Standards, Criteria, and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	1.70×10^{1} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	1.71×10^{1} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	3.00×10^{-5} mg/kg/day
Subchronic Oral RfD:	NA
Subchronic Oral RfD:	3.00×10^{-5} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 7.4×10^{-5} µg/L
	Fish Consumption - 7.9×10^{-5} µg/L

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DDT/DDD/DDE

Background

DDT is an organic pesticide which is generally a combination of several organochlorinated pesticides. Technical grade DDT is a mixture of DDT (1,1,1 trichloro-2,2-bis(4-chlorophenyl)ethane), DDD (1,1 dichloro-2-(2-chlorophyenyl)-2(4-chlorophenyl)ethane) and DDE (1,1 dichloro-2,2-bis(4-chlorophyenyl)ethene). The latter two are frequent degradation products of DDT so the three are often detected together (ICF, 1985). DDT, DDD, and DDE are structurally similar so their behavior in the environment is expected to be comparable.

Use

DDT has been used as a plant-growth regulator and as a pesticide/herbicide.

Chemical and Physical Properties

Chemical Formulas: DDT - $C_{14}H_9Cl_5$;
DDD - $C_{13}H_{10}Cl_4$;
DDE - $C_{14}H_8Cl_4$

MW: DDT - 354.5;
DDD - 320;
DDE - 318

Sol. (water): DDT - 5.5 µg/L
DDD - 20 µg/L
DDE - 14 µg:

Sol. (organics): fat and organic solvents

BP: DDT - 260°C
MP: DDT - 109°C
DDD - 112°C
DDE - 90°C

VP: DDT - 1.9 x 10^{-7} mmHg at 25°C
DDD - 1.0 x 10^{-6} mmHg at 30°C
DDE - 6.5 x 10^{-6} mmHg at 20°C

Fate and Transport

DDT is known to be very persistent in soils. It sorbs well to organic matter and is bioaccumulated by plants and animals (ICF, 1985). DDT does not leach rapidly and has a residence time of over 10 years (IARC, 1974).

In aquatic media, DDT isomers are subject primarily to photolysis. Biotransformation to bis(2-chlorophenol)methadone is usually the ultimate fate of DDT (ICF, 1985).

In the atmosphere, DDT is generally subjected to wet and dry deposition and photochemical degradation. The atmospheric half-life of DDT is estimated to be 17 days (ICF, 1985).

Pharmacokinetics

Jensen, et al. (1957) reported that rats absorb 95 percent of an ingested dose of DDT in the gastrointestinal tract and that 35 percent of the absorbed dose is retained in the body for at least 9 days. Absorption of DDT appears to follow the same pathways as fat, in that it is absorbed slowly but completely within 24 hours. DDT is stored in the adipose tissue, liver, brain and muscle tissue of most animals (IARC, 1974). DDT is known to be absorbed by humans in direct proportion to dietary exposure. The clearance half-life of DDT is 10 to 20 years (IRIS, 1990).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

A study by Laug, et al. (1950) in which weanling rats were fed commercial DDT at levels of 0, 1, 5, 10, or 50 ppm for 15 - 27 weeks revealed morphologic liver changes including increasing hepatocellular hypertrophy at dose levels of 5 ppm and above. In an earlier study, chronic oral exposure of rats to levels between 200 and 800 ppm DDT in food also revealed liver alterations. The toxic effects to the liver appear to increase with dosage and the length of exposure.

The effect of chronic exposure to humans is uncertain, based on the available literature. A single dose of 286 mg caused mild sickness in one man (ACGIH, 1984) but, Laws, et al (1967) noted that no adverse effects occurred in workers exposed to 18 mg DDT/day for 19 years (ACGIH, 1984).

Teratogenic and Other Developmental Effects

Exposure to 1 mg/kg bw on days 10, 12, and 17 of gestation caused morphological changes in the gonads of mice. In the same study, it was noted that the fertility of the female offspring was reduced (McLachlan and Dixon, 1972).

DDT is also known to be embryotoxic when administered to mice daily in a dose of 2.5 mg/kg (Schmidt, 1973).

Mutagenic Effects

Chromosomal aberrations were noted in *in vitro* kangaroo-rat and human lymphocyte cells (Palmer, et al., 1972; Lessa, et al, 1976). DDT caused an increase in dominant lethal mutations in *Drosophila melanogaster* (Clark, 1974), Swiss mice (Clark, 1974), and rats (Palmer, et al., 1972)

Carcinogenic Effects

Three studies were located that noted higher tissue concentrations of DDT in humans dying of cancer than in humans dying of other diseases (Casarett, et al., 1968; Dacre and Jennings, 1970,; Wasserman, et al., 1976). The existing human epidemiological studies on DDT are considered inadequate or inconclusive by EPA.

Liver tumors have been linked to DDT exposure in seven studies of various mouse strains and in three rat studies which looked DDT (U.S. EPA, IRIS).

Eight of nine studies performed on laboratory mice indicate that DDT causes hepatocellular adenomas and carcinomas upon ingestion and lung tumors upon inhalation (IRIS, 1990). Walker (1972) reported an increase in liver tumors in mice exposed to DDT in their food for 2 years.

DDD fed to CF-mice at 250 ppm (TWA) for 130 weeks revealed a significant increase in liver tumors (Tomatis et al., 1974). Dose-dependent incidences of hepatocellular carcinomas and hepatomas have been observed in mice and hamsters (NCI, 1978).

Ecotoxicity

DDT is known to be highly toxic to most freshwater and saltwater organisms. Acute toxicity values range from 0.18 to 1800 µg/L, with the final acute value for freshwater species being 1.1 µg/L. The saltwater final acute value for DDT isomers was 0.18 µg/L (ICF, 1985).

DDT isomers are bioaccumulated by most organisms but by fish in particular. Because of this, DDT has caused the death and decreased fecundity in many species of fish-eating birds (ICF, 1985).

Standards, Criteria and Guidelines

EPA Class B2 Carcinogens

Oral Slope Factor:	DDD - 2.4×10^{-1} $(mg/kg/day)^{-1}$
	DDE - 3.4×10^{-1} $(mg/kg/day)^{-1}$
	DDT - 3.4×10^{-1} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	DDD - NA
	DDE - NA
	DDT - 3.4×10^{-1} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	DDD - NA
	DDE - NA
	DDT - 5×10^{-4} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	DDD - NA
	DDE - NA
	DDT - 5×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	DDD - NA
	DDE - NA
	DDT - NA
MCL:	NA
AWQC:	Water and Fish Consumption - 2.4×10^{-5} µg/L (DDT)
	Fish Consumption - 2.4×10^{-5} µg/L (DDT)

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DIELDRIN

Use

Dieldrin is a manmade cyclodiene insecticide. Cyclodiene insecticides are a subgroup of chlorinated cyclic hydrocarbon insecticides which include aldrin, DDT, BHC and other insecticides. The Shell Chemical Company manufactured dieldrin in the United States until 1974 when the EPA prohibited its manufacture under the Federal Insecticide, Fungicide, and Rodenticide Act. Prior to 1974, the primary use of dieldrin was for the control of corn pests, although they were also used in the citrus industry (Sittig, 1991).

Physical and Chemical Properties

Chemical Formula: $C_{12}H_8Cl_6O$

MW: 380.93 MP: 175-176°C
SG: 1.75 VP: 3.1×10^{-6} mm Hg at 20°C
Sol. (water): 0.186 mg/l at 20°C
Sol. (organics): Soluble in common organic solvents except aliphatic petroleum solvents and methanol.

Fate and Transport

Dieldrin is a common degradation product of the insecticide aldrin in all aerobic and biologically active soils. Dieldrin sorps tightly to soils, and therefore, volatilizes slowly. ATSDR (1989) reports that several studies indicate that microbial degradation in soils is probably a minor fate process for dieldrin. Although several studies in ATSDR (1989) report that movement of dieldrin in waterborne sediments is a major fate process, leaching is minimal.

Glotfelt (1978) reports that evidence supports the view that much of dieldrin used in agriculture reaches the atmosphere. Baldwin et al. (1977), however, states that atmospheric degradation probably prevents significant accumulation of dieldrin in the air.

In aquatic systems, the presence of dieldrin in ground water is rare. In fact, Spalding et al. (1980) and Richard et al. (1975) detected no dieldrin in ground water samples. ATSDR (1989) reports that the lack of leaching of dieldrin from soils may explain its absence from ground water. However, small amounts of dieldrin have been detected in surface waters (Richard et al., 1975; Hindin et al., 1964) suggesting the potential for surface runoff of dieldrin from soils (ATSDR, 1989). Dieldrin is converted to photodieldrin, a stereoisomer, upon exposure to sunlight. It is unlikely, however, that photodieldrin occurs widely in the environment (ATSDR, 1989). Volatilization of dieldrin from water is slow. Mackay and Leinonen (1975) report that evaporation of dieldrin from a 1 m column of 25°C water had a half-life of 539 days.

ATSDR (1989) reports that bioaccumulation and biomagnification are significant fate processes in the environment. In a study by Metcalf et al. (1973), biomagnification of dieldrin was 5,957 in fish and 11, 149 in snails.

Pharmacokinetics

ATSDR (1989) reports that several studies indicate absorption of dieldrin via inhalation, oral, and dermal routes of exposure. A correlation between dieldrin treatment of homes and levels of the insecticide in human breast milk suggests absorption by the inhalation route (Stacey and Tatum, 1985). Hunter and Robinson (1967) and Hunter et al. (1969) found a dose-related increase in dieldrin blood levels following oral administration of the insecticide to human volunteers. Although ATSDR (1989) questions the accuracy, Feldmann and Maibach (1974) found 7.7 ±2.9 percent of a single dose of dieldrin applied dermally to the forearm of human volunteers was absorbed over a five-day period.

Dieldrin appears to be distributed to the blood, brain, liver, and adipose tissue in the human body. Hunter and Robinson (1967) and Hunter et al. (1969) exposed human volunteers to 0, 10, 50, or 211 µg dieldrin/man/day for 24 months. Dieldrin concentrations in the blood and adipose tissue increased in a dose-related manner reaching a steady-state concentration balanced by ingestion and elimination. In mid- and high-dose groups, blood levels increased 4-10 times compared to controls. Dieldrin was bioconcentrated 136 times in the adipose tissue relative to blood levels. Bioconcentration appears to plateau after 9-12 months (Geyer et al., 1986). Following termination of dieldrin administration, its blood level decreased exponentially with a half-life of approximately 369 days (Hunter et al., 1969). In the brain, DeVlieger et al. (1968) reports that the concentration of dieldrin is significantly greater in the white brain matter than in the gray brain matter. Hunter and Robinson (1968) report that the bioconcentration and elimination of dieldrin are related to the lipid mass of the individual. A lean person will attain a higher adipose tissue concentration but the total amount retained will be less than in an individual with increased body fat.

Although information on the metabolites of dieldrin was not available, Ackerman (1980) reports that the concentration of dieldrin in the human body reaches a maximum level at which metabolism and excretion rates equal ingestion and absorption rates. As the concentration of dieldrin in the liver increases, the metabolizing enzyme activity increases and the rate of biodegradation and excretion is enhanced. Jager (1970) and Hunter et al. (1969) report that accumulation occurs at a slower and slower rate until concentrations of dieldrin in blood and tissues reach the maximum limit.

ATSDR (1989) reports that the main route of dieldrin excretion is in the feces primarily via the bile. The insecticide can also be excreted via breast milk. Urine appears to be a minor elimination route.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

In a study by Walker et al. (1969), Carworth Farm "E" rats were exposed to dietary dieldrin concentrations of 0, 0.1, 1.0, and 10.0 ppm for 2 years. These dietary levels are approximately equal to 0, 0.005, 0.05, and 0.5 mg/kg/day. The only effects observed during the study were that the animals became irritable and exhibited tremors and occasional convulsions. At the end of the 2 years, females exposed to the higher doses had increased liver weights and liver-to-body weight ratios. Hepatic lesions characteristic of organochlorine insecticide exposure, including focal proliferation and focal hyperplasia of liver parenchymal cells, were revealed upon histopathological examinations. In a similar experiment with dogs, the same concentrations produced increased liver weights and liver-to-body weight ratios, increased plasma alkaline phosphatase, and decreased serum protein concentration.

Fitzhugh et al. (1964) observed liver enlargement and histopathology in rats exposed to 0.025 mg/kg/day dieldrin for 2 years and weight loss and convulsions in dogs exposed to 0.5 mg/kg/day dieldrin for 25 months. In another study, Walker et al. (1972) reported liver enlargement and histopathology in mice exposed to 0.015 mg/kg/day for 2 years.

Teratogenic and Other Development Effects

Mice exposed to 6.0 mg/kg/day dieldrin during days 7-16 of gestation exhibited decreased maternal weight gain. Fetotoxic effects included decreased number of caudal ossification centers and an increase in supernumerary ribs (Chernoff et al., 1975).

The U.S. EPA (1987) reports that several studies have illustrated reduced fertility and reduced offspring survival following maternal exposure to dieldrin. The lowest dietary dieldrin concentration to cause these reproductive effects was reported by Hodge et al. (1967) to be 2.5 ppm in rats.

Mutagenic Effects

U.S. EPA (IRIS) reports that several studies have indicated that dieldrin causes chromosomal aberrations in mouse cells, human lymphoblastoid cells, forward mutation ion Chinese hamster V79 cells, and unscheduled DNA synthesis in rat and human cells. In addition, Majumdar et al. (1977) reports that dieldrin was mutagenic for *S. typhimurium* with and without metabolic activation. However,

U.S. EPA (IRIS) reports that reverse mutation assays with six strains of *S. typhimurium* yielded negative responses with or without metabolic activation. U.S. EPA (IRIS) also reports that several other studies produced negative responses including assays for gene conversion in *S. cerevisiae*, back mutation in *S. marcesans*, forward mutation (Gal R22 in *E. coli*), and forward mutation to streptomycin resistance in *E. coli*.

Carcinogenic Effects

In a long-term carcinogenic bioassay by the Food and Drug Administration, Davis and Fitzhugh (1962) reported a significant increase in the incidence of hepatomas following 2-year exposure of C3HeB/Fe mice to 10 ppm dieldrin. Davis (1965) reported a significant increase in the incidence of benign hepatomas and hepatic carcinomas in C3H mice orally administered 10 ppm dieldrin. Although both of these studies are compromised by poor survival rate, lack of detailed pathology, loss of a large percentage of the animals to the study, and failure to treat the data for males and females separately; a reevaluation of the histological material of both studies by Reuber in 1974 (Epstein, 1975a, b; 1976) revealed that the hepatomas were malignant and that dieldrin was hepatocarcinogenic for male and female C3HeB/Fe and C3H mice.

Numerous other studies in mice reported by the U.S. EPA (IRIS) also reveal the hepatocarcinogenic effects of dieldrin.

Walker et al. (1972) reports significant increases in the incidence of pulmonary adenomas, pulmonary carcinomas, lymphoid tumors, and "other" tumors in female CF1 mice exposed to dieldrin doses as low as 0.1 ppm for 132-140 weeks.

Ecotoxicity

The U.S. EPA (1980) reports that, in freshwater systems, the acute toxicity of dieldrin has been evaluated for species in all major functional and taxonomic classifications. The most resistant fish species is the goldfish with an LC_{50} of 41 µg/l. The most sensitive fish species is the rainbow trout with an LC_{50} of 2.5 µg/l. Invertebrate tolerance limits ranged from an LC_{50} of 39 µg/l for the stone fly to 5 µg/l for the isopod. Chronically, tolerance limits were found to be 0.22, 0.45, and 57 µg/l (LC_{50}) for the rainbow trout, guppy, and fruit fly, respectively. Bioconcentration factors cover a wide range in freshwater systems. Yearling lake trout which may not have reached steady state had a bioconcentration factor of 68,286 and U.S. EPA (1980) reports that this value may be even higher in older, larger lake trout. Freshwater plants, which demonstrated a minimum tolerance limit of 100 µg/l (LC_{50}) for 10 days, are clearly more resistant to dieldrin than freshwater animals.

In saltwater systems, tolerance limits for invertebrates ranged from an LC_{50} of 0.7 µg/l for the pink shrimp to 50 µg/l for the grass shrimp. In fish, tolerance limits ranged from a minimum of

0.9 µg/l for the American eel to 34 µg/l for the northern puffer. Chronically, mysid shrimp were found to have a tolerance limit of 0.49 - 1.1 µg/l. Bioconcentration factors ranged from 400 to 8000 for fish or shellfish.

No data on the ecotoxicity of dieldrin in terrestrial systems were available in the literature reviewed.

Standards, Criteria, and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	1.61×10^{1} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	1.61×10^{-1} (mg/kg/day)$^{-1}$
Chronic Oral RfD:	5×10^{-5} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5×10^{-5} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 7.1×10^{-5} µg/L
	Fish Consumption - 7.6×10^{-4} µg/L

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ENDOSULFAN

Use

Endosulfan is an agricultural insecticide used for vegetable crops, fruits and nuts.

Chemical and Physical Properties

Chemical Formula: $C_9H_6Cl_6O_3S$

MW: 406.95
SG: 1.735 at 20°C
Sol. (water): Practically insoluble
Sol. (organics): Moderately soluble in most organic solvents

MP: 70° - 100°C (technical) 106°C (pure)
VP: 9×10^{-3} torr (technical)

Fate and Transport

No information on this topic was found in the literature.

Pharmacokinetics

Possible routes of entry include inhalation, ingestion, and eye and skin contract (Sittig, 1991).

Human Toxicity

Noncarcinogenic

Systemic Effects

Symptoms of endosulfan ingestion include nausea, vomiting and diarrhea. Dizziness, bluing of the skin, agitation, nervousness, tremor, incoordination and convulsions are also possible. Depression of the central nervous system may result in respiratory failure. Contact may irritate or burn the skin, eyes and mucous membranes. The oral lethal dose may be as low as 50 mg/kg. Death has been reported within two hours of heavy dust exposure. The target organs of this compound are the central nervous system, lungs and skin (Sittig, 1991).

Reduced growth, enlargement of the kidneys and blood changes were observed in rats fed 5 mg/kg/day of endosulfan for two years (Smith, A.G., 1991).

Teratogenic and Developmental Effects

Endosulfan administered orally to rats on day 6 through 14 of gestation increased the mortality of rat dams and rates of resorption and skeletal abnormalities in the fetuses (Gupta et al., 1978).

Mutagenic Effects

The National Cancer Institute reported that Endosulfan was positive in the mouse lymphoma mutation assay but negative in the *Salmonella* assay and cultured Chinese hamster ovary cells for genotoxic effects (American Conference of Governmental Industrial Hygienists, Inc., 1991).

Carcinogenic Effects

No information on this topic was found in the literature.

Ecotoxicity

The acute and chronic freshwater values are 0.22 and 0.056 µg/l, respectively. Acute and chronic marine values are 0.034 and 0.0087 µg/l, respectively (EPA, 1991).

Standards, Criteria, and Guidelines

Unclassified by EPA as to carcinogenicity.

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	5×10^{-5} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5×10^{-5} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 74 µg/L
	Fish Consumption - 159 µg/L

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ENDRIN/ENDRIN KETONE

Use

Endrin is used primarily as a pesticide agent for field crops. It is also used as a rodenticide and an avicide. Endin Ketone is a biodegradation product of endrin (ATSDR, 1990).

Chemical and Physical Properties

Endrin (no information available on endrin ketone).

Chemical Formula: $C_{12}H_8Cl_6O$

MW: 380.9
SG: 1.65 at 25°C
FP: 27°C
Sol. (water): 250 µg/liter at 25°C
Sol. (organics): Acetone, benzene, carbon tetrachloride, hexane, xylene.

MP: 226° to 230°C (decomposes)
VP: 2.7 1 10^{-7} mm Hg at 25°C

Fate and Transport

Endrin persists in the environment. Volatilization from soil surfaces and surface waters and phototransformation are important transport mechanisms (Nash, 1983). The physical properties of this chemical suggest that absorption to soils and sediments is another important fate mechanism. Aquatic organisms readily bioconcentrate this chemical. Other important fate mechanisms include biotransformation and biodegradation (ICF, 1985). No information on the fate and transport of endrin ketone was available in the literature reviewed.

Pharmacokinetics

Potential routes of entry include ingestion, skin absorption, eye and skin contact. Endrin is rapidly absorbed through the skin. The main target sites of this chemical are the liver and the central nervous system (Sittig, 1991), but the heart, lungs and liver are also susceptible (ICF, 1985). Symptoms may appear between 20 minutes and 12 hours after exposure (Sittig, 1991). After absorpotion, endrin is partly retained in fatty tissues and partly excreted in urine and feces unchanged and as three metabolites (ACGH, 1980). ATSDR (1990) reports that endrin ketone is a metabolite of endrin that appears to be more toxic in rats than endrin itself. In fact, Hutson et al. (1975) reports that endrin ketone is most likely the toxic entity of endrin. No other toxicological information on endrin ketone was available in the literature reviewed and, based on the findings of Hutson et al. (1975), the toxic effects discussed below in regards to endrin may be attributable to endrin ketone as well.

Human Toxicity

Noncarcinogenic

Systemic Effects

Chronic exposure to low levels of endrin results in damage to the nervous system. Endrin can also produce reproductive disorders following chronic exposure (ICF, 1985).

Rats and mice exposed by inhalation to Endrin concentrations of 0.36 ppm seven hours each day, five days each week for 26 weeks showed no signs of intoxication or growth retardation. Two rabbits of a group of four however failed to survive under the same conditions (Treon et al., 1955).

Acute exposure effects include headache, nausea, vomiting, dizziness, tremors, loss of consciousness, rise in blood pressure, frothing of the mouth, deafness, coma and death by respiratory arrest. Doses as low as 1 mg/kg can cause symptoms. Inhalation may cause irritation of the nose and throat and sudden convulsions (Sittig, 1991).

Teratogenic and Other Development Effects

Endrin has been shown to be a potent teratogen and reproductive toxicant in experimental animals. Teratogenic effects include cleft palate, meningo encephales and fused ribs. Developmental effects include fetal mortality and growth retardation (ICF, 1985).

Mutagenic Effects

Endrin has not been shown to be a mutagenic agent. Microbial systems, with and without metabolic activation, did not produce a mutagenic response when exposed to Endrin (Moriya et al., 1983; Probst et al., 1981; Glatt et al., 1983).

Carcinogenic Effects

Human carcinogenic data is insufficient. Animal carcinogenic data is inconclusive. Reuber (1978) reported carcinogenic effects on male and female Osborne-Mendel rats exposed to 0.1 to 25 ppm Endrin in the diet. Malignant tumors of the liver were found in the 0.1 ppm dose group.

However, Deichmann et al. (1970) reported no evidence of carcinogenicity in Osborne-Mendel rats and Treon et al. (1955) reported no evidence of carcinogenicity in Carworth Farm rats when the animals were exposed to between 1 and 100 ppm Endrin in their diets.

Ecotoxicity

Endrin is toxic to aquatic organisms. Freshwater fish are generally more sensitive than fresh or marine invertebrates. LC_{50} values for saltwater organisms range from 0.037 to 14.2 µg/liter. Abnormal behavior, increased postnatal mortality and increased fetal death have been observed in animals exposed to Endrin. The freshwater acute and chronic toxicity levels are 0.18 µg/liter and 0.0023 µg/liter. For saltwater species, these levels are 0.037 µg/liter for acute toxicity and 0.0023 µg/liter for chronic toxicity (ICF, 1985).

Standards, Criteria, and Guidelines

Endrin (no standards for endrin ketone)

Unclassified by EPA as to carcinogenicity.

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	3.0×10^{-4} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5.0×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	2 µg/L
AWQC:	Water and Fish Consumption - 1 µg/L Fish Consumption - .81 µg/L (recalculated)

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HEPTACHLOR/HEPTACHLOR EPOXIDE

Use

Heptachlor is used as an insecticide agent. Heptachlor epoxide is a biodegradation product of heptachlor (EPA, 1985).

Chemical and Physical Properties

Heptachlor

Chemical Formula: $C_{10}H_5Cl_2$

MW: 373.3
SG: 1.57 - 1.59 at 9°C
MP: 95-96°C (pure); 46-74°C (technical grade)
VP: 0.0003 mm Hg at 25°C
Sol. (water): 0.056 to 0.180 mg/liter at 25-29°C depending on particle size
Sol. (organics): soluble in ethanol, ether, benzene, acetone, carbon tetrachloride, xylene, kerosene, cyclohexanone, ligroin

Heptachlor Epoxide

Chemical Formula: $C_{10}H_5Cl_7O$
MW: 389.32
MP: 160 - 161.5°C (99.5% pure)
VP: 0.0003 mm Hg at 25°C
Sol. (water): 0.35 mg/liter at 25°

Fate and Transport

In aquatic media, Callahan et al. (1979) report that heptachlor may undergo significant photolysis. In fact, Verschueren (1983) found complete degradation of 10 mg/liter of heptachlor in a sealed glass jar in 2 weeks (75% in 1 week). EPA (1985) reports that the singlet oxygen reaction with heptachlor, with an estimated half-life of approximately one day, may be significant in aquatic media. Hydrolysis also appears to be an important process. Callahan et al. (1979) and Mabey et al. (1981) estimate the hydrolytic half-life of heptachlor to be in the range of 1-3 days. EPA (1985) reports that the volatilization half-life of heptachlor from pond, river, and lake water is estimated to be 2-10 days.

Callahan et al. (1979) report that sorption of heptachlor onto suspended particles and sediments may play a significant role in determining the fate of heptachlor in aquatic media. Callahan et al. (1979) and Mabey et al. (1981) report that biodegradation is slower than hydrolysis.

In soils, ICF (1985) reports that, although volatilization may occur, it is not an expected fate since heptachlor binds tightly to soil particles and persists for years after surface application. ICF (1985) also reports that certain crops bioaccumulate heptachlor by absorption from soils. Limited atmospheric transport of heptachlor vapors and contaminated particles from soil can occur although this mode of transport is affected by geographical and seasonal factors (ICF, 1985).

Heptachlor is not expected to accumulate in ground water or surface water because of its sorption to soils and capability of volatilization.

EPA (1985) reports that photolysis, oxidation, biodegradation, and hydrolysis are not expected to be significant fate-determining processes for heptachlor epoxide in aquatic media. Sorption and volatilization may determine the fate of heptachlor epoxide in aquatic media (EPA, 1985). Information on the fate and transport of heptachlor epoxide in other environmental media was not available in the literature reviewed.

Pharmacokinetics

ATSDR (1989) states: "Though limited, data suggest that heptachlor is readily absorbed from the gastrointestinal tract and may be absorbed through the skin. A large portion of the absorbed heptachlor is slowly eliminated, primarily via the bile duct into the feces. Heptachlor is readily oxidized to heptachlor epoxide in mammals. Heptachlor epoxide has been detected in various tissues of rats and dogs, with the highest levels found in fat. The accumulation of heptachlor epoxide in fat is dose dependent, and female rats accumulate more than males. Heptachlor epoxide has been detected in several human tissues, blood, milk and amniotic fluid at concentrations of <1 ppm. Unchanged heptachlor was also detected in human milk samples."

Human Toxicity

Noncarcinogenic

Systemic Effects

Chronic exposure to heptachlor produces changes in the liver (ICF, 1985). Velsicol Chemical Corp. (1955) reported lesions of the liver in CF strain white rats fed 7 ppm of heptachlor for two years. Acute exposure to heptachlor by various routes can result in the development of hepatic vein thrombi, effects on the central nervous system and death (ICF, 1985). Liver toxicity has been established as the most important endpoint for this class of compound (IRIS). Acute and chronic toxicity symptoms in humans include tremors, convulsions, kidney damage and respiratory collapse by various routes (ICF, 1985).

Dow Chemical Co. (1985) exposed beagle dogs to 0-7.5 ppm heptachlor epoxide in the diet for 60 weeks. Significantly increased liver-to-body weight ratios were observed at levels as low as 0.5 ppm. In a 2-year feeding study with rats, 0.5

ppm produced vacuolar changes in the central hepatic lobule of females. (Velsicol Chemical, 1959a). 1 ppm hepatochlor epoxide produced vacuolation, enlarged nuclei, and hepatocytomegaly in a chronic feeding study with mice (Velsicol Chemical, 1973a).

Teratogenic and Other Developmental Effects

Developmental and teratogenic effects of heptachlor in rats include decreased litter size, shortened life span of suckling rats and the development of cataracts. Heptachlor has also been found in the tissues of stillborn infants, suggesting an ability to cross the placenta (ICF, 1985). In a 1-generation reproduction study in rats, Velsicol Chemical (1955) found an increase in pup death at 7 ppm. However, a 3-generation reproduction study (Velsicol Chemical, 1967) found no adverse effects at 10 ppm.

Velsicol Chemical (1973b) found that 3 ppm heptachlor epoxide produced liver lesions in pups, and 7 ppm decreased pup survival in 2-generation reproduction study with dogs. In a 3-generation study with rats, Velsicol Chemical (1959b) found increased pup mortality at 10 ppm.

Mutagenic Effects

Gene mutation assays indicate that heptachlor is not mutagenic in bacteria (Probst, 1981; Moriya, 1983) or mammalian liver cells (Telang, 1983). DNA repair assays indicate that this compound is not genotoxic in rodent heptacytes (Maslausky and Williams, 1981; Probst, 1981) but showed evidence of unscheduled DNA synthesis in human fibroblasts (Ahmed, 1977). Results from other studies however suggest, that heptachlor may in fact reduce genotoxic activity (ICF, 1985).

Gene mutation assays conducted by Moriya et al. (1983) indicate that heptachlor epoxide is not mutagenic in bacteria. In mouse dominant lethal assays (Arnold et al., 1977; Epstein et al., 1972), heptachlor epoxide did not induce major chromosomal aberrations in male germinal cells. Ahmed et al. (1977) reported qualitative evidence of unscheduled DNA synthesis in SV40 transformed human fibroblasts in the presence of hepatic homogenates and heptachlor epoxide.

Carcinogenic Effects

Heptachlor has been found to be hepatocarcinogenic when administered orally to mice (ICF, 1985). Davis (1965) fed mice diets of 10 ppm heptachlor over two years. After histologic evaluations (Reuber, as cited in Epstein, 1976), the treated mice showed an increase in liver carcinomas over the control group. The National Cancer Institute (1977) reported a similar dose related increase in hepatocellular carcinomas in male and female

mice but not in Osborne-Mendel rats. Studies by Witherup et al. (1955), Jolley et al. (1966) (see below) and Cabral et al. (1972) also found no carcinogenicity, with heptachlor at levels as high as 10 ppm. Human carcinogenic data, however, is inadequate to draw any conclusions (EPA, IRIS).

Davis (1965) fed groups of C3H mice 0 or 10 ppm heptachlor epoxide for 2 years. A 2-fold increase in benign liver lesions (hepatic hyperplasia and benign tumors) was observed. Reuber (1977) found a significant increase in liver carcinomas upon reevaluation of the Davis (1965) data. Reuber (1977) also found an increase in liver carcinomas in CD-1 mice exposed to 75% heptachlor epoxide: 25% heptachlor mixture at 5 and 10 ppm for 18 months by Velsicol Chemical (1973c). Reuber (1985) reported a significant increase in hepatic carcinomas in female rats exposed to 5 and 10 ppm heptachlor epoxide for 108 weeks by Witherup et al. (1959). Jolley et al. (1966) exposed female CD rats to 5-12.5 ppm 75% heptachlor: 25% heptachlor epoxide for 2 years. Although no malignant lesions of the liver were observed, hepatocytomegaly was increased at levels as low as 7.5 ppm.

Ecotoxicity

Heptachlor is toxic at low concentrations in some aquatic invertebrate and fish species. Heptachlor may bioaccumulate at levels thousands of times greater than the surrounding water in many aquatic organisms. Therefore, chronic exposure to levels greater than .004 µg/liter is considered potentially harmful to aquatic life. Mean acute levels for freshwater species range from 0.9 to 320 µg/liter and for saltwater species, 0.04 to 194 µg/liter. Increased mortality in birds, mammals and aquatic species has been reported in heptachlor treated areas (ICF, 1985).

No data on the ecotoxicity of heptachlor epoxide were available in the literature reviewed.

Standards, Criteria, and Guidelines

HEPTACHLOR/HEPTACHLOR EPOXIDE

EPA Class B2 Carcinogens

Oral Slope Factor:	4.5×10^{0} (mg/kg/day)$^{-1}$/9.1×10^{0} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	4.55×10^{0} (mg/kg/day)$^{-1}$/9.1×10^{0} (mg/kg/day)$^{-1}$
Chronic Oral RfD:	5.0×10^{-4} mg/kg/day/1.3×10^{-5} (mg/kg/day)$^{-1}$
Chronic Inhalation RfD:	NA/NA
Subchronic Oral RfD:	5.0×10^{-4} mg/kg/day/NA
Subchronic Inhalation RfD:	NA/NA
MCL:	0.0004 mg/L/0.0002 mg/l
AWQC:	Water and Fish Consumption - .00028 µg/L/NA
	Fish Consumption - .00029 µg/L/NA

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POLYCHLORINATED BIPHENYLS

Background

The name polychlorinated biphenyl (PCB) categorizes any biphenyl ring in which one or more hydrogen is replaced by a chlorine atom. In commercial PCB mixtures, 40 to 70 different chlorinated biphenyl compounds can be present.

This profile is concerned with PCB-1260, -1254, and -1248, also known as Arochlors. Any statements made, unless otherwise specified, characterize these three PCBs.

Use

PCBs belong to a class of chemically stable, multi-use industrial chemicals that have been distributed widely in the ecosystem (EPA, 1985). PCBs were used in transformers and capacitors as dielectric insulating fluids because of their fire resistant qualities. In 1974, approximately 70 percent of domestically sold PCBs (34 million pounds) were used in capacitors while the remainder was used in transformers. An additional 450,000 pounds were imported for use in investment casting waxes, semi-enclosed heat transfer applications and mining equipment. The use of PCBs in capacitors began in 1930 and was discontinued in 1978. However, most of the electrical equipment containing PCBs are still in service (EPA, 1985).

Chemical and Physical Properties

Chemical Formula: $(C_6H_5Cl_x)_2$

MW: 189-399*
SG: 1.3 to 1.5 at 20°C*
FP: 300 - 500°F*
Sol. (water): 0.003 - 60.6 mg/l
Sol. (organics): soluble in most

BP: >267°C*
MP: 54-310°C
VP: 6.0×10^{-5} mmhg to
1.0×10^{-3} mmhg**

* increases with chlorination
**decreases with chlorination

Fate and Transport

PCBs are extremely persistent in soils containing moderate to high levels of organic matter. Heavily chlorinated PCBs persist longer and degrade slower than lightly chlorinated PCBs. PCBs are known to bioaccumulate readily in adipose tissues, especially in interstitial organisms.

In aquatic media, PCBs tend to volatilize, after which they may be slowly photolyzed in the atmosphere. Aquatic invertebrates are important in the cycling of PCBs within the aquatic environment and between aquatic and terrestrial ecosystems (Eisler, 1986).

Pharmacokinetics

Little data on PCB absorption are available. However, PCBs are expected to be absorbed almost completely in the gastrointestinal tract (90 percent for Aroclor 1248 at dose levels of 1.5 or 3.0 mg/kg body weight) (EPA, 1985). Studies in which PCBs are placed in the diet of Sprague-Dawley dams revealed that tissue concentrations vary in the following descending order: adipose tissue, mammary glands, kidney, liver, and lung. When Aroclor 1254 was administered at 0.25 to 50 mg/kg in the diet of rats starting on day 8 of gestation and continuing through postpartum day 14, the concentration of PCBs in milk was found to be 293 µg/ml by postpartum day 14 (EPA, 1985). Generally, it seems that the degree of chlorination greatly influences the rate of metabolism; the more heavily chlorinated the biphenyl moiety, the slower its metabolism (EPA, 1985). Data on the excretion of PCBs were not available, however, PCBs are expected to be eliminated via the urine (EPA, 1985).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Exposure to PCB fumes results in acneform eruptions, irritation to the respiratory passages, and injury to the liver (ACGIH, 1984). These effects were noted at airborne PCB levels as low as 0.1 g/m^3 (EPA, 1985). Many studies of occupational exposure to PCBs have shown varying degrees of dermatologic and hepatic effects. Accidental human ingestion of PCB-contaminated rice oil in Japan in 1968 resulted in similar symptoms but also included headaches, vomiting, and spasms (ACGIH, 1984).

Teratogenic and Other Developmental Effects

Little evidence of teratogenicity was found in the scientific literature reviewed; most reports concentrated on the fetotoxicity of PCBs. Infants of monkeys exposed to 2.5 ppm Aroclor 1248 in the diet for 6 months before mating were born with hyperpigmentation (EPA, 1984). Reduced birth weights were observed in infants from monkey exposed to 1.0 ppm Aroclor 1016 in the diet. Nursing resulted in the loss of facial hair, edema of the eyelids, gastric hyperplasia, vomiting, liver degeneration, and other signs of PCB-induced toxicity, (EPA, 1984). Dams, exposed to PCB concentrations of 8 mg/kg/day orally, showed sign of lethargy and vaginal bleeding (EPA, 1984).

In one study, Aroclor 1254 was given orally to rabbits at doses of 1, 10, 12.5, 25.0, 50.0 mg/kg-bw for the first 28 days of gestation. At a dose level of 12.5 mg/kg-bw/day, adverse reproductive effects were observed (resorptions, aborted fetuses, decreased litter size). Aroclor 1248 at 2.5 or 5 ppm (0.06 or 0.12 mg/kg/day) in the diet for up to 6 months has been demonstrated to prolong the menstrual cycle in Rhesus monkeys (EPA, 1985).

Mutagenic Effects

The mutagenic activity of PCB mixtures and isomers was studied using *Salmonella typhimurium* (TA1538) in the presence of a microsomal activation system. Aroclor 1221 and 4-chlorobiphenyl were significantly mutagenic in the assay system (EPA, 1985).

Carcinogenic Effects

PCBs are considered Class B2 carcinogens, probable human carcinogens. Human studies are not adequate. In a number of studies on the effects of occupational exposure to PCB fumes, statistically significant evidence points toward carcinogenicity, but these studies did not note the presence or absence of other carcinogens. In the aforementioned studies, polychlorinated dibenzofurans and quinones were also present in the PCB-contaminated rice oils (IRIS).

Norback and Weltmann (1985) fed 140 Sprague-Dawley rats a diet of corn oil mixed with Aroclor 1260 at 100 ppm for 16 months. They followed that with a diet containing 50 ppm Aroclor for 8 months and then a basal diet for 5 months. Female rats exhibited a 91 percent incidence of hepatocellular carcinoma. Males exhibited a 4 percent incidence of carcinoma and an 11 percent incidence of neoplastic nodules.

Other studies on laboratory animals indicated an increase in carcinogenesis amongst PCB exposed animals. From most studies, it appears as though exposure to the more heavily chlorinated PCBs results in an increased risk of cancer.

Ecotoxicity

Polychorinated biphenyls are bioaccumulated and can be biomagnified, therefore, their toxicity increases with length of exposure and position of the exposed species on the food chain (ICF, 1985). Chronic exposure of PCBs for rainbow trout, bluegills, and channel catfish yielded LC_{50} values of about 0.1 mg/liter. Invertebrate species are also adversely affected, with some species having 7-day LC_{50} values as low as 1 µg/liter (ICF, 1985). Present data imply that, in general, juvenile organisms appear more susceptible to the effects of PCBs than either eggs or adults (ICF, 1985), and lower chlorinated biphenyls are more toxic to aquatic organisms than higher chlorinated biphenyls (Eisler, 1986). It is clear that based on the chronic values available in the literature, PCBs are highly toxic to both freshwater and saltwater aquatic life.

Diet is an important exposure route for PCB accumulation within terrestrial species. Sensitive bird species are susceptible to PCB poisoning, mainly as a result of eating contaminated fish or bivalves. LD_{50}s for several avian species ranged from 604 to greater than 6,000 mg Arochlor/kg diet although reproductive impairment has been observed at 5 mg Arochlor/kg diet concentrations (Eisler, 1986). Documentation of PCB exposure to terrestrial animals is limited, however, mammals are generally more sensitive to acute PCB toxicity than birds. Mink are particularly sensitive as 0.64 mg/kg in their diet caused reproductive failure and LD_{50} concentrations less than 10 mg Arochlor/kg have been reported (Eisler, 1986).

Standards, Criteria and Guidelines

EPA Class B2 carcinogen

Oral Slope Factor:	7.7 $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	NA
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	0.5 µg/l
AWQC:	Water and Fish Consumption - 4.4×10^{-5} µg/L
	Fish Consumption - 4.5×10^{-5} µg/L
	Freshwater Acute - 2.0 µg/L
	Freshwater Chronic - 1.4×10^{-2} µg/L
	Marine Acute - 10.0 µg/L
	Marine Chronic - 3.0×10^{-2} µg/L

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INORGANICS

ANTIMONY

Use

Antimony is widely used as an alloy constituent in pewter and white metal, and is used in the manufacture of storage battery plates, solder, and ammunition because of its strength and its resistance to corrosion (Sittig, 1991). It is used as a fire-retardant in textiles and is used to dye steel, aluminum, pewter, and zinc (Sittig, 1991). One compound, antimony potassium tartrate, is used in medicine and as a leather mordant (ACGIH, 1984).

Chemical and Physical Properties

AG: 121.75	BP: 1750°C
SG: 6.684 at 25°C	MP: 630.74°C
Sol.(water): Insoluble	
Sol.(organics): Insoluble	

Fate and Transport

Antimony is present naturally in water bodies as antimony oxide. Antimony oxide is generally reduced to stibine (SbH_3) in benthic sediments. Stibine is highly volatile and is very soluble in water but, in aerobic environments, it is rapidly oxidized to Sb_2O_3. In anaerobic waters, antimony compounds are quite soluble and, when present in rivers and lakes, they rapidly transport to oceans (ICF, 1985). Antimony is known to sorb to clays and minerals so, in soils, antimony would be expected to remain stable. Particulate antimony compounds are known to transport well in the atmosphere (ICF, 1985).

Pharmacokinetics

No pertinent information was located regarding the pharmacokinetics of antimony. It appears as though antimony primarily effects the lungs upon inhalation. Ingestion of antimony leads to kidney and liver damage (ACGIH, 1984) suggesting absorption occurs in these organs.

Human Toxicity

Noncarcinogenic

Systemic Effects

Schroeder et al. (1970) reported that rats administered 5 ppm potassium antimony tartrate in water exhibited reduced lifespans and altered blood chemistries; no increased incidence in tumors was seen.

One study reported that, of 125 workers employed in the abrasives industry, 6 died suddenly and two died of chronic heart disease. Upon examination of 75 of the workers, 37 exhibited EKG problems, 14 had high blood pressure, and 7 had ulcers (ACGIH, 1984). Ambient air levels were found to range from 3 to 5 mg/m^3. These problems were confirmed to be a result of antimony exposure when rats, rabbits, and dogs were exposed to similar concentrations in the air (3.7 to 5.6 mg/m^3). Cardiac dysfunction and parenchymatous degeneration of the myocardium were noted in all species. Chronic inhalation of antimony trioxide caused severe pneumonitis in guinea pigs (ACGIH, 1984).

Teratogenic and Other Developmental Effects

Human case studies suggest that antimony may cause an increase in spontaneous abortions and several other gynecological disorders (ICF, 1985). Decreased weight gain was observed in babies born to mothers exposed to antimony compounds (ICF, 1985).

Mutagenic Effects

Several bacterial studies indicate that antimony compounds are mutagenic (ICF, 1985).

Carcinogenic Effects

Antimony has been shown to increase lung cancer among exposed workers. An inhalation study performed on rats indicated that antimony trioxide increases the risk of lung and liver tumors (ICF, 1985). The number of studies performed, however, has been inadequate to categorize antimony as a carcinogen. EPA has not evaluated antimony for evidence of human carcinogenic potential.

Ecotoxicity

LC_{50} values for the freshwater species, *Daphnia magna,* and the fathead minnow, range between 9,000 and 21,900 mg/l. No detectable bioconcentration of antimony was noted in bluegill (ICF, 1985).

No data regarding toxicity of antimony to terrestrial species other than laboratory species were located in the available literature.

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	4×10^{-4} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	4×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.01/0.005 mg/l
AWQC:	Water and Fish Consumption - .15 mg/l
	Fish Consumption - 45 mg/l

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ARSENIC

Use

Arsenic can be found in the environment in four valence states (-3, 0, +3, +5) and is used industrially in the form of arsenic disulfide, arsenic pentoxide, arsenic trichloride, arsenic trisulfide and lead arsenate, but primarily as arsenic trioxide. Elemental arsenic is a shiny, gray element that possesses both metallic and non-metallic properties. It is present naturally in the environment at low concentrations and is used industrially as arsenic trioxide, in pigment production, glass manufacturing, textile printing, tanning, and in antifouling paints. As arsenic trichloride, it is used in the manufacture of pharmaceuticals (Sittig, 1991).

Metallic arsenic is used as an alloying agent in the smelting of copper, zinc and lead ores.

Chemical and Physical Properties

AW:	74.91	BP:	613°C
SG:	5.72 at 20°C	MP:	817°C
		VP:	1 mmhg at 372°C

Sol. (water): insoluble (except for some salts).

Fate and Transport

Arsenic is generally quite mobile in the environment although, because it occurs in four valence states, it cannot be characterized easily. The most common fate processes of arsenic in the environment are speciation between the +3 and +5 valence states, volatilization, sorption, and biotransformation (EPA, 1984).

In surface waters, arsenic is significantly influenced by the presence of biota. Arsenic is readily bioaccumulated but is often biotransformed to methylated arsenicals, volatile compounds that evaporate from surface waters (EPA, 1985).

In surface soils, arsenic is known to sorb to clays, iron oxides, and particulate matter. The presence of these materials would greatly retard arsenic's leachability (EPA, 1984). In soils with low sorptive capacity, arsenic will leach into ground water, where it would likely be transported readily.

The primary means of removal of atmospheric arsenic are wet and dry precipitation (EPA, 1984).

Pharmacokinetics

Soluble arsenic salts are known to be easily absorbed through the gastrointestinal lining in humans and animals (Coulson, et al., 1935). In humans, peak blood arsenic levels (98 percent of total arsenic ingested) were reached after only 24 hours following the ingestion of 8.25 mg As in three doses (EPA, 1985). Arsenic is distributed, in humans, primarily to the nails, hair, bone and skin, and to a lesser extent, the heart, liver, kidneys and lungs (Kadowski, 1960).

In laboratory animals, arsenic was shown to distribute to the liver, kidneys, lung, spleen, skin, and brain. It is removed rapidly from all organs except for the latter two (EPA, 1985).

Arsenic generally is metabolized to methylated arsenicals such as monomethyl and dimethyl arsenic. Buchet, et al. (1981) reported that 25 percent of arsenic, administered as arsenate to human volunteers, was excreted in the urine as inorganic arsenic, 25 percent as monomethyl and 50 percent as dimethyl arsenic.

Lanz, et al. (1950) noted that, in contrast to a humans metabolic processes, rats retain arsenic in their red blood cells for as long as 180 days. Humans typically remove 90 percent of ingested arsenic within 4 days.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Arsenic is known to be highly toxic to humans. Subchronic exposure of infants to 3 mg/day arsenic in contaminated milk caused several deaths, according to Hamamoto (1955). Oral exposure to 50 to 300 mg of inorganic arsenic was the probable cause of death to several workers, according to Vallee, et al. (1960). From these two case studies, a subacute lethal dose of 0.6 mg/kg/day was estimated for humans (ATSDR, 1989).

Oral exposure of humans to arsenic is known to cause nausea, vomiting, diarrhea, and other gastrointestinal disorders (ATSDR, 1989). Long-term exposure results in paresthesia, weakness, anorexia, bronchitis, and various skin disorders (EPA, 1985). It was reported that children exposed to 0.8 mg/L arsenic in drinking water exhibited evidence of myocardial infarction and arterial thickening (ATSDR, 1989). In Taiwan, chronic exposure to arsenic in drinking water was thought to cause gangrene in the feet and toes in 0.9 percent of the population ("Blackfoot disease"). Concentrations were reported to average 0.5 mg/L arsenic (Tseng, 1977; Tseng et al., 1968).

Exposure to arsenic doses ranging from 2.8 to 5.7 mg/kg/day in newborn Rhesus monkeys caused death in 75 percent of monkeys in the 5.7 mg/kg/day group and death in two of the seven monkeys in the 2.8 mg/kg/day group. Death was attributed to hemorrhaging, edema, and necroses of the brain (EPA, 1985). All of the surviving monkeys had normal cardiovascular and neurological function.

Teratogenic and Other Developmental Effects

Parenteral administration of 10 to 45 mg/kg/day of sodium arsenate to pregnant rats, mice, and hamsters has been reported to increase the frequency of fetal malformations (ATSDR, 1989). Arsenic has also been shown to be teratogenic when administered orally. Hood, et al. (1977) found that a single gavage dose of 29 mgAs^{+5}/kg administered to pregnant mice on day 9, 10 or 11 of gestation resulted in death or resorption of 17-26 percent of the fetuses. Of the live fetuses, 10-16 percent were below average in weight and 1-3 percent were severely malformed.

Mutagenic Effects

Arsenic is known to cause DNA fragmentation and sister chromatid exchange in several cell types in laboratory animals and humans (ATSDR, 1989).

Carcinogenic Effects

Arsenic is classified by EPA as a Class A carcinogen, a known human carcinogen. Oral exposure to elevated levels of arsenic unequivocally increases the risk of skin cancer. Tseng, et al. (1968) and other researchers noted a significant increase in several skin cancer types in populations exposed to elevated arsenic levels in the drinking water (ATSDR, 1989).

Numerous studies of smelter workers have indicated that occupational exposure to arsenic is directly associated with lung cancer (IRIS, 1990). Matanoski, et al. (1981) reported that residents surrounding a pesticide manufacturing plant were at a greater risk of contracting lung cancer than the normal population.

In a supplemental paper, Tseng reported a significant increase in the incidence of bladder, lung, kidney, and colon cancer in a Taiwanese population exposed to elevated arsenic levels in their drinking water.

All evidence from human case studies indicates that chronic exposure to arsenic causes cancer. In laboratory studies, however, attempts to induce cancer in animals have been inconclusive or negative (ATSDR, 1989). Some studies, in which the arsenic

retention time has been artificially increased, have shown that arsenic will produce tumors in rats (ATSDR, 1989).

Ecotoxicity

Arsenic compounds are acutely toxic to both freshwater and saltwater species of organisms, with early life stages being the most susceptible (ICF, 1985). Toxicity can occur at levels as low as 40 µg/l in juvenile aquatic species. Saltwater fish species are susceptible to arsenic's toxic effects at levels around 15 mg/l, but some invertebrates are affected at around 508 µg/l (ICF, 1985).

Information pertaining to arsenic's toxicity to terrestrial species (other than laboratory animals) was not located in the available literature.

Standards, Criteria and Guidelines

EPA Class A Carcinogen

Oral Slope Factor:	1.75×10^{0} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	1.51×10^{1} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	3.0×10^{-4} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	1.0×10^{-3} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.05 mg/L
AWQC:	Water and Fish Consumption - 0.0022 µg/L
	Fish Consumption - 0.018 µg/L

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BARIUM

Use

Barium, a silver white metal, is produced by reduction of barium oxide. In its metallic state, it is used for the removal of residual gas in vacuum tubes and in alloys with nickel, lead, calcium, magnesium, sodium, and lithium.

Barium compounds are used in the manufacture of a variety of products including lithopone (a white pigment in paints), chlorine, sodium hydroxide, valves, and green flares. They are used in synthetic rubber vulcanization, x-ray diagnostic work, glassmaking, papermaking, beet-sugar purification, and animal and vegetable oil refining. They can be found in use in the brick and tile, pyrotechnics, and electronics industries. These compounds are found in lubricants, pesticides, glazes, textile dyes and finishes, pharmaceuticals, and in saltwater cements. Barium is used as a rodenticide, a flux for magnesium alloys, a stabilizer and mold lubricant in the rubber and plastics industries, an extender in paints, a loader for paper, soap, rubber, and linoleum. It is used as a fire extinguisher for uranium and plutonium fires as well (Sittig, 1991).

Chemical and Physical Properties

AW: 137.3 MP: 725°C
SG.: 3.5 BP: 1640°C
Sol. (water): decomposes, combines with sulfate present in natural waters to form $BaSO_4$, which has a solubility of 1.6 mg/l at 20°C.
Sol. (organics): alcohol, insoluble in benzene.

Fate and Transport

Being extremely reactive, barium decomposes in water, and readily forms insoluble carbonate and sulfate salts. In surface or ground waters it is generally found in solution only in trace amounts. Large amounts will not dissolve because of the sulfate found in most natural water (barium sulfate has a low solubility). In water that contains more than a few ppm sulfate, barium will not dissolve at more than a few ppm. Barium sulfate may become considerably more soluble in the presence of chloride and other anions.

It is rare to find barium in drinking water at concentrations greater than 1 mg/l. Atmospheric transport of barium, in the form of particulates, can occur. Bioaccumulation is insignificant for barium (ICF, 1985).

Because of its formation of water-insoluble salts and its inability to form soluble complexes with humic and fulvic materials, barium is not expected to be very mobile in soils. However, some water insoluble barium compounds may be solubilized under acidic conditions and thereby move back into groundwater (US EPA, 1984).

Pharmacokinetics

Barium and its compounds can affect the heart, lungs, central nervous system, skin, respiratory system, and eyes (Sittig, 1991).

Although quantitative data for the absorption of barium from the GI tract was not found in the literature reviewed, McCauley and Washington (1983) found relative absorption rates for barium salts with barium chloride having greater absorption than barium sulfate which, in turn, had greater absorption than barium carbonate.

Gore and Patrick (1982) reported that barium sulfate administered intratracheally to rats was concentrated in the area immediately beneath the basement membrane within 24 hrs. and remained in this area for at least 7 days. This suggests a degree of absorption from the respiratory tract.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Wones et al. (1990) administered barium (as barium chloride) in the drinking water of eleven healthy male volunteers. Subjects ranged in age from 27-61 years and had no previous history of diabetes, hypertension, or cardiovascular disease. Diets were strictly controlled throughout the 10-week study. Subjects were given 1.5 l/day of distilled, charcoal-filtered water with 0 mg/l barium for weeks 0-2, 5 mg/l for weeks 3-6, and 10 mg/l for weeks 7-10.

No changes in blood pressures or serum chemistry were detected. An increase in serum calcium levels, attributed to a decrease in serum albumin levels, although statistically significant, was not clinically significant. An NOAEL of 0.21 mg/kg/day was identified in this study.

Brenniman and Levy (1984) conducted a retrospective epidemiological study by comparing human mortality and morbidity rates in populations ingesting elevated barium levels (2-10 mg/l) in their drinking water to populations ingesting little or no barium (less than or equal to 0.2 mg/l). Differences in mortality rates from cardiovascular diseases were significantly higher in the communities with elevated barium. However, these differences were largely in the 65 and over age group and did not take population mobility, the use of water softeners, or medications into account. Differences in blood pressure, prevalence of hypertension, stroke, and heart and renal disease were also measured and no significant differences occurred between the populations.

In a variety of animal studies (McCauley, 1985; Perry et al., 1983; Schroeder and Mitchener, 1975a,b; Tardiff et al., 1980) no signs of barium toxicity were found at any dose level. Animals treated with the highest dose of barium, 1000 mg/l in McCauley's study did exhibit ultrastructural changes in the kidney glomeruli and the presence of myelin figures (IRIS).

Taransenko et al. (1977) reported on the effects barium carbonate dust had on rats when inhaled. Male rats were exposed to the dust at levels of 5.2 and 1.15 mg/m^3, 4 hrs/day for 6 months. While the rats in the high dose group experienced what Taransenko called "general toxic effects" (decreased body weight, changes in hematologic parameters), the low dose animals exhibited no toxic effects.

Workers exposed to barium dust have been shown by occupational studies to develop "baritosis." No symptoms are illustrated other than a significantly higher incidence of hypertension (IRIS).

Teratogenic and Other Developmental Effects

Taransenko et al. (1977) reported that male rats exposed to an atmospheric concentration of 22.6 mg $BaCO_3/m^3$ for one cycle of spermatogenesis exhibited decrease number of spermatozoids and a lower percentage of motile sperm forms. Female rats exhibited increased mortality in subsequent litters and a general underdevelopment of newborn pups when exposed to 13.4 mg $BaCO_3/m^3$ for 4 months. An atmospheric concentration of 3.1 mg $BaCO_3/m^3$ produced no systematic effects, although some ovarian follicle atresia was observed. When males exposed to an atmospheric concentration of 5.2 mg $BaCO_3/m^3$, 4 hours/day for 4 months when mated with unexposed females, increased mortality of the fetuses resulted.

Mutagenic Effects

Nishioka (1975) found that repair deficient strains of *Bacillus subtillis* did not exhibit an increased mutation frequency when exposed to barium chloride. Loeb et al. (1978) obtained negative results as well in tests of the induction of errors in viral DNA transcription in vitro.

Carcinogenic Effects

Barium has not been evaluated by the US EPA for evidence of human carcinogenic potential (IRIS).

McCauley et al. (1985) found no carcinogenic effect in a study of the histological and cardiovascular effects of drinking water containing 0,10,100, and 250 mg/l barium for 16,36, and 68 weeks on male Sprague-Dawley rats. Female (rats???) were exposed to 0 or 250 mg/l barium for 46 weeks.

Schroeder and Mitchener (1976a,b) investigated the carcinogenicity of barium acetate in drinking water to both rats and mice. The observed differences in tumor incidence in the rats was insignificant statistically and there was essentially no difference in tumor incidence in the mice.

Ecotoxicity

There is sufficient sulfate or carbonate present in most natural water to precipitate any barium present in the water as a virtually insoluble, non-toxic compound. Therefore, it would require a soluble barium concentration of at least 50 mg/l before toxicity to both fresh and marine aquatic life would be expected (US EPA, 1986). Data pertaining to the toxicity of barium to terrestrial life, domestic or wild, was not found in the literature reviewed.

Standards, Criteria, and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	5.0×10^{-2} mg/kg/day
Chronic Inhalation RfD:	1.0×10^{-4} mg/kg/day
Subchronic Oral RfD:	5.0×10^{-2} mg/kg/day
Subchronic Inhalation RfD:	1.0×10^{-3} mg/kg/day
MCL:	2.0 mg/l
AWQC:	Water and Fish Consumption: 1 mg/l
	Fish Consumption: NA

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BERYLLIUM

Use

Beryllium is a gray metal that is used as an alloy in numerous industries because of its light weight and high tensile strength. In the atomic energy field, it is used as a moderator in fission reactors and as a reflector to reduce leakage from the core (Sittig, 1991). Beryllium is alloyed with numerous other metals. As an alloy with copper, beryllium is used in machinery parts subjected to heavy wear or extreme vibration, in springs, in radar components and in non-sparking tools. As an alloy with nickel, beryllium is used on airplanes, in diamond drill-bit matrices, and in watch-balance wheels. Beryllium-bronze is used in switch parts, watch springs, diaphragms, shims, and bushings (Sittig, 1991)

Exposure to beryllium is generally associated with the milling and alloying processes and not in the mining of the beryl ore.

Chemical and Physical Properties

AW:	9.012	BP:	2970°C
SG:	1.85 at 20°C	MP:	1278°C

Sol. (water): insoluble (except for beryllium salts)
Sol. (organics): dilute acid and alkali.

Fate and Transport

The majority of beryllium releases, most of which occurs as a result of coal combustion and milling processes, are to the atmosphere. Because these are releases of particulate, rather than dissolved beryllium, deposition is the most common fate. Generally, the ultimate destination of atmospheric beryllium is the soil (EPA, 1987). In the soil, beryllium tends to sorb to particulate matter in the relatively insoluble form of beryllium oxide.

When deposed or released to surface waters, the more commonly used beryllium compounds, most of which are water soluble, are hydrolyzed to beryllium hydroxide (ICF, 1985). Because beryllium hydroxide is relatively insoluble in the pH range of most surface waters, it remains stable and sorbs to any particulate matter present (ICF, 1985).

Pharmacokinetics

Beryllium is known to accumulate in the lungs of humans and laboratory animals after atmospheric exposure to elevated concentrations. Reeves, et al. (1967) showed that rats exposed to an atmospheric beryllium concentration of 35 $\mu g/m^3$ for 7 hours/day, 5 days/week for 72 weeks accumulated 13.5 µg in the lungs after 36 weeks of exposure. Examinations of

human lung tissue revealed that beryllium concentrations in the lungs of occupationally exposed workers reach levels two to ten times as high as those in normal human lung tissues (EPA, 1987).

Ingested beryllium has, in some studies, been shown to be absorbed slightly through the gastrointestinal lining (less than 1 percent). However, Reeves (1965) exposed rats to beryllium in drinking water at an average daily ingestion concentration of either 6.6 or 66.6 μg Be. Sixty to ninety percent of the ingested beryllium was eliminated in the feces, indicating that an appreciable amount was ingested.

Absorbed beryllium accumulates primarily in the skeleton. Of the soft tissues, the liver and kidneys accumulate the most (EPA, 1987).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Acute occupational exposure to atmospheric beryllium is known to cause lung disease. In a study of six fatal cases of beryllium poisoning, Frieman and Hardy (1970) reported that death occurred between 17 and 70 days after exposure. Interstitial pneumonitis was determined to be the cause of the fatalities.

Chronic exposure to beryllium can also result in lung disease. Hardy and Tabershaw (1946) reported that 5 of 17 workers studied in a fluorescent lamp manufacturing plant died from chronic beryllium exposure. The cause of death was noted to be an inflammation of cells within the alveoli.

It has also been noted that chronic exposure to beryllium can cause enlargement of the heart, liver and spleen; cyanosis; and kidney stone development (ICF, 1985).

Teratogenic and Other Developmental Effects

Three major studies were located in the available literature that provide inconclusive evidence as to the teratogenicity of beryllium. It appears as though no reproductive or teratogenic effects are caused by beryllium (EPA, 1987).

Mutagenic Effects

Beryllium has been proven to be mutagenic to cultured mammalian cells. Miyaki, et al. (1979) noted that Chinese hamster V79 cells, induced with beryllium chloride, were six times more likely to mutate than control V79 cells. The same results were noted by Hsie, et al. (1979) in Chinese hamster ovary cells.

Human lymphocyte cells are also known to mutate more frequently when exposed to beryllium compounds. Larramendy, et al. (1981) exposed human lymphocytes to beryllium sulfate in a single dose of 0.25 µg Be/ml. A six-fold increase in chromosomal aberrations was noted during cell division.

Carcinogenic Effects

Carcinogenicity case studies of occupationally exposed workers have been inconclusive. Of the studies performed, external factors were not appropriately taken into account. In most of the studies, the effects of cigarette smoking were not factored in but, when they were, no significant increase in tumors was noted (IRIS).

Studies performed on laboratory animals indicate that beryllium is carcinogenic. Schroeder and Mitchener (1975) reported a slightly significant increase in the incidence of unspecified cancerous growths in Long-Evans rats administered 5 ppm beryllium sulfate in drinking water for a lifetime.

In numerous studies, osteogenic sarcomas were induced in rabbits exposed to beryllium compounds via intravenous injection (IRIS).

Tumors have also been induced in Wistar rats through the intratracheal injection of metallic beryllium, beryllium-aluminum alloys, and beryllium oxide. Adenomas, adenocarcinomas, and malignant lymphomas were all noted in the lungs of the test rats (IRIS, 1990).

Ecotoxicity

Beryllium's toxicity to freshwater aquatic life appears to be affected by the amount of calcium carbonate in the water. Acute toxicity values for the Fathead Minnow changed from 150 µg/l in water with 20 mg/l calcium carbonate, to 20,000 µg/l in water with 400 mg/l calcium carbonate (ICF, 1985). From the limited data available, beryllium is thought to be mildly toxic to saltwater aquatic species.

Changes in skeletal growth were noted in poultry and livestock after soluble beryllium salts were added to their diets. Rachitis, a condition in which the long bones develop improperly, was noted to occur after the induction of 0.125 percent beryllium carbonate into the diet (IRIS).

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	4.3×10^{0} $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	8.4×10^{0} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	5.0×10^{-3} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5.0×10^{-3} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.001 mg/l
AWQC:	Fish and Water Consumption - 0.0037 µg/L
	Fish Consumption - 0.0641 µg/L

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CADMIUM

Use

Elemental cadmium is a soft white metal similar to lead and zinc in texture and in other physical properties.

Cadmium is obtained as a byproduct during the production of zinc. Commercially, cadmium is used in the metal plating industry; as a stabilizer in paints, pigments, and plastics; and as an energy storage medium in batteries. It is also used in pesticides, as an alloy additive, and in chemical reagents. Cadmium may escape into the air from zinc, lead, or copper smelters. Naturally occurring levels of cadmium in surface and ground water normally fall in the range of 1-10 g/liter (EPA, 1985).

Physical and Chemical Properties

AW: 112.41
SG: 8.642
BP: 765°C
MP: 321°C
VP: 1 mmHg at 394°C
Sol. (water): metal is insoluble, salts of metal are soluble
Sol. (organics): variable

Fate and Transport

The primary vehicle for cadmium exposure in a non-occupational setting is through the ground water. Cadmium is relatively mobile in aquatic environments and sorbs to organic material found in soils (EPA, 1984). It is thought to be transported slowly by ground water, but no comprehensive studies have been performed in this regard. High cadmium levels are often found in ground water surrounding smelting and plating facilities (Sittig, 1991). Occupationally, workers can be exposed to cadmium in the form of dust or fumes.

Pharmacokinetics

Cadmium is absorbed moderately in the lungs but quite poorly in the gastrointestinal tract (1 to 6 percent in both humans and animals). The primary excretory route for absorbed cadmium is the urine (ATSDR, 1989). Urinary excretion is slow, however, and cadmium has a strong tendency to accumulate in the body (mostly in the liver and renal cortex) over time in exposed humans and in animals (cadmium binds tightly to the protein metallothionein or its cellular components). The half lives of cadmium and its compounds in the body range from 17 to 38 years (ATSDR, 1989). Measurements of alveolar absorption in rats indicate 60 to 70 percent absorption over time. Calculations based on increased body burden in smokers compared to that in nonsmokers suggest that respiratory absorption in humans is probably about 30 to 60 percent (ATSDR, 1989). The absorption of cadmium following oral administration of laboratory animals, and presumably humans, is not a simple process and is

modified by many factors including chemical form solubility dose, age, diet, and by the presence of other metals. Small quantities of cadmium may be absorbed through the skin but dermal absorption is not normally significant relative to total cadmium absorption (ATSDR, 1989). In general, soluble compounds such as $CdCl_2$ are better absorbed and are more toxic than highly insoluble compounds such as CdS. (ATSDR, 1989).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

In the case of severe intoxication, sensory disturbances, liver injury, and convulsions may occur. In fatal intoxications, this is followed by shock and/or renal failure and cardiopulmonary depression (EPA, 1985). Exposure to concentrations of 40 to 50 mg/m^3 for 1 hour and 9 mg/m^3 for 5 hours has resulted in fatalities. LD_{50} values in animals exposed to cadmium oxide fumes range from 500 to 15,000 mg/m^3 minute (ATSDR, 1989). Acute oral LD_{50} values in animals for cadmium oxide and common cadmium salts range from 50 to 350 mg/kg (ATSDR, 1989).

Renal effects: The kidney is generally recognized as the most sensitive tissue to low-level cadmium exposure, the major effect being impaired tubular reabsorption. Rats receiving water containing cadmium at 30 or 100 mg/liter developed significant ($p<0.05$) proteinuria after 6 weeks of exposure (EPA, 1985). Various studies indicate that tubular dysfunction does not generally occur in humans until a renal cortical concentration of approximately 200 µg/g wet weight is reached (ATSDR, 1989). Using this figure, it was estimated that a daily oral intake of 352 µg/day over 50 years would not exceed the critical level of cadmium in the renal cortex. A more recent study in which epidemiological studies were reviewed, however, concluded that an average oral exposures of about 200 µg/days will cause tubular proteinuria in about 10 percent of an exposed population by age 45 (ATSDR, 1989). It was also estimated that 10 percent of a working population exposed via inhalation to 50 $\mu g/m^3$ would develop proteinuria in 10 years. (ATSDR, 1989).

Hepatic effects: The next highest tissue levels of cadmium are found in the liver. While structural changes were observed following cadmium exposure in food and water to rats and rabbits, clinical tests revealed normal hepatic function. There is little evidence for liver dysfunction in chronically exposed human populations but hepatic levels may serve as a useful index of exposure and a predictor of future renal dysfunction (ATSDR, 1989).

Cardiovascular effects: Certain animal studies have indicated that increases in average systolic blood pressure occur following exposure to cadmium acetate in the drinking water (0.5 mg/kg/day); not all investigations have succeeded in confirming these findings and other factors may confound the effects of cadmium (ATSDR, 1989). The role of cadmium is human hypertension is uncertain (ATSDR, 1989).

Pulmonary effects: Inhalation exposure to high levels of cadmium oxide fumes is intensely irritating to respiratory tissues (ATSDR, 1989).

Gastrointestinal effects: In humans, the symptoms of cadmium toxicity following acute oral exposure include nausea, vomiting, diarrhea, abdominal pain, and salivation (ATSDR, 1989).

Other systemic effects: Weak evidence exists indicating skeletal effects in humans and animals exposed chronically to cadmium. Studies revealed that relatively low doses of cadmium can alter the immune response in animals (at very low renal cadmium concentrations ranging from 0.3 to 6.0 μg/g) (ATSDR, 1989). Parenteral injection of cadmium has been observed to cause severe acute pathological changes in the gonads of animals (ATSDR, 1989). Exposure by injection of male rats with 2.2 mg/kg of $CdCl_2$ resulted in swelling and inflammation of testes, followed by necrosis and atrophy, in several studies. Another common effect in cadmium-exposed animals is anemia (ATSDR, 1989).

Teratogenic and Other Developmental Effects

Sutou, et al. (1980) administered cadmium at 0, 0.1, 1.0, and 10.0 mg/kg/day (as $CdCl_2$) orally to male and female adult rats for 6 weeks. Males and female were mated for 3 weeks, and cadmium was administered during the mating period. Pregnant females were given cadmium during the gestation period. The number of total implants and live fetuses decreased significantly in the 10 mg/kg group, and the number of resorbed fetuses was markedly increased. Fetuses showed decreased body weight, and delayed ossification of the sternebrae and caudal vertebrae. Ahokas, et al. (1980) observed, in a rat drinking-water-study, fetal growth retardation in animals whose dams were exposed to 100 mg cadmium/L but not in those exposed to 0.1 or 10 mg cadmium/L during gestation. The most common finding is the decreased weight of offspring, with ingestion exposure, usually without significant teratogenic or developmental effects (ATSDR, 1989). Cadmium exposure has not been observed to cause teratogenic or other developmental effects in exposed humans (ATSDR, 1989).

Mutagenic Effect

Studies to assess the mutagenic activity of cadmium, in *Salmonella typhimurium, E. coli,* and yeast, have been inconclusive (ATSDR, 1989). Recombination assays in ***Bacillus subtilis*** have yielded weak positive responses (ATSDR, 1989). Cadmium has been shown to be mutagenic both in the mouse lymphoma assay and in the Chinese hamster cell assay (ATSDR, 1989). Chromosomal aberration studies on human lymphocytes from exposed workers and in human and animal cells treated with cadmium *in vitro* have produced conflicting results (ATSDR, 1989).

Carcinogenic Effects

EPA has evaluated the weight of evidence on the carcinogenicity of cadmium and has concluded that cadmium is a probable human carcinogen (Group B1) by inhalation (ATSDR, 1989/IRIS). An occupational study of smelter workers by Thun, et al. (1985) revealed a two-fold excess risk of lung cancer but confounding factors could not be ruled out. Wistar rats exposed to cadmium chloride developed significant increases in lung tumors (Takenaka, et al., 1983). No sufficient data exists to consider cadmium as carcinogenic by the oral route, nor is there evidence that cadmium, via the dermal route, is carcinogenic to either animals or humans.

Ecotoxicity

The acute LC_{50} values for cadmium exposure in freshwater fish and invertebrates generally range from 100 to 1,000 µg/liter. Salmoids, being very sensitive, would be at the lower end of this range. Saltwater species appear to be, in general, 10-times more tolerant to the acute effects of cadmium than freshwater species (ICF, 1985). Cadmium is strongly accumulated by all organisms (ATSDR, 1989). Bioconcentration factors (BCFs) for cadmium in freshwater range from 164 to 4,190 for invertebrates and from 3 to 2,213 for fish. BCFs for saltwater invertebrates range from 5 to 3,160 (EPA, 1986).

Freshwater acute values for cadmium are available for species in 44 genera and range from 1.0 µg/L for rainbow trout to 28,000 µg/L for mayflies. Chronic tests conducted for cadmium on 12 freshwater fish species and 4 invertebrate species revealed chronic values ranging from 0.15 µg/L for *Daphnia magna* to 156 µg/L for the Atlantic salmon. Acute-chronic ratios, available for eight species, range from 0.9021 for the Chinook salmon to 433.8 for the flagfish (EPA, 1986). Freshwater aquatic plants are affected by cadmium at concentrations ranging from 2 to 7,400 µg/L. The major toxic effect observed in freshwater aquatic plants was growth reduction.

Saltwater acute values for cadmium in five species of fish range from 577 µg/L for Atlantic silverside to 114,000 µg/L for juvenile mummichog. Invertebrate acute values (30 species) range from 15.5 µg/L for a mysid to 135,000 µg/L for an oligochaete worm. Acute toxicity of cadmium usually increases as salinity decreases. Chronic cadmium exposure has been shown to significantly affect the growth of bay scallops at 78 µg/L and the reproduction of certain copepods at 44 µg/L (EPA, 1986).

Standards, Criteria and Guidelines

EPA Class B1 Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	$6.3x10^{0}$ $(mg/kg/day)^{-1}$
Chronic Oral RfD:	1.0×10^{-3} mg/kg/day (food)
	5.0×10^{-4} mg/kg/day (water)
Chronic Inhalation RfD:	Currently under review by EPA
Subchronic Oral RfD:	5.0×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.005 mg/l
AWQC:	Water and Fish Consumption - 10 µg/l
	Fish Consumption - NA

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CHROMIUM

Use

Chromium and chromium compounds are used in stainless and alloy steels, refractory products, tanning agents for leather, pigments, electroplating, catalysts, and in corrosion resistant products (ACGIH, 1991).

Chemical and Physical Properties

AW: 51.996 BP: 2672°C
SG: 7.20 at 28°C MP: 1857 ±20°c
Sol. (water): insoluble, some compounds are soluble.

Fate and Transport

Chromium (VI) is soluble in water and is transported easily in ground water. It may exist in aquatic systems as water soluble, complex anions, and may persist for long periods of time (EPA, 1984a). Chromium (VI) may react with organic matter in the soil or surface waters to form trivalent chromium, therefore chromium (VI) may exhibit a shorter lifespan in soils with high organic content (EPA, 1984a).

The primary means by which chromium (III) is transported out of soils and surface waters is through aerosol formation and runoff. It is also hydrolyzed to chromium hydroxide. Leaching does not generally occur with chromium (III) because it is generally present as insoluble chromium trioxide.

Pharmacokinetics

The amount of ingested chromium (VI) and (III) absorbed is estimated to be 5 percent and 3 percent respectively. Donaldson and Barreras (1966) fed $Na_2{}^{51}Cr(VI)0_4$ to rats and humans and $^{51}Cr(III)Cl_3$ to humans. Based on mean urinary excretion of $^{51}Cr(VI)$, absorption was estimated to be 2.1 percent in humans. In rats, 2 percent of the administered dose was absorbed, based on fecal excretion of $^{51}Cr(VI)$. Based on fecal excretion of $^{51}Cr(III)$, absorption was estimated to be 0.4 percent in humans. However, when $Na_2{}^{51}Cr(VI)0_4$ was administered introduodenally (in humans) or intrajejunally (in rats), absorption was estimated to be 50 and 25 percent, respectively. When $^{51}Cr(III)Cl_3$ was administered introduodenally, absorption was not appreciably changed. A study by Langard, et al. (1978) indicates that water-soluble chromium (VI) is absorbed rapidly via inhalation. Rats were exposed to zinc chromate dust at a level of 7.35 mg/m^3. After 0, 100, 250, and 350 minutes of exposure, the concentrations of chromium in the blood (µg/ml) were 0.007, 0.024, 0.22, and 0.31, respectively. Chromium (III) is absorbed slowly via inhalation. Baetjer et al. (1959)

administered $^{51}Cr(III)Cl_3$ to guinea pigs intratracheally. Only 4 percent of the administered dose was detected in the blood and tissues 10 minutes post-treatment; 69 percent remained in the lungs. 45, 30, and 12 percent of the administered dose was detected in the lungs 1, 30, and 60 days post-treatment, respectively.

Human Toxicity

Noncarcinogenic

Systemic Effects

Bloomfield and Blum (1928) examined 23 men from six chromium plating plants in the U.S. Fourteen of the workers typically spent 2-7 hours/day over vats of chromic acid, which generated airborne hexavalent chromium ranging from 0.12-5.6 mg/m^3. These men experienced nasal tissue damage, including perforated septa, ulcerated septa, chrome holes, nosebleed, and inflamed mucosae. The nine remaining workers not directly exposed to chromium vapors had only inflamed mucosae.

Mackenzie, et al. (1958) exposed groups of rats, both male and female, to potassium dichromate (0-25 ppm of hexavalent chromium) in drinking water for 1 year. No effects were observed at any level of treatment. Pertinent data regarding subchronic exposure of animals to hexavalent chromium via inhalation were not located in the literature (EPA, 1984a).

Ivankovic and Preussman (1975) exposed groups of 60 male and female rats to 0, 1, 2, or 5 percent $Cr(III)_2O_3$ in baked bread, 5 days/week for 600 feedings. The average total amounts of ingested $Cr(III)_2O_3$ were given as 0, 360, 720, and 1800 g/kg bw. No adverse effects were observed at any dose level.

Teratogenic and Other Developmental Effects

The literature available on teratogenic effects resulting from ingestion of chromium is limited. However, several forms of chromium (including chromium (III)), when administered to pregnant rats by stomach intubation in the form of GTF (obtained from yeast), have been found to cross the placental barrier and be recovered by the fetus (EPA, 1985).

Mutagenic Effects

Compounds of both chromium (III) and chromium (VI) increase noncomplementary nucleotide incorporation into DNA with chromium (VI) being effective at lower doses. Exposure of cells from rat liver and kidney to chromium (VI) leads to increased cross-linking in DNA. Positive Ames tests for chromium

(VI) have been reported; however chromium (III) exerted no effect at relatively high concentrations (presumably because of its inability to penetrate cells), (EPA, 1985).

Carcinogenic Effects

Data regarding the carcinogenicity of inhaled chromium (VI) is well established for occupational exposure in humans. The effects are observed only in the respiratory passages and in the lungs (EPA, 1985).

Numerous epidemiological studies indicate that various forms of chromium (VI) cause lung cancer as a result of chronic exposure (Machle and Gregorius, 1948). It has been estimated that workers in the chromate pigment industry who had developed lung cancer were exposed to 0.01 to 0.15 mg/m^3 of water soluble chromium and 0.1 to 0.58 mg/m^3 of water insoluble chromium. From subsequent studies, it appears that water insoluble compounds of chromium (VI) resulted in the increase in lung cancer (ACGIH, 1984).

There is inadequate evidence to determine whether or not oral exposure to chromium (III) can lead to cancer. Rats exposed to chromium (III) at 293, 586, or 1,4676 mg/kg/day in the diet (administered as chromium oxide pigments) for 2 years, displayed no increase in the tumor rates over that of the control animals (EPA, 1985).

Ecotoxicity

Chromium is an essential nutrient and is accumulated in a variety of aquatic and marine biota, especially benthic organisms, to levels much higher than in ambient water. Levels in biota, however, are usually lower than levels in the sediments. Passage of chromium through the food chain can be demonstrated (ICF, 1985). The food chain appears to be a more efficient pathway for chromium uptake than direct uptake from seawater (ICF, 1985). Water hardness, temperature, dissolved oxygen, species, and age of the test organism all modify the toxic effects of chromium on aquatic life. Chromium (III) appears to be more acutely toxic to fish than chromium (VI), yet the reverse is true in long-term chronic exposure studies (ICF, 1985). None of the plants normally used as food or animal feed are chromium accumulators. Chromium absorbed by plants tends to remain primarily in the roots and is poorly translocated to the leaves. There is little tendency for chromium to accumulate in food chains in the trivalent inorganic form. Organic chromium compounds, about which little is known, can have significantly different bioaccumulation tendencies (ICF, 1985).

Standards, Criteria and Guidelines

EPA Class A Carcinogen (Hexavalent Chromium)

Oral Slope Factor:	NA
Inhalation Slope Factor:	$4.2x10^{1}$ **(mg/kg/day)$^{-1}$** (VI)
Chronic Oral RfD:	5.0×10^{-3} **mg/kg/day** (VI)
	1×10^{0} **mg/kg/day** (III)
Chronic Inhalation RfD:	5.71×10^{-7} **mg/kg/day** (VI and III)
Subchronic Oral RfD:	2.0×10^{-2} **mg/kg/day** (VI)
	1.0×10^{1} **mg/kg/day** (III)
Subchronic Inhalation RfD:	5.71×10^{-6} **mg/kg/day**
	5.71×10^{-6} **mg/kg/day**
MCL:	0.1 mg/l (total)
AWQC:	Water and Fish Consumption - 170 mg/L (III)
	Fish Consumption - 3433 mg/L (III)

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COBALT

Use

Cobalt (Co) is a silver-grey, hard, brittle, magnetic metal. It is used in alloy with nickel and aluminum in the manufacture of permanent magnets. Alloys with nickel, aluminum, copper, beryllium, chromium, and molybdenum are used in the electrical, automobile and aircraft industries. Tool steels include Co to improve their cutting qualities. Tungsten carbide tool manufacture utilizes Co as a binder. Various Co compounds are used as pigments in enamels, glazes and paints; as catalysts in afterburners; and in the glass, pottery, photographic, and electroplating industries. Radioactive Co is used in the treatment of cancer. (Sittig, 1991).

Chemical and Physical Properties

Atomic Symbol: Co
AW: 58.93 BP:2870°C
SG: 8.9 at 20°C MP:1495°c
Sol.(water): insoluble; soluble in acid

Fate and Transport

ICF (1985) states that photolysis, volatilization, and bio-transformation are not significant environmental fates for Co. Atmospheric transport of Co can occur, however. In natural aquatic systems, very little Co is present in soluble form, in fact, concentrations greater than 10 µg/liter are rare (ICF, 1985). In aquatic and terrestrial systems, absorption to clay minerals and hydrous oxides of iron, manganese, and aluminum often present in the clay fractions of sediments and soils appears to be the most important control on the mobility of Co (ICF, 1985). Eh, pH, and the concentrations of Co and competing compounds are the principal factors controlling absorption/desorption. Other fate processes include chelation with organic compounds, solubilization by bacteriological activity and slight bioaccumulation (ICF, 1985).

Pharmacokinetics

Co is an essential micronutrient in animals and man. The body, therefore, is capable of metabolizing moderate quantities of Co compounds. Co is an important element in Vitamin B_{12} and certain enzymes, and is associated with the production of erythropoietin, the red cell stimulating factor (Clayton and Clayton, 1981). Schroeder (1967) reports that the normal Co balance in man includes a daily food intake of 140-580 mg/day, a daily water intake of 0-10 mg/day and an inhalation intake <0.1 mg/day. Output includes 120-330 mg/day in urine, 23-60 mg/day in feces, and 6 mg/day in sweat and hair. Forbes et al. (1954) found human tissue

concentrations of Co to range form 0.01 ppm for fat, nerve, muscle and the GI tract to 0.06 ppm for liver.

Gastrointestinal absorption of Co and Co compounds is dose-dependent. Smaller doses, on the order of a few mg/kg, are almost completely absorbed while larger doses are less well absorbed (Clayton and Clayton, 1981). For example, Copp and Greenberg (1941) found 30 percent of radioactive Co (^{60}Co) in urine in rats following a 10 mg orally administered dose, and more than 90 percent in urine following a 10 mg injected dose.

Wehner and Craig (1972) studied the distribution of CoO in hamsters. 87 percent of an inhaled 784 mg dose was distributed throughout the body and 11.3 percent of a 5 mg dose administered by gavage. The greatest amounts, 60 percent and 11 percent, respectively, remained in the GI tract. The carcass retained 23 percent and 0.34 percent; the lung, 3.3 percent and <0.06 percent; and the liver and kidneys retained small fractional percentages of the doses.

In man, an intravenously injected dose of 13 mg Co, as $CoCl_2$, resulted in a tenfold increase in urinary output and a seventeen fold increase in fecal excretion during the first week following injection. A total of 3 mg Co were recovered during this week, indicating slow elimination of Co (Kent and McCance, 1941).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The acute toxicity of Co appears to vary according to Co compound. Clayton and Clayton (1981) report on numerous animal studies that demonstrate toxic effects at concentrations ranging from 20 mg/kg for $CoCl_2$ (administered intravenously to rats) to 1700 mg/kg for CoO (administered orally). Toxic effects observed include diarrhea, loss of appetite, paralysis of the hind legs, and lowering of body temperature prior to death. Smaller doses produced albuminuria while larger doses resulted in anuria. Cutaneous vasodilatation occurs almost immediately and blood pressure may fall. Microscopically, organs become congested with small focal hemorrhages on serosal surfaces and large hemorrhages in the liver and adrenals. Degenerative effects may occur in bone, lung, kidney, heart, and pancreas tissue (Clayton and Clayton, 1981). Frederick and Bradley (1946), however, report no toxic effects for Co_2O_3 administered intraperitoneally to rats at 5000 mg Co/kg.

Chronically, Co toxicity appears to have a cumulative effect where elimination cannot keep pace with absorption. Schepers (1955) found repeated dosing of 5 mg Co intratracheally to be lethal to rats while a single 5 mg dose was not. Similarly, Frederick and Bradley (1946) found repeated 30 mg doses of Co were lethal to rats whereas 1500 mg Co was the lethal single dose. Underhill et al. (1931) revealed that dietary components may affect the toxicity of Co. Rats on a milk diet died at daily doses of 1.0 and 0.5 mg Co after 3.5 months whereas rats on a typical laboratory food diet tolerated 1 mg Co in drinking water for 14 weeks.

However, other studies indicate that a tolerance for Co may be developed if initial doses are sufficiently low to be well tolerated (Clayton and Clayton, 1981). For example, a relatively huge dose of 1g $CoCl_2 \cdot 6H_2O$ was required to be lethal following 13 days of daily subcutaneous injections of 10 mg $CoCl_2 \cdot 6H_2O$ (Seghini, 1940).

Chronic inhalation by animals of a Co-metal blend used in industry containing 6 percent Co resulted in focal fibrotic lesions, hyperplasia of the bronchial epithelium, and developing granulomas in areas of dust deposition after 3 years. The granulomas appear to resemble those reported in industrial workers (Stokinger et al., undated).

Schwartz et al., (1947) has documented a dermatitis of the allergic sensitivity type that appears to be related to the abrasive nature of Co dust. The dermatitis occurred in Co-cemented tungsten carbide workers, Co-alloy workers, and finnish pottery workers using Co-containing clay. Other toxic effects related to occupational exposure to Co dust include a rare "carboloy itch", pneumoconiosis, and sensitization.

Sullivan et al. (1969); McDermott et al., 1966; and Roy et al. (1968) report that Co was unexpectedly found to be the cause of severe lesions in cardiac muscle, hypothyroidism, and thyroid hyperplasia in excessive beer drinkers who drank beer containing $CoSo_4$ as a foam stabilizer. The Co apparently caused acute heart failure that was frequently fatal (50 deaths among 112 beer drinkers). Typically, patients experienced dyspnea, with abdominal pain and edema for 1 to 2 weeks. Extreme cardiomegaly with associated low blood pressure and pulse and peripheral cyanosis was common. Early deaths occurred within 72 hours of hospital admission. Out of 34 survivors in Omaha, Nebraska, 20 regained normal cardiac status and had good exercise tolerance, normal heart size, and minimal EKG changes. Six had recurrent or chronic heart failure. Four patients had neurological and mental deterioration, and 2 died suddenly after leaving the hospital.

Teratogenic Effects

ICF (1985) reports that Co caused craniofacial developmental abnormalities in the offspring of mice exposed by intraperitoneal injection during pregnancy.

No other data on the teratogenic effects of Co were found in the literature reviewed.

Mutagenic Effects

ICF (1985) reports that there is limited data indicating that $CoCl_2$ has mutagenic activity in a variety of test systems. No other data on the mutagenic effects of Co were found in the literature reviewed.

Carcinogenic Effects

Gilman (1962) and Heath (1960) report that Co and Co cause injection site sarcomas in rats. However, ICF (1985) reports that this type of response, by itself, is not generally considered adequate evidence of carcinogenicity. ICF (1985) states that "the absence of positive carcinogenic responses in other studies with experimental animals and the lack of epidemiologic evidence suggest that cobalt and its compounds are unlikely to pose a carcinogenic risk to humans." Other data on the carcinogenicity of Co were not found in the literature reviewed.

Ecotoxicity

ICF (1985) reports that data on the ecotoxicity of Co is limited. 50 ppm per day (3 mg/kg body weight) in the diet was acutely toxic to chickens. In sheep acute toxicity occurred at 6 mg/kg body weight. 3 mg/kg body weight, 1000 times the normal daily intake of Co, did not produce harmful effects in sheep, even after several weeks.

Standards, Criteria, and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

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COPPER

Use

Copper is widely used in the electrical industry because of its high conductivity. It is used in the casting industry and as an important alloying material. Common alloys which contain copper include brass, bronze, bell metal, and German silver (Sittig, 1991). Copper compounds are used in insecticides, fungicides, molluscicides, and paints (Sittig, 1991).

Chemical and Physical Properties

AW: 63.546	BP: 2567°C
SG: 8.92	MP: 1083°C
Sol.(Water): some copper salts.	VP: 1 mmhg at 1628°C
Sol.(Organics): insoluble	

Fate and Transport

Copper is present in the atmosphere primarily as dust and fumes from copper smelting plants. Although the atmospheric fate of copper has not been widely studied, it is thought that any chemical reactions would probably result in speciation (EPA, 1984). The principle atmospheric removal mechanisms are probably wet and dry deposition.

In surface waters, chemical speciation and sorption are the two dominant fate processes of released copper (EPA, 1984). In acidic waters, copper probably exists as Cu^{+2}. In alkaline waters, it probably exists as the carbonate complex. In organically rich waters, copper sorbs and forms complexes with organic material (EPA, 1985).

In the soils, the environmental fate of copper appears to depend on the Ph. In acidic soils, copper mobility would increase and leaching would occur more readily. In highly organic soils, copper would form complexes and would not leach (EPA, 1984).

Pharmacokinetics

In an extensive study using radioactive copper, Weber, et al. (1969) reported that the absorption in humans is diphasic. Primary absorption occurs within one hour of ingestion in the stomach and duodenum. The second phase occurs greater than 3.5 hours after ingestion in the small intestine. The average net absorption of ingested copper was 60 percent. Absorbed copper is stored primarily in the liver, heart, brain, kidneys, and muscles. Other studies indicate that mammals absorb copper in the upper gastrointestinal tract only.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Chattani, et al. (1965) evaluated data on the intentional ingestion of copper (as copper sulfate) by 53 suicide patients. Ingestion amounts ranged between 0.25 - 7.6g copper. Five patients died and those that survived were subject to nausea, vomiting, and epigastric pain.

Inhalation exposure to copper can cause the influenza-like symptoms of "metal fume fever". Symptoms include fever, chills, aching muscles, dryness of mouth and throat, and headaches (ICF, 1985). Chronic toxic effects of copper ingestion can include gastritis, hepatic neurosis, gastrointestinal bleeding, hypotension, and death (ICF, 1985). Copper salts act as skin irritants and can even cause conjunctivitis and corneal ulcerations when in direct contact with the eyes (ICF, 1985).

The chronic effects of copper poisoning can best be illustrated by the effects of Wilson's disease, a disease which inhibits the metabolism of copper in the body. Individuals with this disease accumulate approximately 20 times the normal amount of copper. These elevated concentrations effect the central nervous system, eyes, brain, and kidneys. It is characterized by tremors, drooling, seizures, jaundice, and eventually death (EPA, 1984).

Teratogenic and Other Developmental Effects

Copper compounds are known to be teratogenic to hamster and mice. Lecyk (1980) noted that low doses of copper stimulated embryonic development but higher doses (3000 - 4000 ppm) caused an increase in fetal mortality and embryonic malformations.

Mutagenic Effects

Copper appears to increase the number of mutagenic incidences in bacteria but does not seem to effect humans or animals in the same way (ICF, 1985).

Carcinogenic Effects

Copper and its compounds were not found to be carcinogenic to laboratory animals (EPA, 1985). Data regarding human carcinogenicity were not located in the available literature.

Ecotoxicity

The toxicity of copper to aquatic organisms appears to decrease with alkalinity, hardness, and total organic content (ICF, 1985). Acute toxicity values range between 7.2 mg/l for *Daphnia pulicaria* and 10,200 mg/l for the bluegill. Bioconcentration of copper appears to occur readily in freshwater and saltwater species (ICF, 1985).

Copper is known to be highly toxic to sheep. A dose of 200 mg/kg will generally kill a sheep. Ingestion of 1.5g/day for 30 days is also fatal to many breeds of sheep (ICF, 1985). It appears as though sheep have a reduced ability to excrete or metabolize copper (EPA, 1985).

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	4.0×10^{-2} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	4.0×10^{-2} mg/kg/day
Subchronic Inhalation RfD:	NA
MCLG:	1.3 mg/l
AWQC:	Water and Fish Consumption - 170 mg/l
	Fish Consumption - 3.433 mg/l

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FLUORIDE

Use

Fluoride is the general term applied to the class of compounds with the formula M_xF_y, where M is almost any inorganic group. Fluorides are used as an electrolyte in aluminum manufacture, a flux in smelting nickel, copper, gold, and silver, as a catalyst for organic reactions, a wood preservative, a fluoridation agent for drinking water, a bleaching agent for cane seats, in pesticides, rodenticides, and as a fermentation inhibitor. They are utilized in the manufacture of steel, iron, glass, ceramics, pottery, enamels, in the coagulation of latex, in coatings for welding rods, and in cleaning graphite, metals, windows and glassware. Fluorides may also be released during preparation of fertilizer from phosphate rock by addition of sulfuric acid (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula:	F_2 (soluble fluoride)	BP:	-188.13°C
MW:	38.0	MP:	-219.6°C
SG:	1.5127 at -188.13°C	VP:	760 mm Hg at -187.9°C
Sol. (water):	reactive with water		
Sol. (organics):	NA		

Fate and Transport

IARC (1982) reports that fluoride is widely distributed in the earth's crust and is believed to constitute 0.06 - 0.09 percent by weight of the upper lithosphere. Fluorspar, cryolite, and fluorapatite are the major fluorine-containing minerals (NRC, 1971). Quan (1978) reports that fluorspar is found on every continent.

Atmospherically, gaseous and solid fluorides are discharged naturally by active volcanoes and fumaroles (IARC, 1982). Man-made sources of atmospheric fluorides include hydrogen fluoride manufacture, petroleum refining, magnesium and aluminum founding and metal brazing, atomic energy installations using uranium hexfluoride, and test firing of rocket engines using fluorine or fluorine compounds as oxidizers (NRC, 1971).

Naturally occurring fluorides are present in most waters at levels usually below 1 mg/l (IARC, 1982). Elevated levels of fluoride derived from mineral deposits are present in certain spring waters; and natural concentrations in some parts of the world can reach 10 mg/l and sometimes exceed 20 mg/l (NRC, 1971). WHO (1970) reports that the highest fluoride levels found in water are as follows: in Kenya, 2800 mg/l; Tanganyika, 95 mg/l; South Africa, 53 mg/l; Czechoslovakia, 28 mg/l; and Portugal, 22.8 mg/l. IARC (1982) reports that U.S. surface waters contain an average of 0.25 mg/l fluoride, although rivers have been reported to contain 0 - 6.5 mg/l, and lakes, up to 1627 mg/l. NRC (1980) reports that various

ground waters contain 0 - 35.1 mg/l with U.S. ground water containing an average of 0.4 mg/l. Pacific and Atlantic sea waters contain 1.2 mg/l fluoride (IARC, 1982). Shawe (1976) reports that water that has been trapped in sediments since their deposition and related waters have average fluoride concentrations of 2.7 mg/l, and thermal waters associated with volcanoes and epithermal mineral deposits have an average of 5.4 mg/l. Shawe (1976) also reports that ocean sediments and shales contain 700 - 900 mg/kg fluoride and that marine phosphates have an average greater than 30,000 mg/kg.

Fluoride has been reported in U.S. soils at an average concentration of less than 300 mg/kg (Shawe, 1976). Fluoride concentrations in soils increase with depth. IARC (1982) reports that in a study of 30 different U.S. soils fluoride concentrations were as follows: 0 - 7.5 cm depth, 20 - 500 mg/kg; and 0 - 30 cm depth; 20 - 1620 mg/kg.

Pharmacokinetics

It is generally accepted that the fluoride ion is of principal importance in the toxicology of inorganic fluorides (Hodge, 1961). Cremer and Buttner (1970) report that fluoride ion is rapidly and extensively absorbed from the gut. Several studies cited by IARC (1982) indicate that absorption of the fluoride complexes sodium fluoride, sodium silicofluoride, sodium monofluorophosphate, and stannous fluoride is similar. Other studies cited suggested that the formation of complexes with cations such as Ca, Fe, Mg, and Al decreases the absorption of fluoride ion.

Chen et al. (1956) found that fluoride is transported in the blood in the free form rather than the protein-bound form. Fluoride is distributed rapidly throughout all soft tissues but is not accumulated (Wallace-Durbin, 1954; Armstrong and Singer, 1966).

Several studies reported in IARC (1982) indicate that fluoride is excreted mainly via the kidneys and is pH dependent. Thus, tubular reabsorption is increased and clearance decreased when the urine is acidic (Whitford et al., 1976; Whitford and Pashley, 1979). Perkinson et al. (1955) found that little fluoride was excreted in cow's milk.

Taylor et al. (1961) reports that fluoride accumulates in bones and teeth; in a 3-month experiment in rats 25-35 percent of ingested fluoride was retained in the calcified tissues, regardless of the concentration of fluoride in the drinking water. When fluoride was withdrawn from the drinking water of rats, the fluoride content of the bones decreased by 44 percent within the first eight weeks and more slowly thereafter (Miller and Phillips, 1953).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Fluoride-related compounds are used in the prevention of dental decay. The U.S. EPA (IRIS) reports that extensive human epidemiologic studies with large populations have been carried out over the last 40 years. These studies have revealed that adverse effects can result from exposure concentrations as low as 2 ppm. For example, Hodge (1950) studied children consuming fluoride in their drinking water for incidences of dental mottling. Fluoride levels of 0 - 14 ppm were investigated. An increase in the incidence of mottling occurred with increasing dose in the range of 2 - 10 ppm fluoride. No observable effects occurred in the 0.1 - 1.0 ppm range.

U.S. EPA (IRIS) reports that dental fluorosis, mottling of the teeth with white opaque patches covering 50 percent of a given tooth in its mild form and with black and brown patches accompanied by pitting in its severe form (U.S. EPA, 1985), results from excess exposure to fluoride during the age of calcification of the teeth (up to about 8 years of age for anterior teeth). There is debate, however, over the toxicity of dental fluorosis. The U.S. EPA (1985) has determined that objectionable dental fluorosis is a cosmetic effect and not a toxic and/or adverse health effect.

Crippling skeletal fluorosis can also occur from excessive exposure to fluorides. U.S. EPA (1985) estimates that it requires the consumption of 20 mg or more of fluoride/person/day over a 20-year period to produce the crippling disease. Supporting this, no adverse effects have been observed in the United States associated with the consumption of 2 liters of water/day containing 4 ppm fluoride.

Teratogenic and Other Developmental Effects

Stratmann (1979) reports that high intraperitoneal doses of stannous fluoride (≥10 mg/kg bw) caused varying degrees of embryolethality and teratogenicity in groups of five to seven mice. Impaired reproductive performance has been reported in mice, rats and cattle after intake of large amounts of fluoride (100 mg/l) in drinking water (Cass, 1961; Hodge and Smith, 1965; Messer et al., 1973). However, Messer et al. (1973) concluded that fluoride is essential for reproduction when infertility increased in female mice with their intake restricted to 0.1 - 0.3 mg/l fluoride in drinking water.

Ericsson and Malmnas (1962) report that fetal serum concentrations of 18F did not exceed 25 percent of that in the maternal blood following injection. IARC (1982) reports that fluoride that crosses the placenta is deposited in fetal bones and teeth. Brzezinski et al. (1960) found that the amount deposited was related to the age of the fetus and Gedalia et al. (1964) found that it was related to the fluoride intake of the mother. Smith and Smith (1935) report mottled dental enamel in the teeth of children whose mothers drank water containing 12-18 mg/l fluoride.

IARC (1982) reports that, although earlier reports indicated an association between naturally occurring fluorides in water supplies and cases of Down's Syndrome, more recent studies find no association between fluoride exposure and congenital malformations in humans.

Mutagenic Effects

Martin et al. (1979) report that sodium fluoride did not induce reverse mutations in *S. typhimurium* strains TA1535, TA1537, TA1538, TA98, or TA100 or *S. cerevisiae* strain D4 with or without a liver activation system. Similarly, ,sodium fluoride was found not to induce sex-linked recessive lethals in *D. melanogaster* (Mukherjee and Sobels, 1968) or chromosomal aberrations or an increase in the frequency of sister chromatid exchanges in bone marrow in mice (Kram et al., 1978). Sodium fluoride did not induce chromosomal aberrations in human lymphocytes (Voroshilin et al., 1973; Kralisz and Szymaniak, 1978).

Carcinogenic Effects

IARC (1982) reports that numerous ecological studies attempted to demonstrate a correlation between environmental fluoride exposure and cancer death rates in cities and countries in the U.S. and England. Most of these studies failed to adequately address the many parameters affecting such an assessment and, when accounted for, no statistically significant difference in cancer death rates appeared to be associated with exposure to environmental fluoride.

Kanisawa and Schroeder (1969) found no increase in the incidence of tumors in Swiss CD1 mice exposed to 10 mg/l sodium fluoride in doubly deionized drinking water for life (70 µg fluorine/day). Similarly, Tannenbaum and Silverstone (1949) report a decrease in the incidence of mammary gland carcinomas in surviving female DBA mice exposed to 900 mg/kg sodium fluoride in the diet. The frequency of carcinomas was 37/47 in the control group, and 20/40 in the treated animals.

Taylor (1954) exposed groups of C3H and DBA female mice to 0 (controls), 0.4, 1.0, 4.0, and 10.0 mg/l sodium fluoride in distilled drinking water for seven to twelve or ten to seventeen months. All animals except the 1.0 mg/l and 10.0 mg/l, ten to seventeen month groups were also exposed to a diet containing 20-38 mg/kg fluorine. The percentages of deaths due to mammary gland carcinomas were 54 percent in the controls and 59 percent in the dosed animals. Among the mice that received 10.0 mg/l sodium fluoride for ten to seventeen months, 63 percent died of mammary gland carcinomas as opposed to 50 percent in the control groups.

Ecotoxicity

IARC (1982) reports that plants can take up fluoride from soil, water, and air. Natural concentrations in the foliage of most plants range from 2-20 mg/kg (Brewer, 1966). There does not appear to be a relationship between the fluoride content of most plants and the content of the soil, however, a relationship has been established with increased concentrations of soluble fluoride in water (IARC, 1982). NRC (1971) reports that plants grown in acidic soil generally have higher fluoride concentrations.

NRC (1971) reports that plants may contain fluorides as a result of industrial air pollution. The average fluoride concentrations of foliage from cherry and peach trees growing near an aluminum factory increased from 13 mg/kg before the plant opened to 65 and 76 mg/kg, respectively, after it opened. After the plant opened, alfalfa and pine needles contained 65-166 and 24-104 mg/kg, respectively.

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	6×10^{-2} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	6×10^{-2} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	4 mg/l
AWQC:	NA

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CYANIDE

Use

Cyanide, usually defined as hydrogen cyanide and its salts, can be found in the environment in many different forms (ICF, 1985). Potassium cyanide and sodium cyanide, white solids with a faint almond odor, are the salt forms of cyanide most often used. They are used in various manufacturing processes, electroplating, metal treatment, and the extraction of ores (Sitting, 1981).

Chemical and Physical Properties

MW:	27 (HCN)	BP:	26.7°C (HCN)
SG:	0.699 at 22°C (HCN)	MP:	-14°C (HCN)
Sol. Water:	Soluble (HCN)	VP:	657.8 mm Hg at 21.9°C (HCN)
Sol (Organics):	alcohol, ether		

Fate and Transport

Callahan et al. (1979) illustrated that the fate of cyanides in aquatic media may depend on the chemical compound containing the cyanide. Hydrogen cyanide and alkali metal cyanides may be lost through the volatilization process. Sparingly soluble metal cyanides are expected to be removed from the aquatic media by sedimentation and microbial degradation. Photodecomposition may be the end result of water soluble complex metal cyanides, such as ferrocyanide but the absence of high temperature and extreme pH, destabilizing factors in water, leads to the expectation of long lifetimes and considerable transport.

In the atmosphere, hydrogen cyanide will be the dominant form of cyanide with metal cyanides potentially present as particulate matter in small amounts (U.S. EPA, 1984). Graedel (1978) illustrated that hydrogen cyanide reacts slowly with hydroxyradicals in the air in a reaction with a half-life of 11 years. It appears from this that cyanides are not lost from the troposphere in a significant manner. Physical transfers mechanisms, such as wet and dry deposition, may be the dominant fate of cyanides in the atmosphere (U.S. EPA, 1984).

The fate of cyanides in the soils may be pH dependent (U.S. EPA, 1984). Volatilization may occur in acidic soils. Microbial degradation could occur for small concentrations in subsurface soils. Given cyanides low soil sorption characteristics (Callahan et al., 1979) and its high water solubility, leaching may occur although cyanide has rarely been found in groundwater (U.S. EPA, 1984).

Pharmacokinetics

The gastrointestinal tract readily absorbs cyanide as illustrated in studies by Getter and Bain (1938) and Yamamoto et al. (1982). In both studies, laboratory animals died within minutes of exposure to cyanide salts by gavage in concentrations between 1.57 and 21 mg/kg bw. Because it is a weak acid, hydrogen cyanide occurs predominantly in the unionized from at physiological stomach pH. This facilitates absorption, thereby making the absorption of hydrogen cyanide faster than of cyanide salts (U.S. EPA, 1980).

Absorption of cyanide appears to occur readily by inhalation. Knowles and Bain (1968) illustrated a positive correlation between levels of hydrogen cyanide in the air and in human blood. Landahl and Herman (1950) reported a retention of 60 percent of the hydrogen cyanide humans inhaled by mouth.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Getter and Baube (1938) reported death in dogs who were given cyanide in the form of hydrogen cyanide (inhalation) and potassium cyanide (gavage) in concentrations as low as 1.6 mg/kg bw. Sandberg (1967) reported on a goldsmith apprentice exposed to cyanide when he polished gold five to ten times a day for four years. The polishing solution he used was prepared by adding 15g of potassium cyanide to water, bringing it to a boil, then adding hydrogen peroxide, thereby releasing hydrogen cyanide gas and splattering the skin. The toxic effects this man experienced included headache, listlessness, numbness and partial paralysis of his left arm and leg, and partial loss of vision in his left eye.

Howard et al. (1955), however, found no toxic effects when food fumigated with hydrogen cyanide was administered to rats over two years. Palmer and Olson (1979), although reporting significantly higher live weights in adult rats exposed to 20 mg/liter of potassium cyanide drinking water, reported no effect on liver weight when administered as 200 mg/kg diet.

Teratogenic and Other Developmental Effects

Tewe and Maner (1981) produced a decreased protein efficiency ratio by treating rats during gestation, lactation, and the postweaning growth phase with dietary cyanide at a concentration lower than the currently accepted NOAEL (IRIS), but found no effects on reproductive performance or the F1 generation.

Contrary to this, Amo's (1973) study of mice indicated that 0.05 mg/kg/day of cyanide in drinking water led to a decrease in the fertility rate and survival rate in the F1 generation and produced 100 percent mortality in the F2 generation.

Mutagenic Effects

DeFlora (1981) found that potassium cyanide was not mutagenic to five strains of *Salmonella typhimurium.* Karube et al. (1981) reported negative results from a rec-assay in ***Bacillus subtilis*** as well. Kushi et al. (1983) reported no mutagenicity to *Salmonella typhimurium* TA98, and only marginal mutagenicity to *Salmonella typhimurium* TA100 when treated with hydrogen cyanide.

Carcinogenic Effects

No data regarding the carcinogenicity of cyanide was found in the literature reviewed. Cyanide has not been classified in terms of carcinogenicity by EPA.

Ecotoxicity

Data on the acute toxicity of free cyanide (HCN and CN-) are available for many freshwater species. The acute sensitivities ranged from 44.73 - 2,490 µg/liter with all the acute sensitivities greater than 400 µg/liter, associated with invertebrates. Chronic values of 13.57, 7,849, and 16.39 µg/l were determined in a long-term survival and a partial and life-cycle test with fish, respectively. Chronic values of 18.33 and 34.06 µg/liter were determined for two invertebrate species. Cyanide concentrations ranging from 30 to 26,00 µg/liter affected freshwater plants (AWQC, 1986).

In the saltwater environment, the acute toxicity of free cyanide to species ranged from 4.893 to greater than 10,000 µg/liter with invertebrates being associated with both the highest and the lowest values. A chronic value of 36.12 µg/liter was determined in along-term survival in an early life-stage test with the sheephead minnow and a chronic value of 69.71 µg/liter resulted from a long-term survival in a mysid life-cycle test. While other species were affected at concentrations up to 3,000 µg/l, the red macroalga, *Champia parvula* showed toxicity at 11-25 µg/liter (AWQC, 1986).

An accidental spill of cyanide caused the death of 4,800 fish in Oak Ridge, Tennessee and cyanide leaching from a drum disposal site in Illinois led to livestock death and environmental damage (ICF, 1985).

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2.0 X 10^{-2} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	2.0 x 10^{-2} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.2 mg/l
AWQC:	Water and Fish Consumption - 200 µg/L
	Fish Consumption - 21.5 mg/L (recalculated)

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IRON

Use

Iron is primarily used as an alloy with carbon to produce steel. Occupational exposure generally occurs during mining, transport, and ore preparation (Sittig, 1991).

Iron is present naturally as the fourth most abundant element in the Earth's crust. It is commonly present as elemental iron, iron oxide, and iron pentacarbonyl (Sittig, 1991).

Chemical and Physical Properties

AW: 55.847	BP: 2,750°C
SG: 7.86	MP: 1,535°C

Sol.(water): Insoluble
Sol.(organics): Alcohol, ether

Fate and Transport

Iron can be present in the atmosphere as particulate matter or in compounds that are susceptible to chemical and photochemical reactions (EPA, 1984). The predominant sources of iron in the atmosphere are natural processes such as volcanic activity and wind erosion. The principle man-made sources of iron in the atmosphere are industrial emissions and the burning of fossil fuels (EPA, 1984). Iron is removed from the atmosphere by wet and dry deposition and, to a lesser extent, by photochemical reactions (EPA, 1984).

In aquatic systems, iron is susceptible to precipitation, speciation, oxidation-reduction, and photochemical reactions. The particular reaction depends on the Ph of the body of water and the concentration of microorganisms. In more acidic waters, iron remains in solution and, as a result, is more mobile. Iron is expected to be present in the form of suspended particulates and, to a lesser extent, ions and organic complexes (EPA, 1984). The residence time of iron in aquatic media is expected to be greater than 140 years.

In soils, iron is present primarily as Fe (III). In most soils, iron is not mobile because of its high sorptive qualities. Small amounts may be transported in the form of colloidal ferric oxyhydroxides. The mobility of iron increases in more acidic soils (EPA, 1985).

Pharmacokinetics

Iron is absorbed by humans as heme iron from meats and as non-heme iron from grain and vegetables. Bjorn-Rasmussen, et al. (1974) reported that heme iron is absorbed at a rate of 37 percent whereas non-heme iron is absorbed at a rate of 5 percent. Iron is absorbed in the

mucosal cells of the proximal duodenum and in the small intestine. Absorption is regulated by the amount of available iron already present in the body.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

In children, as little as 0.3 g iron has been associated with severely toxic effects (Greenblatt, et al., 1976). Certain iron compounds, such as ferrous succinate and ferrous sulfate are severely toxic to humans when acutely exposed. Doses as small as 2 grams have caused fatalities. Majumder et al. (1975) reported that ferrous sulfate is much more toxic to rats and guinea pigs whose diets lack sufficient amounts of Vitamin C. Vitamin C deficient guinea pigs treated with 5 mg/day suffered severely toxic effects and mortality.

Chronic exposure to iron can result in irritation to the respiratory system and skin (ICF, 1985). Chronic ingestion is known to cause hemosiderosis and hemochromatosis. Chronic inhalation studies of steel workers have not revealed an association between iron fumes and chronic bronchitis and emphysema (EPA, 1985)

Teratogenic and Other Developmental Effects

Increased iron intake by pregnant women has resulted in only beneficial effects (EPA, 1984). No information regarding the teratogenic effects of iron were located in the available literature.

Mutagenic Effects

Demerec, et al. (1951) reported that high concentrations of ferrous or ferric chloride caused point mutations in *E.coli*. Castro, et al. (1979) reported that ferrous sulfate inhibited the transformation of Syrian hamster embryo cells.

Carcinogenic Effects

Several studies have suggested that iron oxide dust may promote the induction of cancer by known carcinogens. Iron oxide may be co-carcinogenic because of its ability to cause hyperplasia. No conclusive evidence was located in the available literature that suggests that iron compounds are carcinogenic. Some studies have indicated that iron-carbohydrate complexes such as ferric dextran may cause local tumors but, the evidence is not definitive (EPA, 1984.

Ecotoxicity

It is unlikely that iron causes any toxic effects to wildlife, however, the available data are inadequate to draw any conclusions (ICF, 1985)

Standards, Criteria and Guidelines

EPA Class D Carcinogen (elemental iron)
EPA Class C Carcinogen (iron compounds only)

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	5.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.3 mg/l (secondary)
AWQC:	Water and Fish Consumption - 0.3 mg/l
	Fish Consumption - NA

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RECYCLED PAPER

LEAD

Use

Lead is a heavy metal that exists in three oxidation states (0, +2, and +4). In addition to their natural occurrence, lead and its compounds may enter and contaminate the global environment at any stage during mining, smelting, processing, and use. The annual increase in lead consumption in the United States during the 10-year period from 1962-1971 averaged 2.9 percent, largely due to increased demands for electro-chemical batteries and gasoline additives (EPA, 1984). Nonindustrial sources that may contribute to the possibility of ingestion of lead by man include the indoor use of lead-bearing paints and plasters, improperly glazed earthenware, lead fumes or ashes produced in burning lead battery casings, and exhaust from internal combustion engines (EPA, 1984).

Chemical and Physical Properties

AW: 207.19	BP: 1,704°C
SG: 11.35 at 20°C	MP: 327.5°C

Sol. (Water): Insoluble
Sol. (Inorganics): $HN0_3$, hot H_2S0_4

Fate and Transport

Lead is artificially introduced into the environment primarily through the combustion of lead-containing fossil fuels and from lead mining operations (EPA, 1984). Lead fumes undergo decomposition when exposed to light. As a result, fumes that are present around gas stations and in heavily travelled areas are not a significant avenue of contamination (EPA, 1989). Particulate lead, carried in the atmosphere, is removed by either wet or dry deposition. Rainfall is not as significant in the deposition of lead particles as would be expected (EPA, 1984).

The transport of lead in ground water and surface water is highly variable based on its oxidation state. In polluted waters, organic complexation of lead is the primary factor in the determination of toxicity. Lead is adsorbed strongly to organic materials in soils but is not easily absorbed by living plants (EPA, 1984).

Pharmacokinetics

It has been estimated that, in man, approximately 8 percent of the lead ingested daily is absorbed. Absorption of lead consumed by humans after a 6-hour fast was increased up to 8-fold when compared with lead consumed with food. Similar effects were observed in dietary studies of mice given a dose of 3 µg Pb/kg-bw, but not at much higher doses (2,000 µg Pb/kg bw) (EPA, 1984). Numerous dietary factors influence the absorption of lead from the gastrointestinal tract. Lead absorption has been demonstrated to be enhanced by low dietary calcium or iron, high dietary fat, or low or high protein. Four baboons exposed to lead

aerosols (Pb_3O_4) of varying particle size for 4 weeks showed that absorption was faster for 1.6 μm particles than for more fine particles (0.8 μm) (EPA, 1984).

In humans, it appears as though hemoglobin and hemo-proteins are affected by lead more so than any other organ or system (EPA, 1984). At levels of 0.4 μg Pb/ml blood in adults, the amount of hemoglobin and hemo-proteins produced is decreased.

Human Toxicity

Noncarcinogenic

Systemic Effects

The majority of the studies concerned with the effects of lead exposure in humans are based on blood lead levels, not ambient lead levels (EPA, 1984). Decreased hemoglobin production is seen at low blood lead levels of 0.5 μg/ml blood in children.

Chronic exposure of rats to lead acetate produced slight effects on conduction tissue excitability, systolic blood pressure, and cardiac ATP concentrations. This study was performed over a period of 20 weeks on rats given 5 mg Pb/L water in their drinking water (EPA, 1984).

Teratogenic and Other Developmental Effects

Postnatal developmental delays have been reported in pups from rats that received 50-250 mg Pb/liter in drinking water throughout gestation (EPA, 1984). Effects on reproductive parameters were noted in rats and mice in a three-generation study with 25 ppm lead (from an unspecified soluble lead salt) in drinking water. In this study, environmental concentrations of other metals were minimized (EPA, 1984). In high doses, lead compounds have been used to induce abortions. Oliver (1911) noted that the miscarriage rate among British women occupationally exposed to lead was elevated. Several other studies have reported that increases in spontaneous abortions, premature delivery, and early membrane rupture have been associated with lead exposure.

In one study, groups of 60-90, 21-day-old female CD rats were administered a semipurified, nutritionally adequate, virtually lead-free diet. Lead acetate was administered in deionized drinking water at concentrations of 0, 0.5, 5, 50, or 250 mg Pb/liter of water. The treated females were mated with untreated males after 6-7 weeks and were continued on treatment throughout gestation and lactation. There were no treatment-related differences in food or water consumption between the various treatment groups; however, body weights of offspring were depressed at the two highest doses. Sexual maturation, as measured by the time of vaginal opening, was delayed in a dose-dependent

manner, with effects observed at concentrations 25 mg Pb/liter or greater (EPA, 1984).

Mutagenic Effects

DiPaolo, et al. (1978) noted that lead acetate induces cell transformation in Syrian hamster embryo cells and increases the incidence of simian adenovirus induction.

Grandjean, et al. (1983) discovered a relationship between sister-chromatid-exchange and lead exposure in workers.

Carcinogenic Effects

An increase in the incidence of renal tumors was observed in rats exposed to 1000 ppm and 2000 ppm in the diet for 2 years (Azar et al., 1973).

Similar results were observed when Kasprzak, et al. (1985) orally administered a dose of 8500 ppm Pb, as lead subacetate, per day to Sprague-Dawley rats for 79 weeks. Forty-four percent of the treated rats developed renal tumors; four of twenty-nine rats developed adenocarcinomas and the remaining nine developed adenomas. In a similar study, Koller, et al. (1986) administered 2600 ppm Pb, as lead acetate, in drinking water to Sprague-Dawley rats for 76 weeks. Eighty-one percent developed renal tubular carcinoma.

Dietary lead acetate administered in doses of 3-4 mg/day, 500-2000 mg/kg diet or 1 percent in the diet have produced renal tumors in Wistar rats (EPA, 1984). In a separate study, it was shown that a lead acetate produced renal carcinomas or adenomas in Swiss mice and several other rodents.

From available studies, it appears as though inorganic leads are the cause of any carcinogenic effects seen in humans or animals.

Ecotoxicity

Chronic toxicity studies of lead in *Daphnia magna* indicate that water hardness effects lead toxicity. The daphnids were nearly 11 times more sensitive to lead in soft water than in hard water. The chronic toxicity value of lead nitrate in water with a hardness of 52 mg/liter as $CaCO_3$ is 12.26 µg/liter. An early life stage test was conducted on the highly sensitive rainbow trout (*Salmo gairdneri*). For trout raised in water with a hardness of 28 mg/l $CaCO_3$, a chronic toxicity value of 18.80 mg/l was generated. The only chronic study located concerning saltwater species was conducted on mysid shrimp (*Mysidopsis bahia).* The results indicate that this small crustacean is highly sensitive to lead nitrate, yielding a chronic toxicity value of 25.08 µg/liter. The aforementioned chronic values are decisive in showing that lead nitrate is highly toxic to freshwater and saltwater aquatic life (EPA, 1984).

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	No slope factor derived by Carcinogen Assessment Group
Inhalation Slope Factor:	NA
Chronic Oral RfD:	NA/no threshold
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
MCL:	0.015 mg/l
AWQC:	Water and Fish Consumption - 50 µg/l
	Fish Consumption - NA

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MANGANESE

Use

Manganese is used primarily as an alloy in steel and iron manufacturing. Manganese compounds are used in the manufacture of dry cell batteries, paints, varnishes, dyes, inks, fireworks, fertilizers, and disinfectants. Organic manganese compounds have been tested as potential supplemental anti-knock agents in gasolines (Sittig, 1991).

Chemical and Physical Properties

AW: 54.938
SG: 7.20
Sol. (water): decomposes
Sol. (organics): insoluble

BP: 1962°C
MP: 1244°C
VP: 1 mmHg at 1292°C

Fate and Transport

Manganese occurs most often in the +2, +4 and +7 valence states. Elemental manganese, as well as manganese compounds, are present in the atmosphere as a result of natural processes.

In the atmosphere, manganese can be present in particulate form and, as such, it is susceptible to photo-chemical and thermal reactions (EPA, 1984). Manganese reacts with SO_2 and NO_2 and is removed from the atmosphere most effectively through wet and dry deposition (EPA, 1984).

In aquatic media, the fate of manganese is effected primarily by the amount of dissolved oxygen present and by the acidity of the water. In aerobic waters, manganese forms MnO_2 and Mn_3O_4 which either remain suspended or deposit to the sediments. The residence time of insoluble manganese compounds is known to be as much as 300 years (EPA, 1984).

In soils, the solubility of manganese is increased with low Ph and with high concentrations of chlorides, nitrates, or sulfates. Under these conditions, manganese is transported readily and is absorbed rapidly by plants (ICF, 1985).

Pharmacokinetics

Absorption of manganese occurs primarily in the gastrointestinal tract and is controlled homeostatically by the amount of manganese already present in the body. Under normal conditions, approximately 3 percent of ingested manganese is absorbed. Anemia victims appear to absorb more than twice that amount (EPA, 1984). Manganese absorption appears to be competitive with iron absorption.

Inhalation studies indicate that small manganese particles are absorbed in the lungs by the alveoli and are excreted within 4 days. Approximately 40-70 percent of absorbed manganese is excreted in the feces (EPA, 1984).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Orally administered manganese appears to cause minimal toxic effects in humans (ICF, 1985). The World Health Organization (WHO, 1973) reviewed several investigations which studied the effects of average daily consumption of concentrations of manganese ranging from 2 to 8.8 mg/Mn/day in adult diets. Levels from 8 to 9 mg/day were determined to be "perfectly safe". Reference doses (RfDs) were based on these studies, with a NOAEL of 0.14 mg/kg/day.

In one chronic ingestion study, Kawamura, et al. (1941) reported that 14.3 mg Mn/L drinking water causes lethargy, spasms, tremors, and mental disturbances. Both chronic inhalation and ingestion of manganese appear to effect the central nervous system most predominantly.

Teratogenic and Other Developmental Effects

Chronic manganese poisoning has been shown to cause depressed reproductive function in male and female laboratory animals. Penalver (1955) reported that oral exposure to manganese causes impotency in humans. Mandzgaladze (1967) reported that manganese exposure causes an increase in still births and spontaneous abortions in humans.

Mutagenic Effects

Manganese has been reported to be mutagenic to *Salmonella* strains and *E. coli*. Casto, et al. (1979) reported that manganese was moderately effective in enhancing viral transformation in Syrian hamster embryo cells.

Carcinogenic Effects

Manganese compounds, such as manganese chloride, manganese acetylacetonate, and manganese dioxide, caused an increased incidence of injection site tumors in rats but, EPA has determined that these results cannot be extrapolated to include elemental manganese (IRIS). No increase in lymphosarcomas and fibrosarcomas were noted by Furst (1978) in rats orally exposed to manganese powder.

Ecotoxicity

Data regarding the toxicity of manganese to aquatic organisms were not located in the available literature.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	1.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	1.14×10^{-4} mg/kg/day
Subchronic Oral RfD:	1.0×10^{-1} mg/kg/day
MCL:	0.05 mg/l (secondary)
AWQC:	Water and Fish Consumption - 50 µg/l
	Fish Consumption - 100 µg/l

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MERCURY

Use

Mercury is used commonly in its elemental, organic, and inorganic forms.

Organic mercury, seen in both alkyl and aryl forms, is used to treat seeds for airborne diseases and is used as an additive in wood preservatives and disinfectants (Sittig, 1991). Aryl mercury compounds are also used to denature ethyl alcohol, germicides, and bactericides.

Inorganic mercury, commonly found as mercuric or mercurous salts, is utilized in gold, silver, bronze, and tin plating processes. It is also used in tanning, dyeing, felt-making, taxidermy textile manufacturing, and photography (Sittig, 1991).

In its elemental form, mercury is used as a liquid cathode in the production of chlorine and caustics. It can also be found in lamps, batteries, thermometers, and switches (Sittig, 1991).

Chemical and Physical Properties

AW: 200.59
SG: 13.594 at 20°C
Sol. (water): 81.3 µg/l at 30°C

BP: 356.8°C
MP: -38.87°C
VP: 0.0012 mmHg at 20°C

Fate and Transport

Mercury is expected to be present in the atmosphere primarily as Hg(O) from electrical industries and from the burning of fossil fuels. Elemental mercury, several inorganic species and dimethyl mercury can volatilize to the atmosphere when released to surface waters and soils (ICF, 1985). Once released to the atmosphere, mercury is removed primarily by precipitation (EPA, 1984), but certain compounds can also be photolyzed (ICF, 1985).

In aquatic environments, mercury readily adsorbs to organic matter. In waters with high organic content, sedimentation and subsequent bioaccumulation are likely to occur (ICF, 1985).

Mercury binds strongly to soils with high organic matter and, as a result, remains relatively immobile (EPA, 1984). Mercury does not transport well in ground water except when combined with leachate from municipal landfills (EPA, 1984).

Pharmacokinetics

Elemental and inorganic mercury appear to be poorly absorbed through the gastrointestinal tract lining in humans. Less than 15 percent absorption was observed by Suzuki and Tonaka (1971) in a case study performed on individuals who accidentally ingested several grams of metallic mercury. Conversely, organic mercury, in the form of methyl mercury, is almost completely absorbed (EPA, 1984).

When inhaled by humans, approximately 80 percent of a dose of inorganic mercury is absorbed by either the alveoli or the bronchioles. Morrow, et al. (1964) reported that 40 percent of a dose of mercury, administered to dogs in the form of an aerosol, was absorbed.

When absorbed by humans, mercury is known to accumulate in the kidneys. It is excreted in both the urine and the feces (EPA, 1985).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Chronic exposure to organic mercury results mainly in adverse effects to the central nervous system in humans (EPA, 1984). The cortex, in particular the cortical neuron of the occipital lobe, is the region of the brain that is most heavily affected. Clinical symptoms of mercury poisoning include paraesthesia, sensory depression in the extremities, ataxia, and vision and hearing impairment (EPA, 1984). These data were obtained primarily through studies performed on two populations, one in Japan and one in Iraq, that accidently ingested large quantities of mercury. The procedures by which mercury concentrations were measured have been determined to be inadequate for both studies, so the doses received are inaccurate.

Fitzugh, et al. (1950) reported a 10 percent reduction in body weight in male rats after being fed diets of 160 ppm mercuric acetate for 2 years. Female rats appeared to be unaffected, and rats of both sexes appeared to be unaffected by chronic doses of less than 160 ppm. Slight damage was done, to varying degrees, to the proximal convoluted tubules in the kidneys.

Smith, et al. (1970) performed a study of 500 workers exposed to atmospheric mercury in chloroalkali plants. At low exposure concentrations (0.06-0.1 mg Hg/m^3), loss of appetite and weight loss were noted. At exposure concentrations greater than 0.1 mg Hg/m^3, tremors were observed.

In a separate study, the effects of chronic mercury inhalation in workers in a felt hat factory were observed. Mercury vapor, mercuric nitrate, and particulate elemental mercury were all found to be present in the air. Of workers exposed to concentrations greater than 0.24 mg Hg/m^3 for 20 years, 54 percent displayed observable tremors, the classic symptom of mercury poisoning (EPA, 1985).

Teratogenic and Other Developmental Effects

Prenatal exposure to methyl mercury is known to cause brain damage in humans. Numerous case studies of children, accidentally exposed to methyl/mercury through ingestion of contaminated fish, have shown a significant increase in psychomotor retardation (EPA, 1984).

Baranski and Szymczyk (1973) noted that pups of rats exposed to high concentrations of inorganic mercury vapors just before or during gestation, showed a significant increase in fatalities within 6 days after birth.

Mutagenic Effects

5 mg/liter of methyl mercury hydroxide administered to *Drosophila melanogoster* in the diet induced chromosomal aberrations. Methyl and phenyl mercury produced small increases in the rate of point mutations (Ramel, 1972).

Carcinogenic Effects

No form of mercury, either elemental, organic or inorganic, has been shown to cause cancer in humans or laboratory animals or to induce changes in cultured cells (EPA, 1985).

Ecotoxicity

Methylmercury appears to be more toxic to aquatic organisms then mercuric salts, although more testing has been done on the latter (ICF, 1985). LC_{50} values for mercuric salts range between 0.02 µg/l to 2,000 µg/l for freshwater aquatic species. In rainbow trout, methylmercuric salts were found to be approximately 10 times more toxic than mercuric salts. LC_{50} values for saltwater species range from 3.5 to 1,680 µg inorganic mercury/l. Molluscs and crustaceans, both filter feeders, appear to be more sensitive to the toxic effects of inorganic mercury than do planktonic species.

Standards, Criteria and Guidelines

EPA Class D carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	3.0×10^{-4} mg/kg/day
Chronic Inhalation RfD:	8.57×10^{-5} mg/kg/day
Subchronic Oral RfD:	3.0×10^{-4} mg/kg/day
MCL:	0.002 mg/l
AWQC:	Water and Fish Consumption - 0.14 µg/l
	Fish Consumption - 0.15 µg/l

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NICKEL

Use

Elemental nickel is used in electroplating, casting, batteries, and coinage. It is used in the manufacture of acid-resisting alloys, magnetic tapes, surgical, and dental instruments and colored ceramics and glass (Sittig, 1991). Elemental nickels most common use is as an alloy in the production of stainless steel because of its excellent corrosion resistant properties.

Nickel carbonyl, a common nickel compound, is formed in the extraction of pure nickel from ore.

Chemical and Physical Properties

AW: 58.71
SG: 8.90 at 25°C
Sol. (water): insoluble
Sol. (organics): variable

BP: 2,732°C
MP: 1,453°C
VP: 1mm Hg at 1,810°C

Fate and Transport

Nickel is most often released to the atmosphere as dusts and fumes from smelting and processing facilities, coal burning, and diesel oil combustion (EPA, 1985). The principal removal pathways of nickel from the atmosphere are wet and dry deposition. Chemical interactions of nickel in the atmosphere generally result in elemental nickels conversion to nickel oxide (EPA, 1984).

In aquatic environments, nickel generally exists in solution as hydroxide, carbonate, sulfate, and organic complexes. The environmental fate of nickel in aquatic media appears to be dependent on the extent of pollution. In highly polluted waters, nickel is more apt to remain dissolved (EPA, 1984).

In soils, the amount of organic matter, iron oxides, and manganese oxides, may determine the fate of nickel. In soils with high iron and manganese oxide content, nickel would sorb and remain stable but, in soils with high organic content, nickel would complex and become more mobile (EPA, 1984).

Pharmacokinetics

Nickel is absorbed by humans and animals through ingestion, inhalation and, to a lesser extent, percutaneous exposure. Horak and Sunderman (1973) reported that, of the 160 to 500 µg nickel ingested daily by the average man, 1 to 10 percent is absorbed.

Absorbed nickel appears to be distributed throughout the pancreas, testes, and bones in calves and throughout the kidneys, liver, heart, and testes in rats (EPA, 1985). Nickel is transported through the body's sera primarily by serum albumin in man, rabbits, rats, and bovine (EPA, 1985). In man, it is excreted in the urine and is deposited in hair follicles. Ingested metal that is not absorbed is excreted in the feces (EPA, 1985).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Ambrose et al. (1976) exposed rats to nickel sulfate hexahydrate in concentrations of 0, 100, 1000, or 2500 ppm as Nickel in the diet for 2 years. High-dose rats exhibited decreased body weights, increased heart-to-body weight ratios and decreased liver-to-body weight ratios. Inhalation studies indicate that chronic exposure to high concentrations of nickel fumes can cause severe toxic effects, including pathological respiratory changes and death in humans. Less severe effects, including dermatitis, sinusitis, and nasal mucosal injury have been reported by workers occupationally exposed to various nickel compounds (ICF, 1985).

Teratogenic and Other Developmental Effects

Inhalation of nickel carbonyl vapors by dams caused a highly significant increase in eye malformation in newborns. The teratogenic effects of nickel carbonyl were found to be dose related (EPA, 1984).

Mutagenic Effects

Nickel carbonyl has been found to bind to liver and kidney DNA (Hui and Sunderman, 1980). Numerous studies cited in IRIS reveal that nickel subsulfide induces morphologic transformation in Syrian hamster embryos, and baby hamster kidney (BHK-21) cell cultures, sister chromatid exchange in human lymphocytes, and DNA strand breaks. As cited in IRIS, Sunderman (1984) observed nickel subsulfide to concentrate in the cell nucleus in *in vitro* assays.

Carcinogenic Effects

There have been numerous case studies performed on nickel smelting workers indicating that exposure to nickel fumes increases the chance of lung and nasal cavity tumors. Pedersen, et al. (1973) and Doll, et al. (1977) reported that nickel refinery workers exposed to 20 to 26 mg Ni/m^3 on a chronic basis developed a significantly higher number of tumors than would be expected in a normal population. In Pedersen's studies, the risk of lung cancer increased 3.75 fold and the risk of nasal

cancer increased 27 fold. More recent refinery methods and more stringent occupational exposure regulations have greatly reduced the carcinogenic potential to workers.

There is not sufficient evidence concerning oral exposure to nickel to draw any conclusions.

Nickel subsulfide, nickel carbonyl, nickel oxides, and nickel sulfate are all thought to indure tumors in laboratory animals (ICF, 1985).

Ecotoxicity

Nickel tends to be more toxic to aquatic life when there are lower concentrations of iron and manganese in the water (decreased hardness). Nickel salt concentrations between 510 and 46,200 µg/L were determined to be acutely toxic to freshwater species (ICF, 1985). Saltwater algae have shown stunted growth in nickel concentrations as low as 1,000 µg/L.

Standards, Criteria and Guidelines

EPA Class A Carcinogen (refinery dust, subsulfide)
EPA Class B2 Carcinogen (carbonyl)

Oral Slope Factor:	NA
Inhalation Slope Factor:	8.4×10^{-1} (mg/kg/day)$^{-1}$ (refinery dust, subsulfide)
Chronic Oral RfD:	2.0×10^{-2} mg/kg/day (soluble salts)
Chronic Inhalation RfD:	Currently under review by EPA (soluble salts)
Subchronic Oral RfD:	2.0×10^{-2} mg/kg/day (soluble salts)
MCL:	0.1 mg/l
AWQC:	Water and Fish Consumption - 1.3×10^{-7} µg/l
	Fish Consumption - 100 µg/l

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SILVER

Use

Silver is alloyed with copper, aluminum, cadmium, lead, and antimony. The various alloys are used in the manufacture of silverware, jewelry, coins, scientific instruments, and batteries. Silver compounds are frequently used as emulsions in photographic films, plates, and paper; and as bactericides for sterilizing water, fruit juices, and vinegar (Sittig, 1991).

Chemical and Physical Properties

AW: 107.868	BP: 2212°C
SG: 10.5 at 20°C	MP: 961.93°C

Sol. (water): Insoluble
Sol. (organics): Alkali cyanide solutions

Fate and Transport

Atmospheric transport, volatilization, and biotransformation do not appear to be important fate and transport processes for silver and its compounds (ICF, 1985). In general, silver is found in aquatic media and in soils.

In aquatic media, metallic silver generally has very low solubility. Silver cations combine readily with halogen ions, most commonly with chloride to produce silver chloride. Sorption is probably the dominant fate of silver compounds. Silver sorbs readily to clay, ferric hydroxide, and manganese dioxide (ICF, 1985). Sediment concentrations of silver are known to be 1,000 times greater than overlying water concentrations.

Pharmacokinetics

Silver can be absorbed through inhalation or ingestion. Accumulation of silver can occur in the skin, eyes, hair, and internal organs. It is known to cause damage to the kidneys, liver, and central nervous system (ICF, 1985). No other information concerning the pharmacokinetics of silver was located in the available literature.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The inhalation of silver salts has been shown to cause argyria, a visible accumulation of silver characterized by the discoloration of pigments (ACGIH, 1984). It was reported that generalized argyria may result from chronic inhalation exposure whereas, localized argyria of the skin may be caused by cutaneous exposure (ACGIH, 1984). In a separate study, researchers noted after careful observation of workers employed for several years in the photographic industry, that silver exposure did not cause any adverse systemic effects. They determined that, as long as atmospheric concentrations were lower than 0.01 mg/m^3, no effects will occur (ACGIH, 1984).

Intravenous injection of silver nitrate is reported to cause pulmonary edema and congestion in laboratory animals. Various other, unspecified silver compounds are known to cause liver, kidney, and central nervous system damage in laboratory animals (ICF, 1985). Rats exposed to 20 mg silver/L in drinking water for 5 months exhibited signs of growth depression and pathomorphological changes in the liver, kidneys, stomach, and small intestine (ICF, 1985).

Teratogenic and Other Developmental Effects/Mutagenic Effects

Several studies have not proven silver and its compounds to be mutagenic or teratogenic.

Carcinogenic Effects

One study concluded that the implantation of silver foil into the skin of rodents causes fibrosarcomas in 30 percent of test subjects (ACGIH, 1985). More recent studies have indicated, however, that numerous insoluble solids such as ivory and plastic cause local fibrosarcomas when implanted under the skin (IRIS). Schmahl and Steinhoff (1960) reported that colloidal silver injections resulted in tumors in 8 of 26 rats. Furst and Schlander (1977) reported contradictory evidence. Silver, in a trioctanoin suspension, was injected into rats monthly. Other groups were injected with suspension only or with hold or cadmium solutions. Injection site sarcomas were noted in all the groups (control, gold, cadmium) except those treated with silver.

However, there is not sufficient evidence to suggest that silver is carcinogenic to humans.

Ecotoxicity

Acute toxicity values for silver in freshwater invertebrates range from 0.25 µg/L to 4,500 µg/L. Values for freshwater fish range from 3.9 µg/L to 280 µg/L. Various species of saltwater algae exhibit stunted growth when exposed to 130 µg silver/L (ICF, 1985).

Silver has been reported to cause and to aggravate the effects of vitamin and mineral deficiency in domestic animals (ICF, 1985).

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	5.0×10^{-3} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	3.0×10^{-3} mg/kg/day
MCL:	0.10 mg/l (secondary)
AWQC:	Water and Fish Consumption - 50 µg/l
	Fish Consumption - 50 µg/l

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THALLIUM

Use

Thallium and its compounds are used as catalysts in certain organic reactions, in phosphor activators, in bromoiodide crystals for lenses, plates, and prisms in infrared optical instruments, in photoelectric cells, in mineralogical analysis; in alloys with mercury in low-temperature thermometers, switches and closures; in high-density liquids, dyes and pigments; and in the manufacture of optical lenses, fireworks, and imitation precious jewelry. It forms a stainless alloy with silver and a corrosion resistant alloy with lead. Its medicinal use for epilation is almost discontinued (Sittig, 1991). Prior to 1972, thallium and its compounds were used as rodenticides, fungicides, and insecticides (Sittig, 1991; Stokinger, 1981).

Physical and Chemical Properties

AW:	204.37	MP:	303.5°C
SG:	11.85 g/mc^3	BP:	1457°C
VP:	10 mm Hg at 1000°C		
Sol. (water):	insoluble		

Fate and Transport

Cement factories, coal burning power plants, and metal smelters are the principal sources of thallium in the environment (Sharma et al., 1986; Brockhaus et al., 1980, 1981). Atmospherically, thallium may be present in its elemental form, as oxides of Tl, as Tl_2S, or Tl_2So_4. Tl_2S is likely to be speciated to Tl_2SO_4 and Tl_2O will be rapidly hydrolyzed to TlOH by the moisture in the atmosphere (U.S. EPA, 1988).

Both TlOH and Tl_2SO_4 are most likely removed from the atmosphere by wet deposition given their water solubilities. Tl_2O_3, however, may persist in the atmosphere longer because it is insoluble in water and, therefore, will be removed by dry deposition (U.S. EPA, 1988).

In aquatic systems, insoluble forms of thallium will accumulate in the sediment (Mathis and Kevern, 1975). Kempton et al. (1987a,b) reports that thallium may be removed from the water by sorption onto suspended solids in water. Most of the soluble thallium that enters aquatic systems will remain in the soluble state due to its formation of soluble complexes with inorganic and organic ligands (Stephenson and Lester, 1987a,b). These complexes are even more stable at higher pHs (O'Shea and Mancy, 1978). Wallwork-Barber et al. 91985) report that thallium in water may be transported to fish and vegetation. The bioconcentration factor of thallium in whole aquatic organisms ranges from 12-34 (Zitko and Carson, 1975; Barrows et al., 1980).

In soils, leaching of thallium, particularly from sandy soils, appears to be likely given its transport in water (U.S. EPA, 1988). Cataldo and Wildung (1983) report that up to 10 percent of the thallium absorbed in plant roots from soil may be transported from the root to the shoot of the plant.

Pharmacokinetics

U.S. EPA (1988) reports that numerous studies reveal that absorption of soluble thallium by any route of exposure is rapid and virtually complete, although dermal absorption is not likely to be significant in environmental exposure. Several studies indicate that distribution of thallium from the blood is rapid and widespread; with highest levels detected in the kidney, heart, and liver; and lowest levels detected in the nervous system and body fat. Lie et al. (1960) reports that the relative concentrations in different tissues appear to be independent of the route of administration and the time after administration. In addition, Sabbioni et al. (1980) and Gregus and Klaasen (1986) found no correlation between tissue concentrations and the valance of thallium administered or the dosage, respectively. The U.S. EPA (1988) reports that several studies indicate that thallium translocates to the placenta and fetus, but levels in the fetus are substantially lower than those in maternal tissues. Sabbioni et al. (1980) hypothesizes that thallium *in vivo* is transformed to one oxidation state. Barclay et al. (1953) and Richelmi et al. (1980) report that, in humans, excretion of thallium occurs predominantly in the urine. A range of estimated excretion half-lives have been reported with Talas et al. (1983) reporting 2.15 days for tracer doses in ambulatory heart patients and Barclay et al. (1953) and U.S. EPA (1980) reporting 21.7 days in a terminal cancer patient.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

U.S. EPA (1988) reports that thallium salts are potent poisons that cause acute toxicity in humans. Accidental ingestion of thallium salt rodenticides and insecticides, internal and topical use of thallium as a depilatory agent have all resulted in human poisoning (Gettler and Weiss, 1943; Moeschlin, 1980). In children, Bedford (1928) reports that acute toxicity appears to be approximately 6 mg thallium/kg/day. Moeschlin (1980) reports approximately 8-12 mg thallium/kg as the average lethal dose for adults. Independent of the species or the type of thallium salt administered, U.S. EPA (1988) reports that the acute oral LD_{50} values in rats and mice range from 16 to 35 mg thallium/kg.

Chronic oral exposure of a population living in the vicinity of a cement factory that discharged large quantities of thallium into the atmosphere through the ingestion of fruits and vegetables grown in the area appears to have resulted in an increased incidence of neurological and subjective symptoms (Brockhaus et al., 1980, 1981; Dolgner et al., 1983). Subchronic oral exposure of laboratory animals to concentrations greater than 0.25 mg/kg/day resulted in neurological and skeletal muscle effects (Mazo et al., 1983, Deshimaru et al., 1977), hair loss, elevated kidney weights, body weight loss, and mortality (Downs et al., 1960).

U.S. EPA (1979) exposed rats intermittently to thallium (III) oxide via inhalation at 0.5 - 2.0 mg/m_3. Deteriorating health and increased mortality were observed. However, no adverse health effects were observed in workers occupationally exposed to thallium in a magnesium seawater battery plant (Marcus, 1985) or in cement production (Schaller et al., 1980); Ludolph et al., 1986).

Teratogenic and Other Developmental Effects

U.S. EPA (1988) reports on numerous studies which indicate that thallium results in achondroplastic malformations when injected into developing chicken eggs, or tested in mammalian whole embryo cultures or limb bud cultures. Gibson and Becker (1970) observed reduced fetal body weight, hydronephrosis, and the absence of vertebral bodies following parenteral administration of greater than 2 mg thallium/kg/day to pregnant rats. A slight increase in fetal loss was observed following oral administration of thallium to rats (≥2 mg/kg/day) and mice (≥ 4 mg/kg/day) (Roll and Matthiaschk, 1981). Reduced survival at weaning in both species and reduced growth rate in mice were observed in the offspring of rats and mice allowed to deliver, as well. Bornhausen and Hagen (1984) report that adult offspring of dams treated with thallium during gestation had significant learning deficits in a lever-pressing behavior conditioning test.

U.S. EPA (1988) reports that adult male rats exposed to 0.74 mg thallium/kg/day in the drinking water had decreased sperm motility, inhibition of ß-glucoronidase activity, and histopathological alterations of the testes after 60 days of exposure but not after 30 days.

Mutagenic Effects

Data on the mutagenicity of thallium is mixed. Negative results have been obtained in reverse mutation tests (Kanematsu et al., 1980; Singh, 1983) and in tests for effects on cell division (Loveless et al., 1954). Positive results were obtained in a rec assay (Kanematsu et al., 1980) and in several mammalian test systems, including a dominant lethal test in male rats (Zasukhina et al., 1983).

Carcinogenic Effects

Data regarding the carcinogenicity of thallium were not available in the literature reviewed.

Ecotoxicity

In freshwater aquatic systems, U.S. EPA (1980) reports that acute sensitivity of *Daphnia magna* and the fathead minnow to thallium were similar, with LC_{50} values in the range of 910 to 2180 µg/l. LC_{50} values for the bluegill were approximately two orders of magnitude higher. *Daphnia magna* and the fathead minnow also had similar chronic values; 130 and 57 µg Tl/l, respectively. Exposure of an alga to 110 and 100 µg Tl/l resulted in a 50 percent reduction in chlorophyll *a* and cell numbers, respectively. Atlantic salmon had the highest bioconcentration factor for fishes with a value of 130 for muscle tissue. This species appears to be particularly sensitive to thallium; concentrations as low as 20 µg/l resulted in partial mortality after about 100 days exposure.

In saltwater systems, the mysid shrimp had the greatest acute sensitivity with an LC_{50} of 2130 µg thallium/l. The sheepshead minnow and tidewater silverside had exhibited similar sensitivity to thallium with 96-hour LC_{50} values of 20,900 µg/l and 24,000 µg/l, respectively. 8,400 µg/l produced chronic effects in the sheepshead minnow. 4,080 µg/l resulted in a 50 percent reduction in photosynthesis in a saltwater algal species. Bioconcentration factors less than 20 were observed in two bivalve species exposed for 40 to 88 days.

No data on the ecotoxicity of thallium in terrestrial systems were available in the literature reviewed.

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor:	NA	
Inhalation Slope Factor:	NA	
Chronic Oral RfD:	7.0×10^{-5} mg/kg/day	
Chronic Inhalation RfD:	NA	
Subchronic OraL RfD:	7.0×10^{-4} mg/kg/day	
MCL	0.002 mg/l	
AWQC:	Water and Fish Consumption:	13 µg/l
	Fish Consumption:	48 µg/l

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VANADIUM

Use

Vanadium pentoxide is used as a catalyst in the production of several industrial chemicals. It is also used as a photographic developer, as a coating for welding electrodes, and as an alloying agent (ACGIH, 1984).

Chemical and Physical Properties

AW: 50.9	BP: 3,380°C
SG: 5.96 at 20°C	MP: 1,890°C
Sol. (water): at 20°C insoluble	
Sol. (organics): insoluble	

Fate and Transport

The environmental fate of vanadium varies with each compound. Some compounds are volatile so, atmospheric transport would be a legitimate fate process (ICF, 1985). Vanadium appears to become more water soluble in acidic soils, thus becoming more leachable, and is known to bioaccumulate slightly.

Pharmacokinetics

Vanadium is thought to be stored primarily in fat and blood serum but has been detected in the lungs and intestines in humans (U.S. HEW, 1969).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The principle systemic effects of chronic exposure to vanadium are the irritation of the skin and eyes. Oral exposure to vanadium is known to cause gastrointestinal disturbances. Inhalation exposure to vanadium is known to cause irritation of the lungs and after repeated exposures difficulty in breathing and bronchitis are known to occur (NIOSH, 1977). Vanadium's toxicity seems to increase with the increase in valence number (ICF, 1985).

Teratogenic and Other Developmental Effects/Mutagenic Effects

Vanadium and its compounds have not displayed mutagenic, teratogenic, or developmental effects in several studies performed on laboratory animals (ICF, 1985).

Carcinogenic Effects

Vanadium is not classified as to human carcinogenicity because of insufficient human or animal data. In one study, researchers exposed Swiss mice to vanadyl sulfate at concentrations of 19.8 mg/kg bw for their lifetime. There was no evidence that vanadyl sulfate caused tumors in the mice (NIOSH, 1977). Numerous other studies have resulted with similar conclusions (NIOSH, 1977).

Ecotoxicity

Freshwater organisms have LC_{50} values ranging between 5,000 and 100,000 µg/L. The average LC_{50} value for freshwater organisms is around 10,000 µg/L (ICF, 1985).

No further data regarding the ecotoxicity to wildlife were located.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhaltion Slope Factor:	NA
Chronic Oral RfD:	7.0×10^{-3} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	7.0×10^{-3} mg/kg/day
MCL:	NA
AWQC:	NA

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ZINC

Use

Zinc is a bluish-white, lustrous metal with a distorted hexagonal close-packed structure.

It is used in a variety of manners including, galvanizing sheet iron; as an ingredient of such alloys as bronze, brass, Babbitt metal, German silver, and special alloys for die-casting. It is used as a protective coating for other metals to prevent corrosion; for electrical apparatus, especially dry cell batteries; household utensils; castings; printing plates; building materials; railroad car linings; and automotive equipment. Additionally, zinc is utilized as a reducing agent in organic chemistry; for deoxidizing bronze; extracting gold by the cyanide process; purifying fats for soap; bleaching bone glue; manufacturing sodium hydrosulfite; insulin zinc salts, and as a reagent in analytical chemistry (Windholz, 1983).

Physical and Chemical Properties

AW:	65.38	BP:	907°C
SG:	7.133 at 25°C	MP:	419.58°C

Sol. (water): insoluble in water, some salts are soluble
Sol. (organics): soluble in acid and alkali

Fate and Transport

Zinc is likely to be present in the atmosphere as dust and fumes from zinc production facilities, lead smelts, brass works, automobile emissions, fuel combustion, incineration and soil erosion (Lloyd and Showak, 1984). The U.S. EPA (1984) reports that conversion of zinc into a stable species such as zinc oxide, and not removal through decomposition, may be the fate of atmospheric zinc. Fishbein (1981) reports that atmospheric interactions are minimal for particulates with large aerodynamic diameters because of their short air residence time. Zinc, however, is found at the highest concentrations in particles with an aerodynamic diameter less than 3μm (Fishbein, 1981) and zinc oxide emitted from high-temperature processes (e.g., brass foundries, galvanizing, smelting and welding processes) may have particle sizes in the range of 0.01-0.4μm (NIOSH, 1975). These smaller particles may have a long residence time making speciation (conversion) more likely.

Callahan et al. (1979) reports that sorption is probably the dominant fate of zinc in the aquatic environment. The U.S. EPA (1984) reports that zinc introduced into the aquatic environment is partitioned into sediments through sorption onto hydrous iron and manganese oxides, clay minerals and organic material; and that a small part may be partitioned into the aquatic phase through speciation into soluble zinc compounds. They also report that precipitation of zinc sulfide is an important control on the mobility of zinc in reducing environments; and precipitation of hydroxides, carbonate, or basic sulfate may occur at high zinc concentration. Although they have a tendency to be absorbed more strongly onto the sediments, organic and inorganic ligand complexes may increase the mobility of zinc in aquatic media (U.S. EPA, 1984).

The U.S. EPA (1984) states that zinc is likely to be strongly sorbed onto soil and, if not sorbed, leaching may occur. pH and salinity affect sorption with decreasing pH and increasing salinity factoring desorption (U.S. EPA, 1980). Page (1981) detected zinc in 100 percent of ground water samples from New Jersey indicating that leaching is prevalent.

Pharmacokinetics

The U.S. EPA (1980) states that gastrointestinal absorption of zinc is dependent in part upon the zinc status of the organism. In reference to this statement, the U.S. EPA (1984) states:

> "This is a reasonable conjecture, in that zinc levels in the body are rigidly controlled by various homeostatic mechanisms. Also, it appears that dietary levels of other nutrients may influence the kinetics of zinc absorption. The fact that zinc is excreted, in part, through the gastrointestinal tract complicates quantitation of zinc uptake. It is also likely that the anion associated with zinc, chelation or other complexing moieties may influence gastrointestinal absorption."

^{65}Zn, as the chloride, was shown to be rapidly absorbed in human volunteers by Spencer et al. (1965). Peak plasma values were achieved within 4 hours and average absorption was 50 percent with values ranging from 20-80 percent NRC (1978) also reported varying degrees of absorption. Contrary to this, Stokinger (1981) only found small amounts of zinc being absorbed by laboratory animals.

It appears that dietary protein uptake may enhance zinc uptake (NCR, 1978), while high dietary levels of phytate, a complex organic phosphorus-containing compound in cereal products, may inhibit zinc uptake (U.S. EPA, 1984). However, Arvidsson et al. (1978) found that phytate had little or no influence on zinc uptake when ^{65}Zn was added to bread during baking that was then fed to 11 human subjects. Sandstead et al. (1978) suggests that dietary fiber content may influence the uptake of zinc.

Richards and Cousins (1977) speculate that metallothionen, a low-molecular-weight metal-binding protein in the intestinal mucosa, may bind with zinc and facilitate absorption.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Brown et al. (1964) found that high zinc levels in foods stored in galvanized containers led to severe diarrhea, abdominal cramping, nausea and vomiting upon consumption. Murphy (1970) reported lethargy in a 16-yr. old boy administered 12g of zinc in peanut butter over a 2-day period (in a belief that it would accelerate wound healing). Anemia was observed in 3 children exposed to zinc from toy cars made of zinc alloy. The children played with the cars in

the bath and probably ingested some of the bath water. The children were excreting greater than 1 mg/liter zinc in urine.

Ten young men given 150 mg zinc sulfate for 43-61 days to accelerate wound healing complained of gastric discomfort but no other adverse effects were observed, would healing was accelerated (Pories et al., 1967). Greaves and Skillen (1977), as well, found no adverse effects resulting from administration of 150 mg zinc sulfate to 18 patients.

Prasad et al. (1978) found that prolonged zinc therapy for sickle-cell anemia reduced ceruloplasmin levels to 50 percent of what they had been before therapy. Ceruloplasmin levels were returned to normal by discontinuation of the therapy.

Teratogenic and Other Development Effects

Cox et al. (1969) and Ketcheson et al. (1969) reported reduced copper content in fetal livers (and other tissues) as the only effect resulting from administering 4000 or 5000 ppm zinc to pregnant rats during gestation.

Of a "small group" of women supplementing their diet with 100 mg zinc sulfate during the third trimester of pregnancy, 3 experienced premature deliveries and 1 delivered a still born infant (Kumar, 1976). Kumar (1976) supplemented rats with "100 ppm zinc orally" and found a "significant increase" in the number of fetal resorptions.

Mutagenic Effects

No data regarding the mutagenicity of zinc were found in the literature reviewed.

Carcinogenic Effects

Wallenius et al. (1979) exposed female rats to diets containing 15, 50, or 200 ppm zinc. The palatal mucosa was then painted with 4-nitro-quinoline-n-oxide 3 times/week to induce cancer. After cancer of the palate became grossly visible the animals were killed. Animals exposed to 200 ppm dietary zinc developed macroscopically detectable cancer earlier than rats exposed to the two lower doses. However, in an identical study, Mathur et al. (1979) exposed rats to dietary zinc concentration of 5.9, 50, and 260 ppm. Palatal mucosa was sampled at 3, 9, 13, and 23 weeks after exposure, at which time all rats were killed and examined. Animals on the zinc-deficient diet showed the most advanced histologic changes after 3 weeks. After 20 weeks, cancers were found in both the zinc-deficient and zinc-supplemented groups while the rats on the adequate (50 ppm zinc) diet evidenced only moderate dysplasia.

Ecotoxicity

Acute toxicity of zinc to freshwater aquatic life is dependent on the hardness of the water. The concentration (µg/liter) should not exceed the numerical value given by $e^{(0.83\ [\ln(\text{hardness})] + 1.95)}$. The U.S. EPA (1986) gives the following examples: "at hardnesses of 50, 100, and 200 mg/liter $CaCO_3$, the concentration of total recoverable zinc should not exceed 180, 320, and 570 µg/liter at any time". The 24-hour average concentration should not exceed 47 µg/liter (U.S. EPA, 1986).

In saltwater systems, the 24-hour average concentration should not exceed 58 µg/liter and the concentration at any time should not exceed 170 µg/liter (U.S. EPA, 1986).

ICF (1985) reports that zinc poisoning has occurred in cattle. Poisoning was caused by an accidental contamination of food in one outbreak with a zinc concentration of 20 g/kg. It was estimated that the cows had a daily intake of 140 g for about 2 days. The exposed cows exhibited severe enteritis, some died and some had to be slaughtered. Severe pulmonary emphysema with changes in the myocardium, kidneys, and liver with extremely high concentrations of zinc in the liver were detected in post-mortem studies.

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	2.0×10^{-1} mg/kg/day
MCL:	5 mg/l (secondary)
AWQC:	Water and Fish Consumption - NA
	Fish Consumption Only - NA

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