DRAFT FINAL RISK ASSESSMENT
TRONIC PLATING SITE
FARMINGDALE, NEW YORK

Prepared for:

U.S. Environmental Protection Agency

Contract No.: 68-W9-0003

Work Assignment No.: C02073

TES-6

ALLIANCE
Technologies Corporation

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FARMINGDALE, 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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TABLE OF CONTENTS

TABLE OF CONTENTS (CONTINUED)

TABLE OF CONTENTS (CONTINUED)

TABLE OF CONTENTS (CONTINUED)

TABLES

TABLE OF CONTENTS (CONTINUED)

FIGURES

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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 Tronic Plating Company (Tronic) site in Farmingdale, New York, Alliance Technologies Corporation (Alliance) 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 C02073. 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 a qualitative screening analysis and is generally based on previously published information and on information gathered from state and local agencies.

The risk assessment evaluates actual or potential exposures to site contaminants under current and probable future land use scenarios. Existing site documents such as the RI Report prepared by CA Rich Consultants, Inc. (CA Rich, 1991), the associated Project Operations Plan (CA Rich, 1988), and the Agency of Toxic Substances and Disease Registry's (ATSDR) Preliminary Health Assessment for the Tronic site (ATSDR, 1989) have been utilized. Also, 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:

- Facility Workers
- Trespassers
- Residents
- Utility and 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 defines 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. 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. A qualitative evaluation of environmental risk is provided.

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, 1988).

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

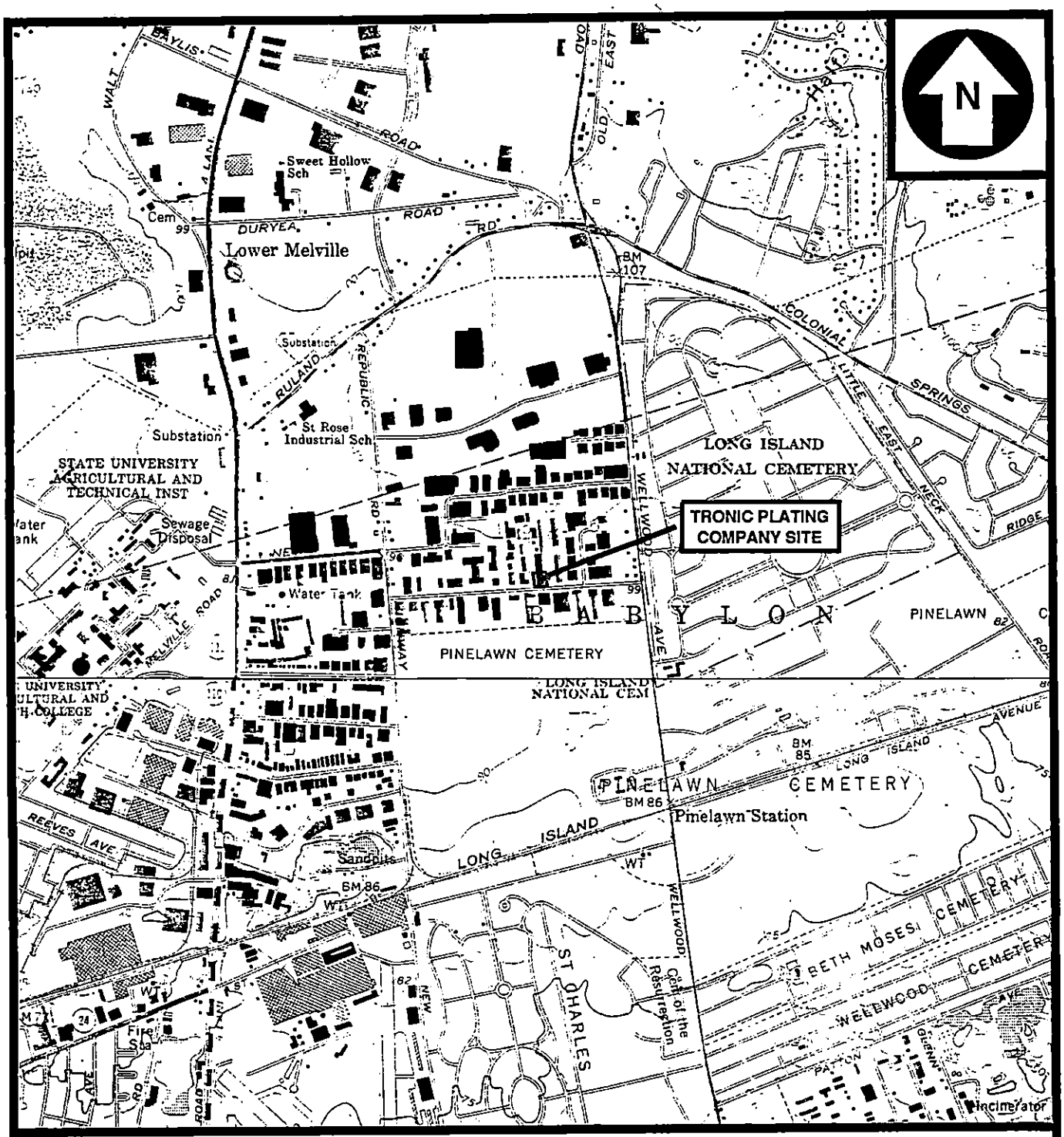
The report is organized into the following sections:

- Section 1 - Introduction
- Section 2 - Hazard Identification
- Section 3 - Contaminant Fate and Transport
- Section 4 - Exposure Assessment
- Section 5 - Toxicity and Dose - Response Assessment
- Section 6 - Risk Characterization
- Section 7 - Discussion of Uncertainties
- 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 and History

The former Tronic facility is located on a 2.68 acre lot at 168 Central Avenue in the hamlet of Farmingdale, Town of Babylon, New York (Figure 1-1). The site consists of the southeastern portion (approximately 7,200 square feet) of an 800 feet long industrial building; a 50 by 75 feet manicured lawn toward the south; and a paved area bordering Commerce Drive toward the east (CA Rich, 1988) (Figure 1-2). Site access is unrestricted by fences or barriers. The site is bordered by commercial and light industrial lots to the north, east, and west, and by Central Avenue to the south. Several cemeteries are located in the vicinity of the site to the south, southeast, and



BASE MAP IS A PORTION OF THE FOLLOWING 7.5' U.S.G.S. QUADRANGLES: AMITYVILLE, NY, 1969, PHOTOREVISED 1979; HUNTINGTON, NY, 1967, PHOTOREVISED 1979

0 1000 2000 3000 feet

QUADRANGLE LOCATION

LOCATION MAP

TRONIC PLATING COMPANY
FARMINGDALE, NEW YORK

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Figure 1-1.

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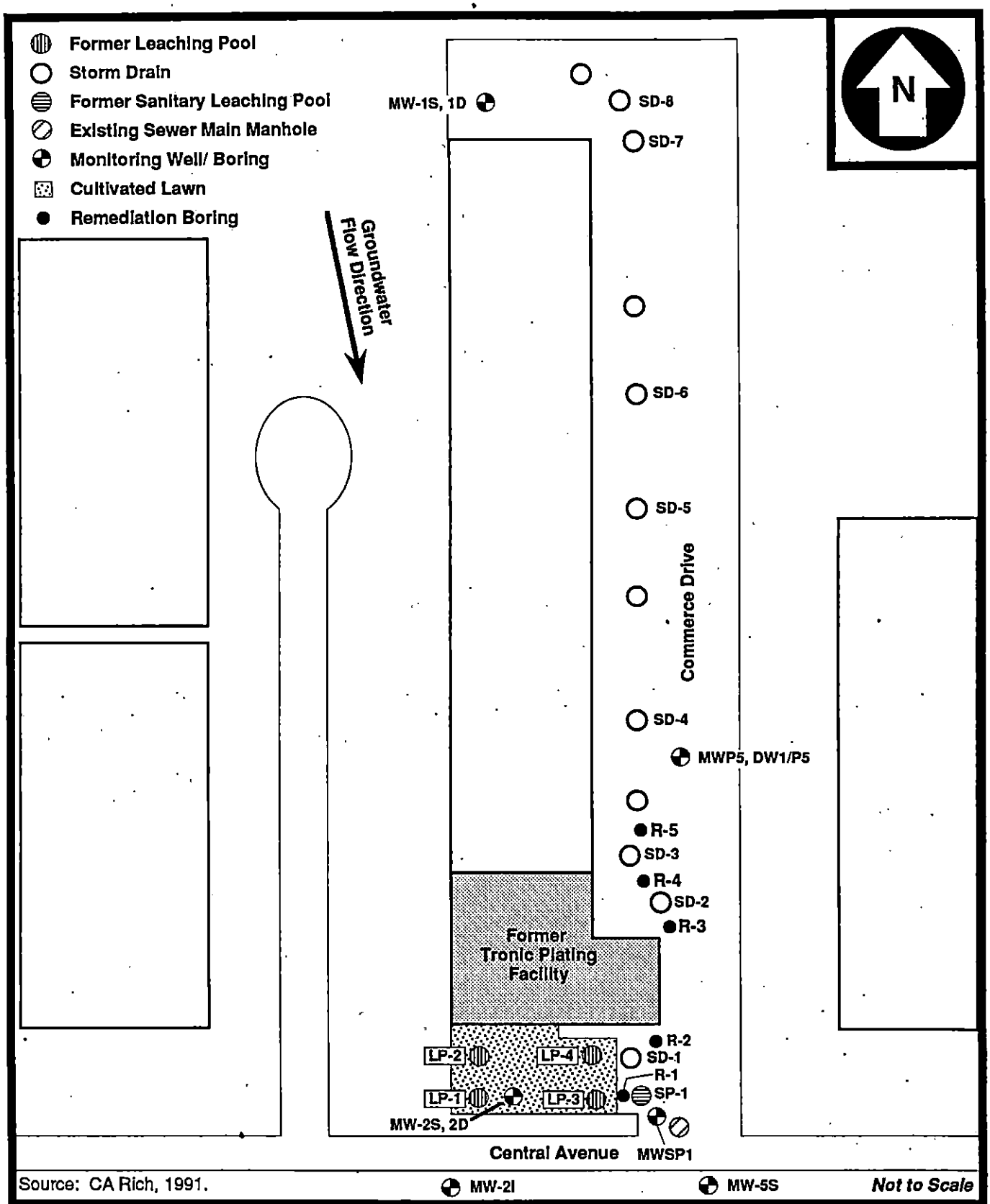

ONSITE SAMPLING LOCATIONS

TRONIC PLATING COMPANY
FARMINGDALE, NEW YORK

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Figure 1-2.

MS39/1/F

east. A heavily wooded area owned by Pinelawn Cemetery and separated from the site by Central Avenue and an industrial lot, is located approximately 500 feet to the south (CA Rich, 1991).

The relatively level surface of the site slopes gently to the south-southeast at a grade of approximately 3 percent. Except for the lawn, the site surface is primarily impermeable given the presence of the building and paved areas. Surface water from precipitation drains from the building and the paved areas into a system of twelve storm drains located along Commerce Drive and the parking area (CA Rich, 1988).

The Tronic Plating Company leased the southeastern portion of the onsite building from Commerce Holding Company, Inc. (CHC, Inc.) between July 1968 and March 1984. In 1984, Tronic moved their operations to 37 Potter Street, in Farmingdale, Nassau County. During their tenancy, Tronic generated approximately 1.25 million gallons of industrial waste water per year from their electroplating, anodizing, and etching processes (CA Rich, 1991). The waste streams reportedly contained cadmium, chromium, copper, cyanide, iron, lead, silver, and zinc, which are characteristically generated by electroplating operations (CA Rich, 1991).

The Tronic facility was serviced by industrial and sanitary leaching pools and a dry well. Four industrial leaching pools are located under the lawn between the facility and Central Avenue. Historical records indicate that these leaching pools received rinsewater discharge from the above processes. The sanitary leaching pool is located below the paved area east of the front lawn and the dry well is situated between the building and Commerce Drive (CA Rich, 1991). Use of the sanitary leaching pool was discontinued in 1983, when the facility was connected to the Suffolk County municipal sewer system.

Indoor features have been altered since Tronic's tenancy. Records indicate that most or all of the metal plating related storage tanks, machinery, and other equipment were removed by the site owners, CHC, Inc., after Tronic vacated the facility. Information regarding subsurface fixtures was not available (CA Rich, 1991).

According to the RI report, four commercial businesses currently operate within the former Tronic building:

- Oakland Supply distributes industrial abrasives and tools;
- EFS Marketing distributes novelty items;
- Farralane Lighting & Audio Company supplies lighting and audio equipment; and
- Infared Optics, Inc. manufactures lenses.

1.3 Summary of Site Investigations

In 1972, the Suffolk County Department of Health Services (SCDHS) analyzed the contents of Tronic's industrial leaching pools and detected cadmium, chromium (total and hexavalent), copper, cyanide, iron, lead, silver, and zinc. Sediments from two storm drains located 40 feet from the facility on Commerce Drive, were subsequently sampled and found to contain similar contaminants.

In 1974, SCDHS contacted Tronic regarding the unpermitted discharge of industrial waste. The company was cited for "...illegal and unauthorized dumping of potentially hazardous wastewater into two storm drains..." (CA Rich, 1991). Administrative Orders on Consent (AOCs), ordering Tronic to cease discharging to the storm drains, were issued by both SCDHS and the New York State Department of Environmental Conservation (NYSDEC). Between 1979 and 1982, SCDHS sampled the contents of

the industrial leaching pools. During the initial sampling event, SCDHS personnel observed blue-green stains leading from Tronic's rear door to a storm drain located approximately 33 feet away. Samples were subsequently collected from the storm drain. The results of their analyses indicated the presence of heavy metals, identical to those detected in Tronic's waste water, in both the industrial leaching pool and storm drains. Similar sampling results were obtained in 1981 and 1982. The contents of the sanitary leaching pool were also examined and found to contain heavy metal contamination (CA Rich, 1991).

SCDHS records indicate that in 1983, Tronic hired Chemical Management, Inc., a local hazardous waste management service, to remove the contents of the industrial leaching pools. However, the records did not indicate that Tronic removed the contents of the storm drains, sanitary leach pit, or dry well. Tronic vacated the property in 1984 before complying with the AOCs (CA Rich, 1991).

In 1984, NYSDEC conducted a Preliminary Inspection of the Tronic facility resulting in the proposed placement of the site on EPA's National Priorities List (NPL) (CA Rich, 1991). In 1986, the Tronic site was placed on the NPL.

The most comprehensive investigation conducted at the Tronic site was a RI performed by CA Rich, pursuant to an Administrative Order issued by EPA to CHC, Inc. Phase I RI activities consisted of soil, ground water, and storm drain sampling; geophysical and topographic surveys; and permeability testing. The study was performed in the spring of 1989. Upon review of the Phase I report, EPA recommended that supplemental work, or Phase II studies be conducted at the site. Phase II activities were similar to, but more extensive than Phase I activities. The Phase II investigation was completed in 1991 (CA Rich, 1991).

This risk assessment is based primarily on data collected during Phases I and II of the RI. Onsite sampling locations evaluated for this report are indicated in Figure 1-2 (see above). Offsite sampling locations are indicated in Figure 1-3. The sample location designations used by CA Rich in the RI report (CA Rich, 1991) have been adopted in this report with some modification (see Section 2).

1.4 Summary of Site Contamination

Results of the studies discussed previously indicate that ground water, soils, and storm drains at the Tronic site are contaminated primarily with volatile organic compounds (VOCs) and metals. The discussion that follows summarizes the analytical results for each medium sampled to date. The discussion is based primarily on a direct review of the Phase I and Phase II RI analytical results, which are summarized in Appendix A.

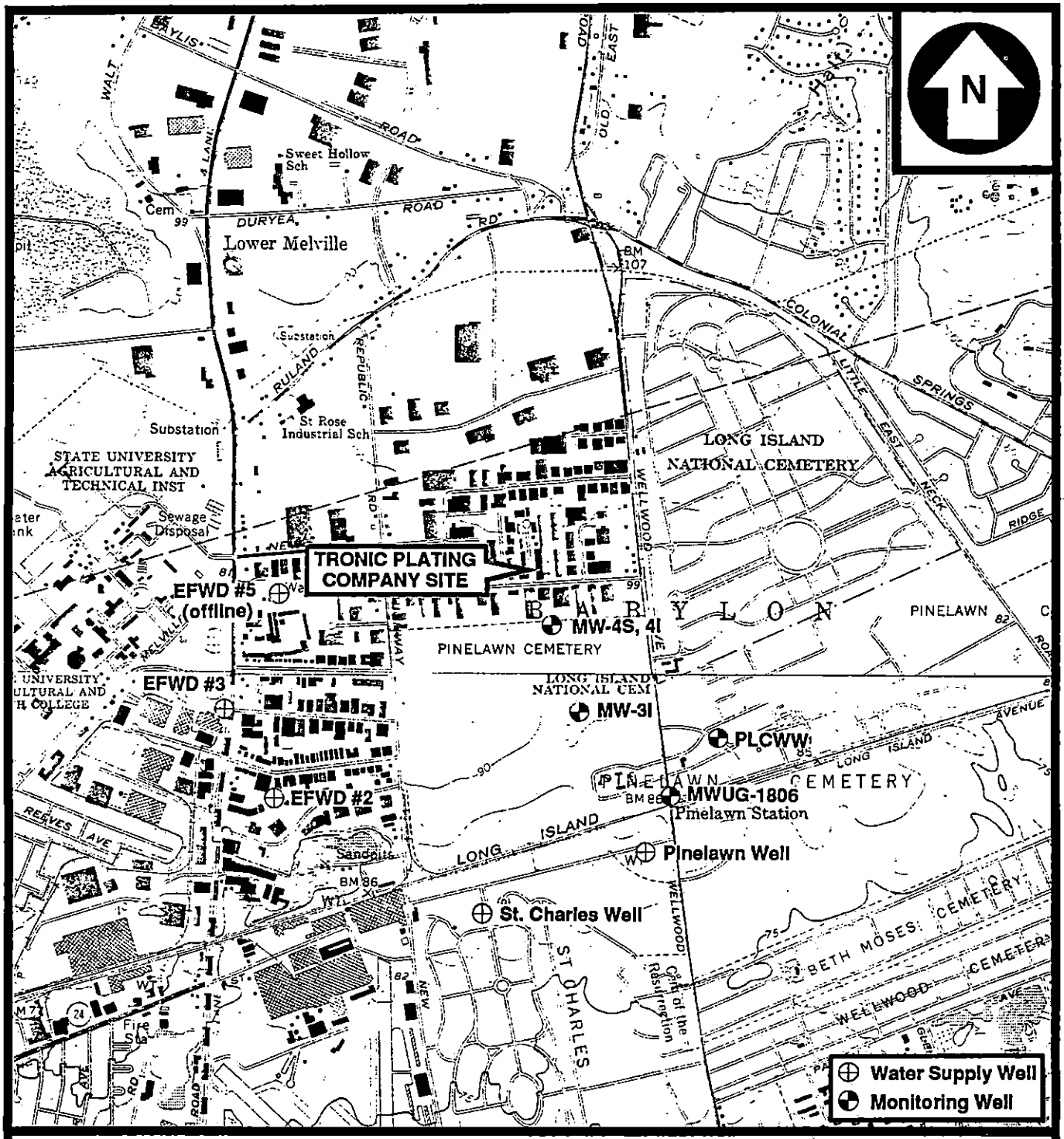
1.4.1 Ground Water Contamination

Ground water samples collected from wells at and in the vicinity of the Tronic site contained primarily VOCs and inorganics. Bis(2-ethylhexyl)phthalate, a base-neutral/acid extractable (BNA), was detected in a single sample.

This discussion focuses on results from the eight monitoring wells sampled during the RI. Results from a USGS well (MWUG-1806) were not included in the quantitative risk assessment (see Section 2) and are not described here.

Volatile Organic Compounds

Thirteen VOCs were detected in ground water samples collected during three RI sampling rounds. Three were present at concentrations equal to or exceeding the federal Maximum Contaminant Levels (MCLs). The most prevalent of these were trichloroethylene (TCE) (MCL = 5 µg/l), detected in 23 of 24 samples at



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0 1000 2000 3000 feet

QUADRANGLE LOCATION

OFFSITE SAMPLING LOCATIONS AND WATER SUPPLY WELLS

TRONIC PLATING COMPANY
FARMINGDALE, NEW YORK

ALLIANCE Technologies Corporation

Figure 1-3.

MS39/1/O

concentrations up to 490 µg/l, and tetrachloroethylene (PCE) (MCL = 5 µg/l) detected in 20 of 24 samples at concentrations up to 21 µg/l. One other VOC, 1,1-dichloroethylene (1,1-DCE), was detected at a concentration equal to the MCL of 7 µg/l.

Inorganics

Twenty-three inorganics were detected in unfiltered ground water samples collected during the RI. The metals most frequently detected were aluminum, barium, lead, magnesium, manganese, nickel, and hexavalent chromium. Six analytes, antimony, beryllium, cadmium, lead, nickel, and thallium, were detected at concentrations exceeding MCLs or Federal Action Levels (lead only). Of these, only lead (17 of 18) and nickel (10 of 24) were detected frequently. Lead concentrations ranged from 4 to 75.5 µg/l (Federal Action Level = 15 µg/l). Nickel was detected at concentrations ranging from 31.2 to 114 µg/l (MCL = 100 µ/l).

1.4.2 Soil Contamination

Surface Soils

The site is largely paved or covered with a building. No surface (under the lawn) or near surface (directly beneath pavement) soil samples were collected during the RI.

Subsurface Soils

This discussion focuses on samples collected from depths of 16 feet or less below grade. Two samples from beneath the storm drains (SD1[14-16] and SD2[14-16]) are included with the other soil samples. These samples are assumed to be of greatest relevance to public health in light of potential future excavation and/or redevelopment of the site. Results from deep subsurface soil samples (greater than 16 feet) are

summarized in Appendix A. Results from shallow (0-1 foot) and deep (greater than 16 feet) storm drain sediments are presented in Section 1.4.3 and Appendix A.

Subsurface soil samples were collected below and around the leaching pools, sanitary leaching pit, and storm drains. Samples to characterize background concentrations were also collected from three depths at one soil boring located immediately north of the Tronic facility.

Subsurface soil samples collected during the RI contained VOCs, BNAs, and inorganics.

Volatile Organic Compounds

Five VOCs were detected: methylene chloride, acetone, styrene, xylene, and Freon-113. However, only two compounds were detected in a relatively large number of samples: styrene and acetone were detected in approximately 50 percent of the 12 samples analyzed. All VOC concentrations were low, i.e., within an order of magnitude of quantitation limits.

Base-Neutral/Acid Extractables

Two BNAs, di-n-butylphthalate and bis(2-ethylhexyl)phthalate (BEHP), were detected in four and five of the twelve subsurface soil samples, respectively. Di-n-butylphthalate was detected at concentrations ranging from 94 to 2,600 µg/kg and BEHP was detected at concentrations ranging from 160 to 2,800 µg/kg. Both were detected at concentrations approximately one order of magnitude higher than quantitation limits. Other BNAs were detected infrequently.

Inorganics

Twelve metals (aluminum, barium, calcium, chromium, copper, iron, lead, magnesium, manganese, sodium, vanadium, and zinc) were detected in greater than half of the subsurface soil samples. The highest detected concentrations for each of these analytes exceeded the maximum levels measured in the background boring (MW-1, See Appendix A). In addition, several other metals that were less frequently detected exceeded the concentrations in background samples or were not detected in background samples.

In addition to metals, cyanide was detected in 4 of 18 subsurface soil samples collected at the site. Cyanide concentrations ranged from 0.4 to 46.9 mg/kg.

1.4.3 Storm Drain Sediments

Sediments collected from the onsite storm drains contained numerous VOCs, BNAs, and inorganic compounds. This discussion focuses on results of samples collected from the 0 to 1 foot depth. Results from other sediment samples are summarized in Appendix A ("Deep Sediments" [>16]) or are included with subsurface soil data (see Section 1.4.2).

Volatile Organic Compounds

Twelve VOCs were identified in the onsite storm drains, with most at concentrations less than 40 µg/kg. Acetone, trichloroethylene, and xylene were present at levels exceeding quantitation limits by approximately an order of magnitude.

Base-Neutral/Acid Extractables

The most frequent detected BNAs detected in the storm drain sediments were polycyclic aromatic hydrocarbons (PAHs). Four PAHs, 2-methylnaphthalene, phenanthrene, fluoranthene, and pyrene, were detected in half or more of the samples collected. Concentrations were at least three times greater than quantitation limits; the highest of these was 2-methylnaphthalene detected at 20,000 µg/kg.

- In addition to PAHs, BEHP was detected in each of the storm drain sediment samples collected at concentrations ranging from 2,400 to 43,000 µg/kg.

Inorganics

All inorganics were detected at concentrations higher than those measured in on-site background soil samples. Seven metals were detected in each of the storm drain sediment samples at concentrations greater than one order of magnitude higher than background concentrations.

Cadmium, not present in the background samples, was detected in storm drain sediments. Minimum detected concentrations of total chromium, copper, magnesium, vanadium, and zinc were at least five times greater than background levels.

1.4.4 Storm Drain Water

Two aqueous samples were collected during the RI from onsite storm drains. Contamination in these samples was limited to inorganics. Storm drain water contaminant concentrations are compared with MCLs or Action Levels because the storm drains discharge directly to ground water. Three analytes were detected at levels in excess of two orders of magnitude higher than MCLs. These were cadmium

(MCL = 5 µg/l), which was detected at 24.7 and 8,270 µg/l; lead (Action Level = 15 µg/l), detected at 138 and 14,100 µg/l; and nickel (MCL = 100 µg/l), detected at 32 and 11,900 µg/l. In addition, one storm drain water sample exhibited concentrations of antimony (73.5 µg/l) and beryllium (27.5 µg/l) in excess of their respective MCLs of 5 and 1 µg/l.

2.0 HAZARD IDENTIFICATION

2.1 Data Evaluation

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

2.1.1 Data Sources

Environmental sampling data used in this risk assessment were collected during the RI investigations conducted by CA Rich. Prior to use in the risk assessment, the analytical results and accompanying documentation were reviewed to ensure that all appropriate, validated data were included. A number of issues arose which were resolved through review of the Remedial Investigation Report (CA Rich, 1991) and communication with Alliance field oversight personnel and EPA.

Analytical results were provided to Alliance on laboratory summary sheets (Form I) and summary tables. Data were available for all environmental media sampled during the RI. The results were manually entered into a computerized data base. During data entry, it became evident that the same sample number was often used twice, once for Phase I, Round 1 samples and once for Phase I, Round 2 samples from the same location. In these cases, the Round 1 sample was identified by adding the suffix 'R1' to the sample number, and Round 2 by adding 'R2' to the sample number.

Table 2-1 presents a list of all samples evaluated for the risk assessment and lists the types of analytical results available. This risk assessment contains data from two phases of sampling. Phase I consisted of soil, storm drain water, storm drain sediment, and two rounds of ground water sampling. Phase II consisted of another round of ground water sampling and additional storm drain sediment sampling.

TABLE 2-1. SAMPLES INCLUDED IN THE TRONIC SITE RISK ASSESSMENT

Medium/Area	Sample Number	VOCs	BNAs	Pest./PCBs	Inorganics
Ground Water	MW5S-B	X			X
Ground Water	MW4S-B (DUP)	X			X
Ground Water	MWP5-R2	X			X
Ground Water	MWSP1-R2	X			X
Ground Water	PLCWW1	X			X
Ground Water	MW1D-B	X			X
Ground Water	MW1D-R1	X	X	X	X
Ground Water	MW1D-R2	X			X
Ground Water	MW1S-B	X			X
Ground Water	MW1S-R1	X	X	X	X
Ground Water	MW1S-R2	X			X
Ground Water	MW2D-B	X			X
Ground Water	MW2D-R1	X	X	X	X
Ground Water	MW2D-R2	X			X
Ground Water	MW2I-B	X			X
Ground Water	MW2I-R1	X	X	X	X
Ground Water	MW2I-R2	X			X
Ground Water	MW2S-B	X			X
Ground Water	MW2S-R1	X	X	X	X
Ground Water	MW2S-R1 (DUP)	X	X	X	X
Ground Water	MW2S-R2	X			X
Ground Water	MW3I-B	X			X
Ground Water	MW3I-R1	X	X	X	X
Ground Water	MW3I-R2	X			X
Ground Water	MW3I-R2 (DUP)	X			X
Ground Water	MW4I-B	X			X
Ground Water	MW4S-B	X			X
Subsurface Soils (a)	DW1P5(5-7)	X	X	X	X
Subsurface Soils (a)	LP1(6-10)	X	X	X	X

TABLE 2-1. (CONTINUED)

Medium/Area	Sample Number	VOCs	BNAs	Pest./PCBs	Inorganics
Subsurface Soils (a)	LP2(14-16)	X	X	X	X
Subsurface Soils (a)	LP2(8-10)	X	X	X	X
Subsurface Soils (a)	LP3(14-16)	X	X	X	X
Subsurface Soils (a)	LP3(5-9)	X	X	X	X
Subsurface Soils (a)	LP4(14-16)	X	X	X	X
Subsurface Soils (a)	LP4(5-9)	X	X	X	X
Subsurface Soils (a)	MW2D(10-12)	X	X	X	X
Subsurface Soils (a)	MW2D(DUP)	X	X	X	X
Subsurface Soils (a)	MW2I(10-12.5)	X	X	X	X
Subsurface Soils (a)	MW3I(10-12)	X	X	X	X
Subsurface Soils (a)	R1(9-11)				X
Subsurface Soils (a)	R2(9-11)				X
Subsurface Soils (a)	R3(9-11)				X
Subsurface Soils (a)	R4(1-3)				X
Subsurface Soils (a)	R4(9-11)				X
Subsurface Soils (a)	R5(9-11)				X
Subsurface Soils (a)	SP1(7-9)	X	X	X	X
Background Soils	MW1D(10-12)	X	X	X	X
Background Soils	MW1D(25-27)	X	X		X
Background Soils	MW1D(37-40)	X	X		X
Deep Subsurface Soils*	DW1P5(19-21)	X	X	X	X
Deep Subsurface Soils*	DW1P5(37-39)	X	X		X
Deep Subsurface Soils*	LP1(14-19)	X	X		X
Deep Subsurface Soils*	LP1(18-20)	X	X		X
Deep Subsurface Soils*	LP1(38-40)	X	X		X
Deep Subsurface Soils*	LP2(18-20)	X	X		X
Deep Subsurface Soils*	LP2(38-40)	X	X		X
Deep Subsurface Soils*	LP3(18-20)	X	X		X
Deep Subsurface Soils*	LP3(38-40)	X	X		X

TABLE 2-1. (CONTINUED)

Medium/Area	Sample Number	VOCs	BNAs	Pest./PCBs	Inorganics
Deep Subsurface Soils*	LP4(16-20)	X	X		X
Deep Subsurface Soils*	LP4(38-40)	X	X		X
Deep Subsurface Soils*	MW2D(20-22)	X	X		X
Deep Subsurface Soils*	MW2D(37-40)	X	X		X
Deep Subsurface Soils*	MW2I(22.5-25)	X	X		X
Deep Subsurface Soils*	MW2I(37.5-40)	X	X		X
Deep Subsurface Soils*	MW3I(32-35)	X	X		X
Deep Subsurface Soils*	MW3I(75-77.5)	X	X		X
Deep Subsurface Soils*	MW4I(64-66)	X			X
Deep Subsurface Soils*	MW4S(39-41)	X			X
Deep Subsurface Soils*	MW5S(39-41)	X			X
Deep Subsurface Soils*	R1(19-21)				X
Deep Subsurface Soils*	R1(29-31)				X
Deep Subsurface Soils*	R2(19-21)				X
Deep Subsurface Soils*	R2(29-31)				X
Deep Subsurface Soils*	R3(19-21)				X
Deep Subsurface Soils*	R3(29-31)				X
Deep Subsurface Soils*	R4(19-21)				X
Deep Subsurface Soils*	R4(29-31)				X
Deep Subsurface Soils*	R5(19-21)				X
Deep Subsurface Soils*	R5(29-31)				X
Deep Subsurface Soils*	SP1(19-21)	X	X	X	X
Deep Subsurface Soils*	SP1(37-39)	X	X	X	X
Deep Subsurface Soils*	SP2(37-39)	X	X		X
Storm Drain Water	SD1-R1	X			X
Storm Drain Water	SD2-R1	X			X
Storm Drain Sediments (c)	SD1(BOTTOM)		X		
Storm Drain Sediments (c)	SD1(BOTTOM)-R1	X			X
Storm Drain Sediments (c)	SD2(BOTTOM)		X		

TABLE 2-1. (CONTINUED)

Medium/Area	Sample Number	VOCs	BNAs	Pest./PCBs	Inorganics
Storm Drain Sediments (c)	SD2(BOTTOM)-R1	X			X
Storm Drain Sediments (c)	SD3(BOTTOM)	X	X		X
Storm Drain Sediments (c)	SD4(BOTTOM)	X	X		X
Storm Drain Sediments (c)	SD5(BOTTOM)	X	X		X
Storm Drain Sediments (c)	SD6(BOTTOM)	X	X		X
Storm Drain Sediments (c)	SD7(BOTTOM)	X	X		X
Storm Drain Sediments (c)	SD8(BOTTOM)	X	X		X
Storm Drain Sediments (c)	SD2(BOTTOM) (DUP)		X		
Deep Storm Drain Sediments (b)	SD1(14-16)	X			X
Deep Storm Drain Sediments*	SD1(21-23)	X			X
Deep Storm Drain Sediments*	SD1(31-33)	X			X
Deep Storm Drain Sediments (b)	SD2(14-16)	X			X
Deep Storm Drain Sediments*	SD2(21-23)	X			X
Deep Storm Drain Sediments*	SD2(31-33)	X			X
Deep Storm Drain Sediments*	SD3(17-19)	X			X
Deep Storm Drain Sediments*	SD3(23-25)	X			X
Deep Storm Drain Sediments*	SD3(31-33)	X			X
Deep Storm Drain Sediments*	SD1(21-23) (DUP)	X			X
Deep Storm Drain Sediments*	SD2(21-23) (DUP)	X			X
USGS Observ. Well*	MWUG-1806-R1	X	X	X	X
USGS Observ. Well*	MWUG-1806-R2	X			X

*Evaluated but not included in the quantitative risk assessment.
(a) Group A Subsurface Soils.
(b) Sediments samples between 1 and 16 feet were included with Group A Subsurface Soils (Group B).
(c) Surface sediment samples (0-1 foot) were included with Group B Subsurface Soils (Group C).
See Section 2.1.2 for further discussion of subsurface soil groups A, B, and C.

2.1.2 Data Analysis

Samples from the following media were considered for analysis in the risk assessment:

- Ground water
- Subsurface soil
- Storm Drain Water
- Storm Drain Sediments

Multiple ground water results were available from the three sampling events occurring between 1989 and 1991. Alliance reviewed these data noting strong similarities in both the detected chemical constituents and concentrations between sampling rounds. In addition, the data included results from two piezometers. Although the difference in construction of these piezometers versus monitoring wells could potentially alter the contaminant concentrations detected, an evaluation of the results indicated the data were comparable. Thus, they were evaluated with other ground water samples.

In accordance with current risk assessment guidance (EPA, 1989a), the results of all samples from all sampling events were considered as individual data points in calculating summary statistics and exposure point concentrations. This approach maximized the size of the data base used to quantify site risks. This approach is also advantageous because maximum concentrations for each monitoring well were retained and not "masked" by averaging with data from other sampling rounds.

Results from the off-site USGS observation well MWUG-1806 (see Figure 1-3) were not included due to unknown well construction and high turbidity. Results for two samples collected from this well during the RI are presented in Appendix A.

Subsurface soil samples from depths at or shallower than 16 feet and storm drain sediments collected from 0 to 1 foot were evaluated in this risk assessment because they are considered accessible under maintenance/excavation scenarios. To fully

characterize potential exposures to subsurface contamination, subsurface soil/sediment data were evaluated under three different data groupings. Group A consists of those subsurface soils collected from onsite soil borings as well as leach pit, sanitary pit, and dry well soils. Group B includes these samples as well as storm drain sediment samples collected between 1 and 16 feet. Group C includes all the Group B samples as well as the storm drain sediments collected between 0 and 1 foot. These 0 to 1-foot sediment samples were also evaluated independently from subsurface soils. Deep subsurface soils and sediments (> 16 feet) were eliminated from the quantitative analysis because exposure to soils and sediments at these depths was deemed to be negligible. Soil sample R4 (1-3) was evaluated with subsurface soils since it is covered by pavement and was collected across an interval (1-3 feet) that typically separates surface (0-2 feet) and subsurface (> 2 feet) soils. Leach pit, sanitary pit, and dry well soils were grouped with the boring soil samples because an evaluation of the analytical results indicated that contamination in these potential source areas did not differ significantly from the site-wide subsurface soil contamination profile; i.e., no "hot spots" exist.

Analytical results of subsurface soils from boring MW-1D were examined and deemed to be representative of background concentrations for inorganics.

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

- Validation qualifiers on concentration values;
- Sample duplicates;
- Sample dilutions;
- Sample re-extracts; and
- Elevated quantitation limits.

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 (SQL) was used. This procedure is described further below. Estimated results, usually indicated by a "J" qualifier, were included in summary statistics.

Duplicates of the following seven samples were included with the RI sampling data:

MW2S-R1
MW3I-R2
SD1(21-23)
SD2(21-23)
SD2(BOTTOM)
MW4S-B
MW2D(10-12)

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 negative results (non-detects). Conservatively, if one sample showed a positive result and the other a negative result, the positive result was used.

The RI 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 RI data also included several samples that required re-extraction during analysis. Based on notations on the Form Is, 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.

Sample quantitation limits were reviewed to identify unusually high quantitation limits that caused the upper 95 percent confidence limit concentration to exceed the maximum detected concentration. This condition occurred with methylene chloride and chloroform in ground water, and vinyl chloride in storm drain sediments. In each instance, the risk assessment evaluated the observed concentration (maximum detected value) rather than the calculated upper 95 percent confidence limit concentration.

Summary statistics for all chemicals detected in each medium/area evaluated 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 including the number of the sample showing the highest concentration, the geometric mean concentration, the upper 95-percent confidence limit of the mean, and the lowest and highest detection limits for non-detects. 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 lognormally 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:

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

where:

exp = inverse natural log function;

$\bar{Y}$ = 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 Tronic site. The selected COCs are a subset of the total list of contaminants detected and represent those contaminants likely to contribute most to the overall public health risk at the site. COCs were chosen for ground water, subsurface soils (Groups A, B, and C), storm drain sediments (0-1 foot), and storm drain water.

2.2.2 Methodology

A qualitative method was employed to select the COCs. For each medium, all detected contaminants were first grouped by chemical class (VOCs, BNAs, pesticides, and inorganics) to ensure that, where appropriate, contaminants from each class were

represented as COCs. The COCs were then selected based on the following criteria: contaminant concentration, toxicity, and frequency of detection; comparison of inorganic contaminant concentrations to background concentrations, if available; and consideration of the chemical and physical properties of organic contaminants that determine their mobility, persistence, and likelihood for bioaccumulation in the environment.

In general, the most toxic, mobile and persistent contaminants, and those found frequently and at high concentrations at the site were selected as COCs. However, the method used was conservative, favoring the inclusion of most contaminants in the analysis rather than the selection of only a few "indicator" chemicals. Each selection criterion is discussed in detail below:

- **Concentration, toxicity, and frequency of detection.** The concentration of a contaminant, its toxicity, and the frequency at which it was detected in each medium were considered collectively in the selection of COCs. Concentration and frequency of detection are summarized in Appendix A. Carcinogenic potential, slope factors, RfDs, and health advisories were used to assess relative toxicities. Appendix B summarizes these toxicity values for each of the chemicals detected at the Tronic site. In general, if the detection frequency of a contaminant was less than 5 percent, the contaminant was not selected as a COC unless it was detected at a relatively high concentration.

- **Chemical and physical properties.** The water solubility, octanol/water partition coefficient (log K_{ow}), organic carbon partition coefficient (K_{oc}), fish bioconcentration factor, Henry's Law constant, vapor pressure, and half-life were also considered in the COC selection process. In general, those contaminants with the greatest migration potential, relatively long half-lives, and high bioconcentration factors were considered for inclusion as COCs. Appendix C presents a summary of these properties for each of the organic chemicals detected at the Tronic site.

  The tendency of an organic compound to partition between an organic phase (e.g., fish, soil) and water may be predicted with log K_{ow} values. Chemicals with low log K_{ow} values (i.e., <2) tend to have high water solubilities, low propensities to adsorb to soils/sediments, and low tendencies to bioconcentrate

in aquatic organisms (Lyman et. al., 1982). These compounds are relatively hydrophilic; whereas, compounds with high log K_{ow} values (i.e., >4) are relatively hydrophobic.

The K_{oc} indicates the sorption potential of organic compounds. A typical range of K_{oc} values is from 1 to 10^7 mL/g. Chemicals with low K_{oc} values tend to leach from soil and be mobile in ground water. High K_{oc} values indicate greater sorption potential, less mobility, and corresponding high bioconcentration factors.

Vapor pressure and Henry's Law constant are indicators of chemical volatility. The properties are used to assess the potential for chemical releases to air from spills, contaminated surface soil, and surface water.

- **Comparison of inorganic contaminants in soil to background concentrations.** During the RI, three soil samples (MW-1D[10-12], MW-1D[25-27], MW-1D[37-40]) were collected to characterize local background conditions. These samples were collected from one soil boring location approximately 800 feet north of the former Tronic facility. This area is believed to be uninfluenced by past Tronic activities. Although sample results indicate that a small amount of organic contamination may be present in these samples (Appendix A), concentrations of inorganics were deemed to be representative of background conditions. Table 2-2 presents concentration ranges, frequencies of detection, and geometric mean concentrations of inorganics detected in the background samples. One-half the sample detection limit for all non-detect results was used to calculate geometric means.

  A large number of inorganic contaminants were detected in most soil samples analyzed. Many of the inorganics are common constituents of soil, (e.g., aluminum, arsenic, barium, chromium, copper, lead, nickel, and zinc). EPA guidance suggests excluding the elements from further consideration in the risk assessment if they only slightly exceed background concentrations (EPA, 1989a). However, because these contaminants may be toxic at high concentrations, this risk assessment evaluated all inorganics present at concentrations higher than site background.

  Both site background data and regional background data from the USGS (also presented in Table 2-2) were available for comparison purposes. In selecting the COCs, the site background data were used as the primary reference source. Regional background is referenced in those instances where inorganics with low toxicities are excluded even though their concentrations exceeded site background. Given the relatively low concentrations of inorganics detected in

TABLE 2-2. BACKGROUND CONCENTRATIONS OF INORGANICS IN SOILS FOR THE TRONIC SITE AND THE EASTERN U.S. (mg/kg)*

Analyte	Number of Times Detected	Number of Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Geom. Mean Conc.	Conc. Range For Soils In Eastern US	
Aluminum	3	3	651	1140	862	7000 -	>100000
Antimony	0	3	--	--	--	<1 -	8.8
Arsenic	0	3	--	--	--	<0.1 -	73
Barium	0	3	--	--	--	10 -	1500
Beryllium	0	3	--	--	--	<1 -	7
Cadmium	0	3	--	--	--	--	
Calcium	3	3	493	538	514	100 -	280000
Chromium (total)	3	3	3.7	6.9	5.13	1 -	10000
Chromium (hexavalent)	3	3	0.12	0.45	0.21	--	--
Cobalt	2	3	2.6	3.3	2.05	<0.3 -	70
Copper	3	3	4.0	7.2	5.69	<1 -	7000
Iron	3	3	1740	3790	2531	100 -	>100000
Lead	1	3	1.1	1.1	0.65	<10 -	300
Magnesium	3	3	247	322	291	50 -	50000
Manganese	3	3	52.6	74.5	62.6	<2 -	7000
Mercury	0	3	--	--	--	0.01 -	3.4
Nickel	0	3	--	--	--	<5 -	700
Potassium	0	3	--	--	--	50 -	37000
Selenium	0	3	--	--	--	<0.1 -	3.9
Silver	0	3	--	--	--	--	
Sodium	3	3	65.8	85.6	75.1	<500 -	50000
Thallium	0	3	--	--	--	2.2 -	23
Vanadium	3	3	2.5	4.5	3.59	<7 -	300
Zinc	3	3	8.3	12.5	9.74	<5 -	2900

*Tronic background results from samples MW-1D(10-12), MW-1D(25-27), and MW-1D(37-40). Eastern U.S. results from Shacklette and Boerngen, 1984.

site background samples, this approach is considered extremely conservative. If maximum regional background data were used, far fewer soil COCs would have been selected.

- **Evaluation of inorganic compounds in ground water.** Numerous inorganic chemicals were detected in the ground water samples analyzed. Both filtered and unfiltered ground water samples were analyzed, and in accordance with EPA guidance, unfiltered results (representing both suspended and dissolved species) were used to assess human health risks because unfiltered ground water is of potable quality (EPA, 1989a). Concentrations of inorganics detected at the Tronic site were compared to concentrations in unfiltered drinking water samples from three East Farmingdale Water District production wells (Veilson, 1992; see Table 2-3). Contaminants detected in concentrations above the East Farmingdale water samples or with high toxicities were retained. Only those inorganics of low toxicity (e.g. essential nutrients), detected in ground water at low frequencies and at low concentrations, were excluded as COCs.

  Site-specific background concentrations were not used to select COCs in ground water because the background sample may not represent naturally-occurring levels. The intended background sample (MW-1S) exhibited elevated concentrations of volatile organic compounds and some metals above regulatory limits (MCLs).

- **Historical information.** Consideration was given to those chemicals which were previously detected in the industrial effluent discharged into the industrial leach pit system (i.e., metals). In most cases, these contaminants were included regardless of the concentration of the contaminant or its frequency of detection.

- **PAHs.** Several different PAHs were detected at the Tronic site at varying concentrations and frequencies. Generally, detected carcinogenic PAHs were included as COCs to account for potential additive effects of individual compounds, regardless of concentration. Noncarcinogenic PAHs were evaluated as potential COCs using the same methodology employed for other organics.

- **Suspect laboratory contaminants.** Common laboratory contaminants, including methylene chloride, acetone, chloroform, and bis(2-ethylhexyl)phthalate, were detected during the RI in several media. None of these chemicals met the criteria for elimination as potential laboratory contaminants.

TABLE 2-3. COMPARISON OF INORGANIC CONCENTRATIONS AT THE TRONIC SITE AND IN UNFILTERED EAST FARMINGDALE, NEW YORK DRINKING WATER (µg/l)*

	Concentrations at the Tronic Site					
	Number of Times Detected	**Number of Samples Analyzed**	**Lowest Detected Concentration**	**Highest Detected Concentration**	**Geom. Mean Concentration**	**Concentration Range for Unfiltered Drinking Water from East Farmingdale Water District****
Copper	8	18	23.2	89.6	12.4	<20
Iron	7	7	22.0	931	121	57 - 164
Manganese	23	24	7.30	3180	82.8	<20 - 48
Sodium	24	24	8710	30500	13803	2360 - 2980
Zinc	3	3	41.6	90.7	55.4	21 -41

*Only inorganics detected in public drinking water samples are listed. See Appendix A for other inorganics detected at the Tronic site.

**Sample results of four active production wells (Nos. 2-2, 3-1, 4-1, 4-2) tested in July, 1991. Laboratory analyses were restricted to the five indicated analytes; full TAL analyses were not performed (Veilson, 1992).

- **Tentatively Identified Compounds.** The Target Compound List (TCL) represents a subset of organic compounds that can be present at a site. However, organic compounds may be 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).

  Three TICs, aldol condensate, unknown amine, and unknown aromatic hydrocarbons, were infrequently detected. They were identified at relatively low concentrations (<19 µg/kg). Toxicity values are not available for these compounds. Aldol condensate belongs to the group of aliphatic alcohols used as detergents (i.e., surfactants), emollients, lubricants, and plasticizers and have relatively low toxicities (acute oral LD_{50}s range from 1.5 to 40 g/kg; Kirk-Othmer, 1978). Further information on TICs detected can be found in the RI report (CA Rich, 1991).

  TICs were not selected as COCs for the following reasons:

  - a large degree of uncertainty is associated with both the identity and estimated concentration 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., unknown aromatic hydrocarbons); and
  - comparatively fewer TICs than TCL compounds were present.

Freon-113, while detected as a TIC in Phase I sampling, was included in the laboratory target analyses in Phase II and is evaluated in the risk assessment.

2.2.3 Medium-Specific Selection of Contaminants of Concern

The COCs selected for each environmental medium sampled at the Tronic site are listed in Table 2-4. The rationale for their selection is presented below.

TABLE 2-4. TRONIC SITE: CONTAMINANTS OF CONCERN

	Ground Water	Subsurface Soils			Storm Drain Water	Storm Drain Sediments
		Group A	Group B	Group C		
Volatiles						
Acetone	X	X	X	X		X
Chloromethane				X		X
1,1-Dichloroethane	X					
1,1-Dichloroethylene	X					
1,2-Dichloroethylene (total)	X			X		X
Ethylbenzene				X		X
Freon-113	X	X	X	X		
Methylene Chloride			X	X		X
Styrene		X	X	X		
1,1,2,2-Tetrachloroethane				X		X
Tetrachloroethylene	X			X		X
Toluene	X					X
1,1,1-Trichloroethane	X					
Trichloroethylene	X			X		X
Vinyl Chloride				X		X
Xylenes (total)		X	X	X		X
(BNAs) Semivolatiles						
Acenaphthene				X		X
Bis(2-ethylhexyl)phthalate	X	X	X	X		X
Chrysene				X		X
Dibenzofuran				X		X
Dimethylphthalate				X		X
di-n-Butylphthalate		X	X	X		
Fluoranthene				X		X
Fluorene				X		X
Indeno(1,2,3-cd)pyrene		X	X	X		
2-Methylnaphthalene				X		X
Naphthalene				X		X
3-Nitroaniline		X	X	X		
Phenanthrene				X		X

TABLE 2-4 (CONTINUED)

	Ground Water	Subsurface Soils			Storm Drain Water	Storm Drain Sediments
		Group A	Group B	Group C		
Pyrene				X		X
Inorganics						
Aluminum	X	X	X	X	X	X
Antimony	X	X	X	X	X	
Arsenic	X	X	X	X	X	X
Barium	X	X	X	X	X	
Beryllium	X			X	X	X
Cadmium	X	X	X	X	X	X
Chromium, hexavalent	X	X	X	X	X	X
Chromium, total	X	X	X	X		X
Cobalt	X	X	X	X	X	X
Copper	X	X	X	X		X
Cyanide	X	X	X	X	X	X
Iron	X	X	X	X		X
Lead	X	X	X	X	X	X
Manganese	X	X	X	X	X	X
Mercury				X	X	X
Nickel	X	X	X	X	X	X
Selenium	X			X	X	X
Silver	X	X	X	X	X	
Thallium	X					
Vanadium	X	X	X	X	X	X
Zinc	X	X	X	X		X

Ground Water

Since ground water is of potable quality without filtration, unfiltered samples (representing both suspended and dissolved contaminants) were assessed.

Volatile Organics: Four of the volatile organic compounds detected in the ground water were eliminated from consideration as COCs (benzene, chloroform, 1,2-dichloropropane, and methylene chloride). They were eliminated because of their isolated occurrence (<5%) and relatively low concentrations (<4 µg/l).

BNAs (Semivolatiles): Only one semivolatile, bis(2-ethylhexyl)phthalate, was detected in ground water. It was retained as a COC.

Pesticides: No pesticides were detected in ground water.

Inorganics: Four of the inorganics detected in the ground water were eliminated from consideration as COCs (calcium, magnesium, potassium, and sodium). They were eliminated because they are regarded as human nutrients with low toxicity. Although sodium was not selected as a COC, elevated levels can be of concern to sensitive populations (e.g., individuals with high blood pressure).

Subsurface Soils

Contaminants detected in deep subsurface soils (>16 feet) were not included in the risk assessment. Humans are highly unlikely to come into contact with such deep soils. Thus, COCs were selected for contaminants detected in subsurface soils to a depth of 16 feet.

Group A Subsurface Soils

Volatile Organics: Methylene chloride was eliminated because of its isolated occurrence at a concentration near the quantitation limit.

BNAs (Semivolatiles): Benzyl alcohol was eliminated because of its isolated occurrence at a concentration near the quantitation limit.

Pesticides: Two pesticides, 4,4'-DDT and 4,4'-DDE, were detected in Group A subsurface soils. Both were eliminated from consideration as COCs for the following reasons:

- isolated occurrence,
- detection in only one medium,
- low sample concentrations (~ 5 times greater than quantitation limits), and
- compounds not associated with site activities.

Inorganics: Of the inorganics detected in Group A subsurface soils, 17 were retained as COCs. Calcium, magnesium, and sodium were not selected as COCs because of low toxicities and because they are regarded as human nutrients.

Group B Subsurface Soils

Volatile Organics: All five of the volatile organic compounds detected in Group B subsurface soils were retained as COCs.

BNAS (semivolatiles): Benzyl alcohol was eliminated because of its isolated occurrence at a concentration near the quantitation limit.

Pesticides: As with Group A subsurface soils, 4,4'-DDT and 4,4'-DDE were eliminated as COCs.

Inorganics: Of the inorganics detected in Group B subsurface soils, 17 were retained as COCs. Calcium, magnesium, potassium and sodium were not selected as COCs because of low toxicities and because they are regarded as human nutrients.

Group C Subsurface Soils

Volatile Organics: 2-Butanone was eliminated because of its isolated occurrence at a trace level (less than the quantitation limit).

BNAS (semivolatiles): Benzyl alcohol was eliminated because of its isolated occurrence at a concentration near the quantitation limit.

Pesticides: As with Group A and Group B subsurface soils, 4,4′-DDT and 4,4′-DDE were eliminated as COCs.

Inorganics: Of the inorganics detected in Group C subsurface soils, 20 were retained as COCs. Calcium, magnesium, potassium, and sodium were not selected as COCs because of low toxicities and because they are regarded as human nutrients.

Storm Drain Water

No VOCs or BNAs were detected in surface water. All but four of the detected inorganics were retained as COCs. Calcium, magnesium, potassium, and sodium were not selected as COCs because of low toxicities.

Storm Drain Sediments

Numerous organic and inorganic contaminants were identified in the storm drain sediments at the Tronic site.

Volatile Organics: Of 12 detected volatile organic contaminants, only 2-butanone was not selected as a COC based on an isolated occurrence at a trace level (less than the quantitation limit).

BNAs (Semivolatiles): All 11 semivolatile organic compounds detected were retained as COCs.

Pesticides: Storm drain sediments were not submitted for pesticide and PCB analysis during the RI effort.

Inorganics: Of the detected inorganics, only four were not selected as COCs (calcium, magnesium, potassium, and sodium). All four exhibit relatively low toxicities and are human nutrients.

3.0 CONTAMINANT FATE AND TRANSPORT

3.1 Introduction

This section describes the fate and transport of chemical contaminants detected at the Tronic site. 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 organic and inorganic (metals) contaminants that are of primary concern from a human health/environmental 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 (semivolatiles), and inorganics. Table 3-1 presents a summary of the physical and chemical properties of the organic contaminants of concern based on values obtained from the literature.

3.2 Potential Routes of Migration

Contamination at the site may be transported from source areas to uncontaminated areas by the movement of contaminated media via natural processes. In the case of the Tronic site, the primary source areas are contaminated storm drain sediments and to a lesser extent the subsurface soils beneath the former industrial leaching pits. In general, contaminant movement will occur as ground water moves 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 transport via ground water. In the case of surface transport, the site features of most concern are surface

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

Detected Compounds	Chem. Class	Water Solubility (mg/L)	Koc Organic Carbon Partition Coeff. (mL/g)	Log Kow Octanol Water Partition Coeff.	Fish Bioconc. Factor (L/Kg)	Vapor Pressure (mm Hg)	Henry's Law Constant (atm*m3/mol)	Soil Half-Life (days)	Surface Water Half-Life (days)	Ref
Acenaphthene	PAH	3.42	4600	4	242	1.55E-03	9.20E-05			A
Acetone	VOC	1000000	2.2	-0.24		2.70E+02	2.06E-05		1.9	A
Bis(2-ethylhexyl)phthalate	BN	0.4		8.73		2.00E-07 (F)	2.57E-07 *			E
Chloromethane	VOC	6500	35	0.95		3.31E+03	4.40E-02			H
Chrysene	PAH	0.0018	200000	5.61		6.30E-09	1.05E-06		0.2	A
Dibenzofuran	BN			4.12						H
Dichloroethane (1,1-)	VOC	5500	30	1.79		1.82E+02	4.31E-03		1-5	A
Dichloroethylene (1,1-)	VOC	2250	65	1.84	5.6	6.00E+02	3.40E-02		1-6	A
Dichloroethylene (1,2-) **	VOC	3500	49	0.7	1.6	2.08E+02	7.58E-03		1-6	A
Dimethylphthalate	BN	4320		2.12		<1.00E-02				H
Di-n-butyl phthalate	BN	13	170000	5.6		1.00E-05	2.82E-07			A
Ethylbenzene	VOC	152	1100	3.15	37.5	7.00E+00	6.43E-03		1.5-7.5	A
Fluoranthene	PAH	0.206	38000	4.9	1150	5.00E-06	6.46E-06		1-2	A
Fluorene	PAH	1.69	7300	4.2	1300	7.10E-04	6.42E-05			A
Freon-113	VOC	10		2		2.70E+02				H
Indeno(1,2,3-cd)pyrene	PAH	0.00053	1600000	6.5		1.00E-10	6.86E-08		0.02-2.08	A
Methylene chloride	VOC	20000	8.8	1.3	5	3.62E+02	2.03E-03		1.2-5.8	A
Methylnaphthalene (2-)	PAH	25.596		3.86			4.81E-04 (D)			C
Naphthalene	PAH	30.6		3.35		2.30E-01 (H)	4.48E-04 (D)			C
Nitroaniline (3-)	BN	890		1.37						H
Phenanthrene	PAH	1	14000	4.46	2630	6.80E-04	1.59E-04		0.38-2.00	A
Pyrene	PAH	0.132	38000	4.88		2.50E-06	5.04E-06			A
Styrene	VOC	300				4.50E+00	2.05E-03			H
Tetrachloroethane (1,1,2,2-)	VOC	2900	118	2.39		5.00E+00	3.81E-04		0.04	H
Tetrachloroethylene	VOC	150	364	2.6	31	1.78E+01	2.59E-02		1.0-30.0	A

TABLE 3-1. (CONTINUED)

Detected Compounds	Chem. Class	Water Solubility (mg/L)	Koc Organic Carbon Partition Coeff. (mL/g)	Log Kow Octanol Water Partition Coeff.	Fish Bioconc. Factor (L/Kg)	Vapor Pressure (mm Hg)	Henry's Law Constant (atm*m3/mol)	Soil Half-Life (days)	Surface Water Half-Life (days)	Ref
Toluene	VOC	535	300	2.73	10.7	2.81E+01	6.37E-03		0.2	A
Trichloroethane (1,1,1-)	VOC	1500	152	2.5	5.6	1.23E+02	1.44E-02		0.1-7.0	A
Trichloroethylene	VOC	1100	126	2.38	10.6	5.79E+01	9.10E-03		1.0-90.0	A
Vinyl chloride	VOC	2670	57	1.38	1.17	2.66E+03	8.19E-02		1.0-5.0	A
Xylene (total)	VOC	198	240	3.26		1.00E+01	7.04E-03		1.5-9.0	A

LEGEND:

VOC - Volatile Organic Compound
A - Acid Extractable Organic Compound
BN - Base/Neutral Extractable Organic Compound
PAH - Polycyclic Aromatic Hydrocarbon
P/PCB - Pesticide/Polychlorinated Biphenyl
* - Estimated Value
** - Properties are reported for cis-1,2-dichloroethylene

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: Treatablility Data, September, 1981.
G - Handbook of Environmental Data on Organic Chemicals, Verschueren, 1977.
H - EPA Basics of Pump-and-Treat 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 in the site vicinity is defined by three aquifers: a shallow unconfined unit, an intermediate semi-confined unit, and a deep confined unit (CA Rich, 1991). The unconfined upper aquifer and semi-confined aquifer may be hydraulically connected under the site. A thick clay layer separates the confined deep aquifer from the two overlying aquifers. While ground water may flow upward or downward between the two shallower aquifers, the predominant flow direction is horizontal. Ground water flows in a southerly direction and occurs at depths ranging from 29 to 35 feet below the surface (CA Rich, 1991).

Contamination was documented during the RI in the upper unconfined aquifer. The lower aquifers were not investigated. Dissolved contaminants leached from the soils will be transported in a southerly direction within the upper aquifer. These contaminants may also be transported downward under natural hydraulic gradients to the semi-confined water bearing unit where they may accumulate further. Dissolved contaminants that reach the semi-confined aquifer will be transported in the direction of ground water flow (not specified in the 1991 RI report).

3.2.2 Surface Transport

Industrial wastewaters were discharged from the Tronic facility to the subsurface environment through leaching pits and storm drains. This disposal history suggests that surface soils are not contaminated with site contaminants, although these soils were not chemically characterized in the 1991 RI Report. In addition, most of the site surface is paved, precluding erosion or other transport of surface soil layers. Subsurface contaminants associated with storm drain and leaching pits are not subject

to surface transport processes. Contaminants in subsurface soils are unlikely to be transported to the surface given the site-specific hydrogeologic processes described in the 1991 RI Report.

3.2.3 Contaminant Releases to Air

The primary contaminated media are storm drain sediments and subsurface soils beneath the leaching pits. The extensive ground cover (pavement and buildings) and remote subsurface location of contaminants will preclude fugitive particulate or vapor emissions from the site. Low levels of VOCs in subsurface soil samples also indicate that volatilization to subsurface spaces (e.g., basements) will be negligible.

3.3 Contaminant Persistence and Migration

Organic and inorganic contaminants were identified in ground water, subsurface soils, and sediments. Only inorganics were detected in storm drain water. Intermedia transfer of contaminants may occur by a variety of processes. The general processes associated with each medium and contaminant type are discussed separately below.

3.3.1 Ground Water

Contamination reported in the ground water consists primarily of VOCs and inorganics. Of the VOCs, chlorinated compounds were detected frequently in monitoring wells; they include: TCE, 1,1,1-trichloroethane (1,1,1-TCA), PCE, and, 1,2-dichloroethylene (1,2-DCE). Acetone and toluene were less abundant in ground water. The high water solubilities of these VOCs indicate they will be transported in a southerly direction with the flow of ground water. The chlorinated VOCs may also be transported downward from the upper to the underlying aquifer since they are heavier than water (specific gravity >1). Their concentrations will be diminished by dispersion and dilution within the water volume and by adsorption to geologic materials.

Metals migration in ground water is a complex process and is dependent upon the phase in which the metal exists (e.g., dissolved species or suspended particulate). At the Tronic site, metals primarily occur in a dissolved phase as indicated by similar concentrations in filtered and unfiltered samples (CA Rich, 1991). Only aluminum, copper, and lead frequently exist as suspended particulates. These three metals are expected to be less mobile than those in the dissolved phase since their movement as suspended particles will be impeded by geologic material. Dissolved metals will be transported south in the direction of ground water flow; concentrations will be lessened by dispersion, dilution, and adsorption to geologic materials.

3.3.2 Subsurface Soils

Few organic contaminants were detected in subsurface soils (Groups A, B, C) at the Tronic site. Among the most abundant organics were phthalates. These chemically stable and persistent compounds are moderately to highly sorbed to soils as evidenced by log K_{ow} values in excess of 4. Phthalates are likely to remain bound to soils with sorption dominating any transport processes. The isolated occurrence of one phthalate in ground water supports this prediction.

Subsurface soils contaminated by metals can act as a source of contamination to ground water through leaching processes. The extent of leaching cannot be evaluated at this time because the pH and eH of the soils and ground water are unknown. These measurements, not determined during the 1991 RI effort, provide information on the acidity and oxidation state of the soils and ground water. This information is required to predict the equilibrium state of the metal ions. Based on the vertical distribution of metals in soils, it appears that these metals are immobile to a large extent. This is suggested by concentrations which tend to diminish with increasing depth.

Regardless of contaminant type, it is likely that extensive pavement and building cover will diminish infiltration of water to the subsurface, thereby reducing movement of contaminants in subsurface soils.

3.3.3 Storm Drain Sediments

Numerous VOCs were identified in the storm drain sediments; acetone, TCE, and xylenes were detected at the highest concentrations. However, since overall concentrations were still relatively low ($\leq$ 180 µg/kg), it is unlikely that significant airborne concentrations will result from volatilization. High water solubilities (>1,000 mg/L) indicate that these compounds may be leached from sediments and transported to ground water; the presence of several volatiles in ground water supports this.

Hydrolysis and microbial degradation are likely to reduce the concentrations of VOCs in sediments (Smith and Dragun, 1984). Though these processes are extremely slow (as long as 178 years) (EPA, not dated), the presence of DCE and vinyl chloride at the site may be indicative of the biodegradation of TCE and/or PCE.

Several PAHs were identified in storm drain sediments. These chemically stable and persistent compounds are moderately to highly sorbed to soils as evidenced by K_{oc} values in excess of 1,000 mL/g. Sorption is expected to dominate the transport process with negligible leaching of PAHs to ground water. The lack of PAH contamination in ground water supports this prediction.

The highest concentrations of metals were present in the upper surface (0-12 inches) of the storm drain sediments. Concentrations of most metals diminished by at least an order of magnitude within approximately five vertical feet. Arsenic, barium, cadmium, chromium, and lead were demonstrated by TCLP analyses to be leachable from these storm drain sediments (CA Rich, 1991). Of these, lead and cadmium were highly

leachable as indicated by leachate concentrations in excess of the TCLP regulatory limits. Leached metals will ultimately be transported with ground water.

3.3.4 Storm Drain Water

Metals constitute the only contaminants identified in the storm drain water. These metals are present primarily as suspended particulates as indicated by higher concentrations in unfiltered than filtered samples (CA Rich, 1991). Antimony, arsenic, barium, chromium, cobalt, lead, and mercury appear to be exclusively present as suspended particulates or colloids. These suspended metals are unlikely to be transported to ground water. Sorption to sediments is expected, with very limited lateral or vertical migration. Conversely, metals occurring as dissolved species, such as cadmium and zinc, are expected to be leached to ground water and transported with ground water in a southerly direction. Their concentrations will be lessened by dispersion, dilution, and adsorption to geologic materials.

4.0 EXPOSURE ASSESSMENT

4.1 Introduction

This section evaluates the likelihood, magnitude, and frequency of exposure to the contaminants of concern at the Tronic 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 must be evaluated to determine which parameters might influence exposure to site contaminants. This information will help support identification of exposure pathways. The physical setting of the Tronic site was described in Section 1.0. This section focuses on actual and potential receptors.

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 Tronic site. The purpose of this analysis was to assess the likelihood of various groups, including sensitive populations, of becoming exposed to site contaminants.

Receptors under Present Land Use: The industrial park which formerly included the Tronic facility is currently occupied by four commercial businesses employing an undetermined number of employees. Surrounding properties engage in light manufacturing and commercial retail (Buttera, 1992). The nearest properties currently zoned for residential use are the Pinelawn Cemetery and Long Island National Cemetery to the south and east of the site. Actual residences are not located within a quarter mile of the Tronic site. Therefore, the only current receptors potentially exposed to site contaminants are facility employees and other area workers.

Receptors under Future Land Use: Future land use zoning is unlikely to involve residential development of the Tronic site or neighboring properties (Buttera, 1992). The Pinelawn Cemetery and Long Island Cemetery cannot be rezoned for 100 years

after the last burial. Both cemeteries have open plots so they are currently active. Therefore future receptors are unlikely to include residents or young trespassers. Facility employees and other workers are predicted to be possible future receptors as well as current receptors.

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).

4.3.1 Media and Exposure Routes of Concern

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

- ground water,
- surface soils,
- subsurface soils (Groups A, B, and C),
- sediments,
- surface water, and
- air.

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.

It should be noted that current EPA Region II guidance (EPA, 1992a) calls for limiting the extent to which dermal contact exposures are evaluated in the quantitative risk assessment. The high degree of uncertainty in inputs for this pathway (e.g., absorption factors) is the primary reason for this recommendation. Guidance is currently available to quantify dermal exposures for three types of contaminants: cadmium, PCBs, and dioxins. Cadmium is the only one of these to be a COC for the Tronic site and is included in the quantitative risk assessment.

Ground Water

Ground water in the vicinity of the Tronic site is considered to be potable without treatment (Beilson, 1992), and is classified by NYSDEC's Division of Water as GA, fresh ground waters "best used as a source of potable water supply" (NYSDEC, 1991; Tucker, 1992). Anthropogenic contamination cannot alter a ground water classification (Tucker, 1992).

Long Island residents draw their drinking water almost exclusively from the ground water. Two of the area's three aquifers are typically utilized as drinking water sources. The Upper Glacial (unconfined) aquifer is the primary source for smaller, private wells and the Magothy (semi-confined) aquifer is the primary source for municipal systems (CA Rich, 1991). The 1991 RI report describes the hydrogeology of the site vicinity in more detail.

The local population obtains drinking water from the public water supply or private ground water wells. Two private municipal water companies, the East Farmingdale Water District (EFWD) and the Suffolk County Water Authority, supply water to the residents of Babylon. The hamlet of Farmingdale is serviced primarily by the EFWD.

Two other water companies, the South Huntington Water District and the Dix Hills Water District, service the area immediately upgradient of the site, in the town of Huntington. Only the EFWD draws ground water from wells located hydraulically downgradient of the site (Beilson, 1992).

The EFWD currently supplies drinking water to approximately 5,500 residents of Farmingdale. They maintain five production wells and are constructing one more in the vicinity of the site. Two of these production wells are located 2.4 miles downgradient of the site near the intersections of Route 109 and the Southern State Parkway. The remaining wells are hydraulically cross-gradient to the site in areas to the south and southwest.

Ground water drawn from EFWD's production wells is mixed before distribution to the public. It is treated with calcium hydroxide to maintain a neutral pH and reduce pipe corrosion, and with sodium hypochlorite to control bacterial growth (Beilson, 1992). The drinking water supplied by EFWD is not filtered prior to public distribution.

Numerous private wells, described in the 1991 RI Report, exist in the site vicinity (CA Rich, 1991). Some are used to supply drinking water to businesses, but most are used as process/cooling water or for irrigation. The nearest downgradient private ground water well services an office at the Saint Charles Cemetery located 0.9 miles south of the site. Wells located closer to the site at the Pinelawn Cemetery are used strictly for irrigation (CA Rich, 1991).

Given that public drinking water supply wells are more than two miles downgradient, residential exposures are unlikely. However, given the presence of private wells on nearby commercial/industrial properties, ingestion of ground water by local workers is evaluated in the exposure assessment. Since unfiltered ground water is of potable quality, data from unfiltered ground water samples were used to estimate drinking water exposures.

Other ground water exposure routes such as dermal contact are considered insignificant in relation to ingestion exposures and are not evaluated in the risk assessment.

Surface and Subsurface Soils

Subsurface soil was selected unequivocally as an exposure medium because plating waste streams were formerly discharged to subsurface soils through a leach pit system and through storm drains. The exposure assessment evaluates both dermal contact with and incidental ingestion of subsurface soils sampled on the site. Three different groupings of subsurface soils/sediments were evaluated in order to ensure that all reasonable potential exposures to subsurface contamination were considered in the risk assessment (see Section 2).

Exposures to surface soils cannot be evaluated quantitatively because this medium was not chemically characterized in the 1991 RI Report (CA Rich, 1991). Risks resulting from exposure to surface soils are likely to be negligible as indicated by: (1) the presence of pavement over a majority of the site which precludes current exposures; (2) the lack of evidence (based on disposal history) to suggest contamination in soils immediately beneath the pavement; and (3) the remote location of contaminants in the subsurface which renders them inaccessible during assumed exposure scenarios. Therefore, surface soil exposures were not evaluated in the risk assessment.

Sediments

Site plans included in the 1991 RI Report (CA Rich, 1991) indicate the presence of 12 storm drains, some with overflow catch basins, on the Tronic site. This report also indicates that wastewater from the Tronic facility was reportedly discharged illegally to two onsite storm drains.

Periodic maintenance of storm drains is typically necessary to maintain flow efficiency. This commonly involves removing the upper layers of the bottom sediments. Maintenance workers are likely to become exposed to these bottom sediments by direct contact or incidental ingestion. This risk assessment evaluates maintenance worker exposures to sediments from upper layers only (0-12 inches), because deeper sediments are less likely to be contacted during these activities. Exposures to sediments are also evaluated in conjunction with subsurface soils, as described above.

No samples were collected from the sanitary pit and dry well. Contents of the industrial leach pits were pumped out and replaced with backfill; exposures are therefore evaluated under subsurface soil scenarios.

Storm Drain Water

Most onsite precipitation is directed to the storm drain system by pavement which covers a majority of the site. Two of these storm drains also reportedly received wastewater from the Tronic facility.

Storm drain water is likely to be contacted by workers during maintenance to sustain flow efficiency; dermal contact was therefore evaluated in the risk assessment. Oral exposures are expected to be negligible during these events and were not evaluated in the risk assessment.

Ambient Air

Aside from routine field screening, no air quality monitoring was performed as part of the RI. The remote location of contamination in the subsurface, and the presence of pavement, buildings, and grass over the site make air contamination unlikely. Short-

term potential future excavation is unlikely to result in significant inhalation exposures to fugitive emissions.

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 include:

- ingestion of ground water;
- incidental ingestion of subsurface soils and storm drain sediments; and
- dermal contact with subsurface soils, sediments, and storm drain water (quantitative evaluation for cadmium only).

4.4 Exposure Scenarios

Multiple exposure scenarios were developed for both present and future use of the Tronic 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 subsections discuss the present and future scenarios examined and the specific exposure parameters used to calculate exposure doses.

TABLE 4-1 TRONIC SITE: SUMMARY OF EXPOSURE PATHWAYS

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
Ground Water							
Ingestion of Ground Water	Worker	No	Yes	X		No residents are located or anticipated in vicinity of site but private wells are located on commercial/industrial property.	All ground water samples except MW-UG1806, which was deemed unacceptable based on turbidity and well construction.
Dermal Contact with Ground Water	Worker	No	No			Considered insignificant compared to ingestion exposures.	
Surface Soils							
Incidental Ingestion of Onsite Surface Soils	Trespasser	No	No			Present exposure precluded by pavement. Future exposures following removal of pavement are expected to be minimal given past disposal history.	
	Resident	No	No				
Dermal Contact with Onsite Surface Soils	Trespasser	No	No			Present exposure precluded by pavement. Future exposures following removal of pavement are expected to be minimal given past disposal history.	
	Resident	No	No				
Subsurface Soils							
Incidental Ingestion of Onsite Subsurface Soils	Excavation Worker	No	Yes	X		Exposure to subsurface soils (≤16') may occur during excavations for utility maintenance/future development.	Group A: All subsurface soils less than or equal to 16'. Group B: Group A plus storm drain sediments between 1 and 16'. Group C: Group B plus storm drain sediments less than or equal to 1'.
	Utility Worker	Yes	Yes	X			
Dermal Contact with Onsite Subsurface Soils	Excavation Worker	No	Yes	X		Exposure to subsurface soils (≤16') may occur during excavations for utility maintenance/future development.	Group A: All subsurface soils less than or equal to 16'. Group B: Group A plus storm drain sediments between 1 and 16'. Group C: Group B plus storm drain sediments less than or equal to 1'.
	Utility Worker	Yes	Yes	X			

TABLE 4-1. (CONTINUED)

Pathway	Receptor	Time-Frame Evaluated		Degree of Assessment		Rationale for Selection or Exclusion	Data Grouping
		Present	Future	Quant.	Qual.		
Storm Drain Sediments							
Incidental Ingestion of Storm Drain Sediments	Utility Worker	Yes	Yes	X		Exposures may occur during periodic maintenance.	Samples from upper sediment surface (0-12").
Dermal Contact with Storm Drain Sediments	Utility Worker	Yes	Yes	X		Exposures may occur during periodic maintenance.	Samples from upper sediment surface (0-12").
Incidental Ingestion of Dry Well, Sanitary Pit, and Leach Pit Sediments	Excavation/Utility Worker	No	No			No dry well or sanitary pit sediments collected. Leach pits were pumped out and backfilled; exposures evaluated under subsurface soil scenarios.	
Dermal Contact with Dry Well, Sanitary Pit, and Leach Pit Sediments	Excavation/Utility Worker	No	No			No dry well or sanitary pit sediments collected. Leach pits were pumped out and backfilled; exposures evaluated under subsurface soil scenarios.	
Storm Drain Water							
Incidental Ingestion of Storm Drain Water	Utility Worker	No	No			Anticipated method of maintenance involves negligible exposure via oral route.	
Dermal Contact with Storm Drain Water	Utility Worker	Yes	Yes	X		Exposures may occur during periodic maintenance.	Storm drain water samples.
Air							
Inhalation of Fugitive Emissions	Worker	No	No			Disposal history and surface cover suggest negligible releases.	

4.4.1 Present and Future Scenarios

The rationale used in selecting present and future exposure scenarios and assumptions used are summarized in Table 4-1 and described below.

Present Conditions

Ground Water: Ingestion of contaminants from drinking water sources was not assessed under present conditions. Private wells were not sampled during the RI, but there is no evidence to suggest they are contaminated. Public supply wells are more than 2 miles downgradient.

Subsurface Soils: Exposure to subsurface soils (Groups A, B, and C) was assumed to be possible for utility workers under present scenarios but was considered rare for purposes of assessing risk. An adult worker was assumed to contact contaminated material during the maintenance of buried utilities.

Storm Drain Sediments (0-12 inches) and Water: Contact with these media was evaluated for present scenarios. Maintenance workers could be exposed to storm drain sediments via dermal contact and incidental ingestion, and to storm drain water by dermal contact. An adult worker was assumed to contact contaminated material during maintenance of storm drains to maintain flow efficiency. Infrequent exposure was assessed.

Future Conditions

Ground Water: Under future scenarios, it was assumed that private wells located on commercial/industrial property downgradient of the site become contaminated by site-related contaminants. This scenario is very conservative and may overestimate exposures since it assumes that onsite contaminant concentrations will migrate to offsite private wells without dilution or degradation. It was assumed that exposures

may result from ingestion of drinking water; exposures resulting from other routes (e.g., dermal contact) were assumed to be insignificant compared to ingestion exposures.

Subsurface Soils: Dermal contact with and incidental ingestion of subsurface soils (Groups A, B, and C) by utility workers and excavation workers was assumed under future scenarios; however, excavation was assumed to occur over a short period of time.

Storm Drain Sediments (0-12 inches) and Water: Maintenance workers were assumed to be exposed to storm drain sediments via dermal contact and incidental ingestion under future scenarios. Dermal contact with storm drain water was also evaluated under future scenarios, while incidental ingestion was considered to be negligible and was not evaluated.

4.4.2 Exposure Parameters

The parameters used to calculate exposure doses are summarized in Tables 4-2 through 4-9 at the end of Section 4. The tables correspond directly to the scenarios presented in Table 4-1. Note that parameters are summarized only for those scenarios evaluated quantitatively.

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, 1988), and *The Exposure Factors Handbook* (EPA, 1989c). In addition, current EPA Region II guidelines (EPA, 1992a) were used for cadmium dermal contact scenarios.

The general worker (ground water ingestion exposure) and the utility worker (soil, sediment, and storm drain water exposures) were each assumed to be a 70 kg adult, exposed to contaminants over a 25-year working lifetime. Exposure frequencies were daily (ground water), 10 days per year (subsurface soils), or 2 days per year (storm drain water and sediments). 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. As recommended by EPA for workers exposed to subsurface soils for short durations, the excavation worker ingests greater quantities of soil than the utility worker (EPA, 1991a).

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 4-9 were applied to this general equation. The specific exposure dose calculations are presented in Appendix D.

To evaluate noncarcinogenic health effects, exposure point concentrations were calculated for acute (single event), subchronic (short-term), and chronic (long-term) exposure periods. For acute doses, the terms for exposure frequency (EF), exposure duration (ED), and averaging time (AT) in the above equation are removed. Subchronic and chronic doses were calculated for exposures over different exposure periods depending on the receptor population. These population-specific exposure periods are identified in Tables 4-2 through 4-9. 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 A WORKER FOR FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Worker	
Body Weight (kg)					
Adult Worker	--	--	70	Per EPA Guidance	RAGS Suppl.
Duration of Exposure (years)					
Adult Worker	1 - 70	35	25	Duration of Employment	RAGS Suppl.
Exposure Frequency (days/year)	1 - 365	182.5	250	Per EPA Guidance	RAGS Suppl.
Ingestion Rate (l/day)					
Adult Worker	--	--	1	Per EPA Guidance	RAGS Suppl.
Averaging Time (days)					
noncarcinogenic	--	--	9125	Values used are based on exposure duration for noncarcinogens and lifetime exposure for carcinogens	RAGS
carcinogenic	--	--	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.

TABLE 4-3. EXPOSURE PATHWAY: INCIDENTAL INGESTION OF SUBSURFACE SOILS* 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

*Groups A, B, and C (see Section 2).
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 4-4. EXPOSURE PATHWAY: DERMAL CONTACT WITH SUBSURFACE SOILS* 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)	0.2 - 1.0	0.6	0.6	Per EPA Region II guidance.	
Absorption Factor (percent) Cadmium	0.1-1	0.5	0.5	Per EPA Guidance	EPA, Reg. II
Averaging Time (days) noncarcinogenic carcinogenic	 -- --		 91 25550	Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS

*Groups A, B, and C (see Section 2).

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 Reg. II: Personal communication from Peter Grevatt, EPA Region II Risk Assessment, February 3, 1992.

TABLE 4-5. EXPOSURE PATHWAY: INCIDENTAL INGESTION OF SUBSURFACE SOILS* BY UTILITY WORKERS FOR PRESENT AND FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Utility Workers	
Body Weight (kg) Utility Worker	--	--	70	Per EPA Guidance	RAGS Suppl.
Duration of Exposure (years) Utility Worker	1 - 30	15	25	Per EPA Guidance	RAGS Suppl.
Exposure Frequency (days/year) Utility Worker	1 - 365	183	10	Assumes maintenance of buried utilities is necessary 10 days/yr	
Ingestion Rate (mg/day) Utility Worker	--	--	100	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	-- --	-- --	9125 25550	Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS

*Groups A, B, and C (see Section 2).
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 4-6. EXPOSURE PATHWAY: DERMAL CONTACT WITH SUBSURFACE SOILS* BY UTILITY WORKERS FOR PRESENT AND FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Utility Workers	
Body Weight (kg)					
Utility Worker	--	--	70	Per EPA Guidance	RAGS Suppl.
Duration of Exposure (years)					
Utility Worker	1 - 30	15	25	Per EPA Guidance	RAGS Suppl.
Exposure Frequency (days/year)					
Utility Worker	1 - 365	183	10	Assumes maintenance of buried utilities is necessary 10 days/yr.	
Skin Surface Area Contacted (sq. cm)					
Arms	--	--	2300	Per EPA Guidance	RAGS
Hands	--	--	820		
Total Area of These Limbs	--	--	3120		
Soil Skin Adherence Factor (mg/sq. cm)	0.2 - 1.0	0.6	0.6	Per EPA Region II guidance.	
Absorption Factor (percent)					
Cadmium	0.1-1	0.5	0.5	Per EPA Guidance	EPA, Reg. II
Averaging Time (days)				Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS
noncarcinogenic	--		9125		
carcinogenic	--		25550		

*Groups A, B, and C (see Section 2).

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 Reg. II: Personal communication from Peter Grevatt, EPA Region II Risk Assessment, February 3, 1992.

TABLE 4-7. EXPOSURE PATHWAY: INCIDENTAL INGESTION OF STORM DRAIN SEDIMENTS BY UTILITY WORKERS FOR PRESENT AND FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Utility Workers	
Body Weight (kg) Utility Worker	--	--	70	Per EPA Guidance	RAGS Suppl.
Duration of Exposure (years) Utility Worker	1 - 30	15	25	Per EPA Guidance	RAGS Suppl.
Exposure Frequency (days/year) Utility Worker	1 - 365	183	2	Assumes maintenance of storm drains is necessary 2 days/yr.	
Ingestion Rate (mg/day) Utility Worker	--	--	100	Value used is specified for adults	RAGS Suppl.
Fraction Ingested from Contaminated Source (unitless)	--	--	1	Assumes that all sediment contacted is contaminated	RAGS
Averaging Time (days) noncarcinogenic carcinogenic	-- --	-- --	9125 25550	Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS

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

TABLE 4-8. EXPOSURE PATHWAY: DERMAL CONTACT WITH STORM DRAIN SEDIMENTS BY UTILITY WORKERS FOR PRESENT AND FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Utility Worker	
Body Weight (kg)					
Utility Worker	--	--	70	Per EPA Guidance	RAGS Suppl.
Duration of Exposure (years)					
Utility Worker	1 - 30	15	25	Per EPA Guidance	
Exposure Frequency (days/year)					
Utility Worker	1 - 365	183	2	Assumes maintenance of storm drains is necessary 2 days/yr.	
Skin Surface Area Contacted (sq. cm)					
Arms	--	--	2300	Per EPA Guidance	RAGS
Hands	--	--	820		
Total Area of These Limbs	--	--	3120		
Soil Skin Adherence Factor (mg/sq. cm)	0.2 - 1.0	0.6	0.6	Per EPA Region II guidance.	
Absorption Factor (percent)					
Cadmium	0.1-1	0.5	0.5	Per EPA Guidance	EPA, Reg. II
Averaging Time (days)				Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS
noncarcinogenic	--		9125		
carcinogenic	--		25550		

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.

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.

TABLE 4-9. EXPOSURE PATHWAY: DERMAL CONTACT WITH STORM DRAIN WATER BY UTILITY WORKER FOR PRESENT AND FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Utility Worker	
Body Weight (kg)					
Utility Worker	--	--	70	Per EPA Guidance	RAGS Suppl.
Exposure Time (hours/day)	--	--	4	Assumes contact for half of working day	
Duration of Exposure (years)					
Utility Worker	1-30	15	25	Per EPA Guidance	RAGS Suppl.
Exposure Frequency (days/year)					
Utility Worker	1-365	183	2	Assumes maintenance of storm drains is necessary 2 days/yr.	
Skin Surface Area Contacted (sq. cm)					
Arms	--	--	2300	Per EPA Guidance	RAGS
Hands	--	--	820		
Total Area of These Limbs	--	--	3120		
Dermal Permeability Constants (cm/hour)					
Cadmium			0.00084	Values used is for water	RAGS
Averaging Time (days)					
noncarcinogenic	--	--	9125	Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS
carcinogenic	--	--	25550		

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

5.0 TOXICITY AND DOSE - RESPONSE ASSESSMENT

5.1 Introduction

This section presents information that relates chemical exposure (dose) to anticipated health effects (response) 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.

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

- IRIS (EPA, 1992b);
- Health Effects Assessment Summary Tables (HEAST) (EPA, 1991b);
- Direct Communication with EPA's Environmental Criteria and Assessment Office (ECAO) (EPA, 1992c; EPA, 1992d; EPA, 1992e; EPA, 1992f; EPA, 1992g);
- EPA Criteria Documents; and
- Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles.

A summary of the relevant toxicity values 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 chronic oral reference doses (RfDs), subchronic oral RfDs, and

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

Chemical	CARCINOGENIC Oral Slope Factor (mg/kg/day)-1	CHRONIC Chronic Oral RfD (mg/kg/day)	SUBCHRONIC Subchronic Oral RfD (mg/kg/day)	ACUTE Acute Oral "RfD" [1-Day HA/10] (mg/kg/day)
Volatiles				
Acetone		1.00E-01 a	1.00E+00 b	
Chloromethane (methyl chloride)	1.30E-02 b			9.00E-01 c
1,1-Dichloroethane		1.00E-01 b	1.00E+00 b	
1,1-Dichloroethylene	6.00E-01 a	9.00E-03 a	9.00E-03 b	2.00E-01 c
1,2-Dichloroethylene (total)		1.00E-02 k	1.00E-01 k	4.00E-01 k
Ethylbenzene		1.00E-01 a	1.00E+00 b	3.20E+00 a
Methylene chloride	7.50E-03 a	6.00E-02 a	6.00E-02 b	1.33E+00 a
Styrene	3.00E-02 b	2.00E-01 a	2.00E+00 b	2.00E+00 a
1,1,2,2-Tetrachloroethane	2.00E-01 a			
Tetrachloroethylene	5.10E-02 b	1.00E-02 a	1.00E-01 b	2.00E-01 a
Toluene		2.00E-01 b	2.00E+00 b	2.00E+00 c
1,1,1-Trichloroethane		9.00E-02 b	9.00E-01 b	1.00E+01 a
Trichloroethylene	1.10E-02 b	6.00E-03 d	6.00E-03 j	
Trichlorotrifluoroethane (Freon-113)		3.00E+01 b	3.00E+00 b	
Vinyl chloride (chloroethylene)	1.90E+00 b			3.00E-01 c
Xylenes		2.00E+00 a	4.00E+00 b	4.00E+00 c
Semivolatiles				
Acenaphthene		6.00E-02 a	6.00E-01 b	
Bis(2-ethylhexyl)phthalate	1.40E-02 a	2.00E-02 a	2.00E-02 b	
Chrysene	5.79E+00 e			
Dibenzofuran		4.00E-03 d	4.00E-03 j	
Di-n-butyl phthalate		1.00E-01 a	1.00E+00 b	
Dimethylphthalate		1.00E+00 b	1.00E+00 b	
Fluoranthene		4.00E-02 a	4.00E-01 b	
Fluorene		4.00E-02 a	4.00E-01 b	
Indeno(1,2,3-cd)pyrene	5.79E+00 e			
2-Methylnaphthalene				
Naphthalene		4.00E-03 b	4.00E-02 b	5.00E-02 c
3-Nitroaniline	4.00E-02 d	3.00E-04 d	3.00E-04 j	
Phenanthrene				
Pyrene		3.00E-02 a	3.00E-01 b	
Inorganics				
Aluminum		1.00E+00 d	1.00E+00 j	
Antimony		4.00E-04 a	4.00E-04 b	1.50E-03 c
Arsenic	1.75E+00 f	3.00E-04 a	1.00E-03 b	
Barium		5.00E-02 b	5.00E-02 b	
Beryllium	4.30E+00 a	5.00E-03 a	5.00E-03 b	3.00E+00 c
Cadmium (l)		5.00E-04 a,g	5.00E-04 j	4.00E-03 c
Chromium, total		8.76E-01 i	8.75E+00 i	1.40E-01 a
* Chromium, III		1.00E+00 a	1.00E+01 b	
Chromium, VI		5.00E-03 a	2.00E-02 b	
Cobalt		d		
Copper		4.00E-02 d	4.00E-02 j	
Cyanide		2.00E-02 a	2.00E-02 b	2.00E-02 a
Iron		5.00E-01 d	5.00E-01 j	
Lead				
Manganese		1.00E-01 a	1.00E-01 b	
Mercury		3.00E-04 b	3.00E-04 b	
Nickel		2.00E-02 a,h	2.00E-02 b	1.00E-01 c
Selenium		5.00E-03 a	5.00E-03 j	
Silver		5.00E-03 a	3.00E-03 b	2.00E-02 c

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

Chemical	CARCINOGENIC Oral Slope Factor (mg/kg/day)-1	CHRONIC Chronic Oral RfD (mg/kg/day)	SUBCHRONIC Subchronic Oral RfD (mg/kg/day)	ACUTE Acute Oral "RfD" [1-Day HA/10] (mg/kg/day)
Thallium		7.00E-05 b	7.00E-04 b	7.00E-04 c
Vanadium		7.00E-03 b	7.00E-03 b	8.00E-03 c
Zinc		2.00E-01 b	2.00E-01 b	4.00E-01 c

* Not analyzed for, used in derivation of Total Chromium toxicity values.
a. From Integrated Risk Information System (IRIS) 4/01/92.
b. From Health Effects Assessment Summary Tables (HEAST) FY 1991.
c. From Drinking Water Regulations and Health Advisories, November 1991.
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.
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 hexavalent chromium and trivalent 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 0.10 (10%) per EPA guidance (see text for specific reference). The dermal values are:
Chronic Dermal RfD: 5.00E-05 mg/kg/day
Subchronic Dermal RfD: 5.00E-05 mg/kg/day

acute protective body doses derived from one-day health advisories used to evaluate noncarcinogenic risks. Interim toxicity values obtained from ECAO (EPA, 1992c; EPA, 1992d; EPA, 1992e; EDPA, 1992f; EPA, 1992g) are also included in this table for contaminants where values were not available in IRIS or HEAST.

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

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. 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 EPA weight of evidence classification, the type of cancer and species from which the slope factors were derived, and the reference source of the slope factor. 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 TRONIC SITE COCS

Chemical	EPA Weight of Evidence Classification	Type of Cancer	Species	Source of Data
Volatiles				
Acetone	D	Oral: lack of data in animals and humans		IRIS (4/1/92)
Chloromethane	C	Oral: kidney tumors	mouse	HEAST (1991)
1,1-Dichloroethane	C	Oral: hemangiosarcomas/mammary carcinomas/liver carcinoma.	mouse	IRIS (4/1/92)
				IRIS (4/1/92)
1,1-Dichloroethylene	C	Oral: inadequate animal data		IRIS (4/1/92)
1,2-Dichloroethylene (total)	D(cis)	Oral: cis: lack of data in animals and humans trans: lack of data in animals and humans		IRIS (4/1/92)
Ethylbenzene	D	Oral: lack of data in animals and humans		IRIS (4/1/92)
Freon-113	--			
Methylene Chloride	B2	Oral: hepatocellular adenomas or carcinomas and hepatocellular cancer and neoplastic nodules	mouse	IRIS (4/1/92)
			mouse	IRIS (4/1/92)
Styrene	B2	Oral: lung and bronchi tumors	mouse	HEAST (1991)
1,1,2,2-Tetrachloroethane	C	Oral: hepatocellular carcinoma	rat	IRIS (4/1/92)

TABLE 5-2 (CONTINUED)

Chemical	EPA Weight of Evidence Classification	Type of Cancer	Species	Source of Data
Tetrachloroethylene	B2	Oral: liver tumors	mouse	HEAST (1991)
Toluene	D	Oral: no human data/inadequate animal data		IRIS (4/1/92)
1,1,1-Trichloroethane	D	Oral: no human data/inadequate animal data		IRIS (4/1/92)
Trichloroethylene	B2	Oral: liver tumors	mouse	HEAST (1991)
Vinyl chloride	A	Oral: lung tumors	rat	HEAST (1991)
Xylenes (total)	D	Oral: no human data/inadequate animal data		IRIS (4/1/92)
Base Neutral/Acid Extractable (Semivolatiles)				
Acenaphthene	--	Oral: lack of data in animals and humans		IRIS (4/1/92)
Bis(2-ethylhexyl)phthalate	B2	Oral: liver tumors	rat	IRIS (4/1/92)
Chrysene	B2	Oral: carcinoma/malignant lymphoma	mouse	IRIS (4/1/92)
Dibenzofuran	D	Oral: lack of data in animals and humans		IRIS (4/1/92)
Dimethyl phthalate	D	Oral: lack of data in animals and humans		IRIS (4/1/92)
di-n-Butyl phthalate	D	Oral: lack of data in animals and humans		IRIS (4/1/92)
Fluoranthene	D	Oral: no human data/inadequate animal data		IRIS (4/1/92)
Fluorene	D	Oral: no human data/inadequate animal data		IRIS (4/1/92)
Indeno(1,2,3-cd)pyrene	B2	Oral: lung epidermoid carcinomas	rat	IRIS (4/1/92)
2-Methylnaphthalene	--			

TABLE 5-2 (CONTINUED)

Chemical	EPA Weight of Evidence Classification	Type of Cancer	Species	Source of Data
Naphthalene	D	Oral: no human data/inadequate animal data		IRIS (4/1/92)
3-Nitroaniline	C	Oral: vascular liver tumors	mouse	EPA, 1992f
Phenanthrene	D	Oral: no human data/inadequate animal data		IRIS (4/1/92)
Pyrene	D	Oral: no human data/inadequate animals data		IRIS (4/1/92)
Inorganics				
Aluminum	D	Oral: lack of data in animals and humans		---
Antimony	--	Oral: lack of data in animals and humans		IRIS (4/1/92)
Arsenic	A	Oral: skin cancer	human	IRIS (4/1/92)
Barium	--	Oral: lack of data in animals and humans		IRIS (4/1/92)
Beryllium	B2	Oral: osteosarcomas	rabbit	IRIS (4/1/92)
Cadmium	B1	Oral: no human data/inadequate animal data	rat	IRIS (4/1/92)
Chromium, hexavalent	A	Oral: lack of data in animals and humans		IRIS (4/1/92)
Chromium, total	--	Oral: Chromium III: lack of data in animals and humans [also see chromium, hexavalent]		IRIS (4/1/92)
Cobalt	--	Oral: lack of data in animals and humans		---
Copper	D	Oral: no human data/inadequate animal data		IRIS (4/1/92)

TABLE 5-2 (CONTINUED)

Chemical	EPA Weight of Evidence Classification	Type of Cancer	Species	Source of Data
Cyanide, total	D	Oral: lack of data in animals and humans		IRIS (4/1/92)
Iron	D	Oral: lack of data in animals and humans		---
Lead	B2	Oral: bilateral renal carcinoma	rat	IRIS (4/1/92)
Manganese	D	Oral: inadequate data in animals and humans		IRIS (4/1/92)
Mercury	D	Oral: no human data/inadequate animal data		IRIS (4/1/92)
Nickel	A (refinery dust)	Oral: lack of data in animals and humans		IRIS (4/1/92)
Selenium	D	Oral: inadequate data in animals and humans		IRIS (4/1/92)
Silver	D	Oral: questionable animal data		IRIS (4/1/92)
Thallium	--			
Vanadium	D	Oral: lack of data in animals and humans		---
Zinc	D	Oral: inadequate human and animal data		IRIS (4/1/92)

NA Not Available
-- Unclassified by EPA as to carcinogenicity
--- No data source available
IRIS: Integrated Risk Information System on-line database. April 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

Two carcinogenic PAHs (cPAHs), chrysene and indeno(1,2,3-cd)pyrene, were detected at the Tronic site. Both of the cPAHs are classified as B2 carcinogens. Per EPA guidance, the oral slope factor for benzo(a)pyrene (B(a)P) was applied to these compounds. The oral slope factor available on IRIS of 5.79 $(mg/kg/day)^{-1}$ was used.

The assumption that these cPAHs have the same slope factor as B(a)P may result in an overestimation of risk. B(a)P is considered to be one of the most potent PAHs, and chrysene and indeno(1,2,3-cd)pyrene may not be as potent. However, application of the B(a)P oral slope factor to these compounds is the more conservative approach.

5.3 Noncarcinogenic Effects

5.3.1 General Method

Noncarcinogenic human health risks were evaluated by analyzing long-term exposures (chronic), short-term exposures (subchronic), and single event exposures (acute) to COCs. To evaluate long-term exposures, chronic RfDs were used. A chronic RfD 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.

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 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 5-5. Included in the table is the COC, the confidence level of the study used to determine the chronic RfD, a description of the critical physiological effect and the species of animal used in the study, the uncertainty/modifying factors, and the

TABLE 5-5. POTENTIAL CHRONIC NONCARCINOGENIC EFFECTS OF TRONIC SITE COCS

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
Volatiles					
Acetone	low	Oral: increased liver/kidney weight;nephrotoxicity	rat	UF: 1000 MF: 1	IRIS (4/1/92)
Chloromethane	NA	Oral: NA		UF: --	HEAST (1991)
1,1-Dichloroethane	**	Oral: no adverse effects observed	rat	UF: 1000	IRIS (4/1/92), HEAST (1991)
1,1-Dichloroethylene	medium	Oral: hepatic lesions	rat	UF: 1000 MF: 1	IRIS (4/1/92)
1,2-Dichloroethylene (total)	cis: **	Oral: decreased hematocrit and hemoglobin	rat	UF: 3000	IRIS (4/1/92), HEAST (1991)
	trans: low	Oral: increased serum alkaline phosphatase	mouse	UF: 1000 MF: 1	IRIS (4/1/92)
Ethylbenzene	low	Oral: liver and kidney toxicity	rat	UF: 1000 MF: 1	IRIS (4/1/92)
Freon-113	NA	Oral: decreased body weight	rat	UF: 100	HEAST (1991)
Methylene Chloride	NA	Oral: liver toxicity	rat	UF: 100	HEAST (1991)
Styrene	medium	Oral: red blood cell and liver effects	dog	UF: 1000 MF: 1	IRIS (4/1/92)
1,1,2,2-Tetrachloroethane	**	Oral: NA		UF: -- MF: --	IRIS (4/1/92)

TABLE 5-5 (CONTINUED)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
Tetrachloroethylene	medium	Oral: hepatotoxicity, weight gain	rodent	UF: 1000 MF: 1	IRIS (4/1/92)
Toluene	NA	Oral: changes in liver and kidney weight	rat	UF: 1000	IRIS (4/1/92), HEAST (1991)
1,1,1-Trichloroethane	NA	Oral: hepatotoxicity	guinea pig	UF: 1000	IRIS (4/1/92), HEAST (1991)
Trichloroethylene	**	Oral: increased relative liver weight	mouse	UF: 3000 MF: --	EPA, 1992e
Vinyl Chloride	NA	Oral: NA		UF: -- MF: --	---
Xylenes (total)	medium	Oral: hyperactivity, decreased body weight and increased mortality	rat	UF: 100 MF: 1	IRIS (4/1/92)
Base Neutral/Acid Extractables (Semivolatiles)					
Acenaphthene	low	Oral: hepatotoxicity	mouse	UF: 3000 MF: 1	IRIS (4/1/92)
Bis(2-ethylhexyl)phthalate	medium	Oral: increased relative liver weight	guinea pig	UF: 1000 MF: 1	IRIS (4/1/92)
Chrysene	NA	Oral: NA		UF: -- MF: --	IRIS (4/1/92)
Dibenzofuran	NA	Oral: decreased organ weight and body length; kidney abnorm.		UF: 10,000 MF: 1	EPA, 1992c

TABLE 5-5 (CONTINUED)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
Dimethyl phthalate	NA	Oral: minor effect on growth; some nephritic involvement	rat	UF: 100	IRIS (4/1/92), HEAST (1991)
di-n-Butyl phthalate	low	Oral: increased mortality	rat	UF: 1000 MF: 1	IRIS (4/1/92)
Fluoranthene	low	Oral: nephropathy, increased liver weights, hematological alternations, clinical effects	mouse	UF: 3000 MF: 1	IRIS (4/1/92)
Fluorene	low	Oral: decreased RBC, packed cell volume and hemoglobin	mouse	UF: 3000 MF: 1	IRIS (4/1/92)
Indeno(1,2,3-cd)pyrene	NA	Oral: NA		UF: -- MF: --	IRIS (4/1/92)
2-Methylnaphthalene	NA	Oral:		UF: MF:	
Naphthalene	NA	Oral: decreased body weight gain	rat	UF: 10,000	IRIS (4/1/92), HEAST (1991)
3-Nitroaniline	NA	Oral:		UF: MF:	EPA, 1992f
Phenanthrene	NA	Oral: NA		UF: -- MF: --	IRIS (4/1/92)
Pyrene	low	Oral: kidney effects	mouse	UF: 3000 MF: 1	IRIS (4/1/92)
Inorganics					
Aluminum	medium	Oral: decreased body weight gain; neurotoxicity		UF: 100	EPA, 1992g

TABLE 5-5 (CONTINUED)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
Antimony	low	Oral: longevity, blood glucose, cholesterol	rat	UF: 1000 MF: 1	IRIS (4/1/92)
Arsenic	medium	Oral: hyperpigmentation, keratosis, and possible vascular complications	human	UF: 3 MF: 1	IRIS (4/1/92)
Barium	medium	Oral: increased blood pressure	human	UF: 3 MF: 1	IRIS (4/1/92)
Beryllium	low	Oral: no adverse effects	rat	UF: 100 MF: 1	IRIS (4/1/92)
Cadmium	high	Oral: significant proteinuria	human	UF: 10 MF: 1	IRIS (4/1/92)
Chromium, hexavalent	low	Oral: no effects reported	rat	UF: 500 MF: 1	IRIS (4/1/92)
Chromium, total	low	Oral: Chromium III: no adverse effects observed [also see chromium, hexavalent]	rat	UF: 100 MF: 1	IRIS (4/1/92)
Cobalt	NA	Oral: cardiomyopathy	human	UF: -- MF: --	EPA, 1992d
Copper	NA	Oral: local GI irritation	human	UF: NA	EPA, 1992c
Cyanide, total	medium	Oral: weight loss, thyroid effects and myelin degeneration	rat	UF: 100 MF: 5	IRIS (4/1/92)

TABLE 5-5 (CONTINUED)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
Iron	NA	Oral: liver cirrhosis		UF: -- MF: --	EPA, 1992c
Lead	NA	Oral: CNS effects		UF: --	IRIS (4/1/92), HEAST (1991)
Manganese	medium	Oral: CNS effects	human	UF: 1 MF: 1	IRIS (4/1/92),
Mercury	**	Oral: kidney effects	rat	UF: 1000	IRIS (4/1/92), HEAST (1991)
Nickel (data for soluble salts)	medium	Oral: decreased body and organ weights	rat	UF: 300 MF: 1	IRIS (4/1/92)
Selenium	high	Oral: clinical selenosis	human	UF: 3 MF: 1	IRIS (4/1/92)
Silver	low	Oral: argyria	human	UF: 3 MF: 1	IRIS (4/1/92)
Thallium	NA	Oral: increased SGOT and serum LDH, alopecia	rat	UF: 3000 MF: --	HEAST (1991)
Vanadium	NA	Oral: no adverse effects observed	rat	UF: 100	HEAST (1991)
Zinc	**	Oral: anemia	human	UF: 10	IRIS (4/1/92), HEAST (1991)

**	Pending in IRIS	IRIS:	Integrated Risk Information System. April 1, 1991.
NA	Not Available	HEAST:	Health Effects Assessment Summary Tables, FY 1991.
--	Not Applicable	ECAO:	EPA, 1992c.
---	No data source available		

reference source of the study used to derive the chronic RfD. If the source of data is HEAST, or interim guidance from ECAO, the uncertainty factors provided are a multiplication of the uncertainty factor and modifying factor otherwise listed in IRIS. The confidence levels were provided by IRIS as either high, medium, or low.

Table 5-6 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, and the uncertainty factor. 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 a chronic oral RfD nor a subchronic oral RfD was available for a given COC, long-term and short-term oral exposures were not assessed quantitatively.

To evaluate single-event oral exposures, one-day Health Advisories (HA) (EPA, 1991c) were used (see Table 5-1). The one-day HA represents the concentration in drinking water which protects against toxic effects from a single exposure event for a 10 kg child. In order to utilize the one-day HA for the acute risk analysis, the body dose resulting from ingestion of water containing the concentration of the chemical indicated in the HA must be derived. The HA was converted to the protective body dose using the following formula:

$$\text{Protective Dose (mg/kg/day)} = \frac{\text{1-day HA (mg/L) x 1L exposure/day}}{\text{10 kg}}$$

The resulting dose is referred to as the acute protective body dose.

TABLE 5-6. POTENTIAL SUBCHRONIC NONCARCINOGENIC EFFECTS OF TRONIC SITE COCS

Chemical	Critical Effect	Species	Uncert. Factor
Volatiles			
Acetone	Oral: increased liver/kidney weight; nephrotoxicity	rat	100
Chloromethane	Oral: --		
1,1-Dichloroethane	Oral: no adverse effects observed	rat	100
1,1-Dichloroethylene	Oral: liver lesions	rat	100
1,2-Dichloroethylene (total)	Oral: cis: decreased hematocrit and hemoglobin trans: increased serum alkaline phosphatase	rat rat	300 100
Ethylbenzene	Oral: hepatotoxicity and nephrotoxicity	rat	100
Freon-113	Oral: decreased body weight	rat	100
Methylene Chloride	Oral: liver toxicity	rat	100
Styrene	Oral: red blood cell and liver effects	dog	100
1,1,2,2-Tetrachloroethane	Oral: --		
Tetrachloroethylene	Oral: hepatotoxicity	mouse	100
Toluene	Oral: changes in liver and kidney weights	rat	100
1,1,1-Trichloroethane	Oral: hepatotoxicity	guinea pig	100

TABLE 5-6 (CONTINUED)

Chemical	Critical Effect	Species	Uncert. Factor
Trichloroethylene	Oral: --		
Vinyl Chloride	Oral: --		
Xylenes (total)	Oral: hyperactivity, decreased body weight and increased mortality	rat	100
Base Neutral/Acid Extractables (Semivolatiles)			
Acenaphthene	Oral: hepatotoxicity	mouse	300
Bis(2-ethylhexyl)phthalate	Oral: increased relative liver weight	guinea pig	1000
Chrysene	Oral: --		
Dibenzofuran	Oral: --		
Dimethyl phthalate	Oral: minor effect on growth; some nephritic involvement	rat	100
di-n-Butyl phthalate	Oral: mortality	rat	100
Fluoranthene	Oral: nephropathy; liver weight changes; hematological changes	mouse	300

TABLE 5-6 (CONTINUED)

Chemical	Critical Effect	Species	Uncert. Factor
Fluorene	Oral: hematological changes (decreased RBC)	mouse	300
Indeno(1,2,3-cd)pyrene	Oral: --		
2-Methylnaphthalene	Oral: --		
Naphthalene	Oral: decreased body weight gain	rat	1000
3-Nitroaniline	Oral: --		
Phenanthrene	Oral: --		
Pyrene	Oral: renal effects	mouse	300
Inorganics			
Aluminum	Oral: --		
Antimony	Oral: reduced lifespan, altered blood chemistries	rat	1000
Arsenic	Oral: keratosis and hyperpigmentation	human	1
Barium	Oral: increased blood pressure	rat	100
Beryllium	Oral: no adverse effects observed	rat	100
Cadmium	Oral: --	NA	NA

TABLE 5-6 (CONTINUED)

Chemical	Critical Effect	Species	Uncert. Factor
Chromium, hexavalent	Oral: not defined	rat	100
Chromium, total	Oral: Chromium III: hepatotoxicity (also see Chromium, hexavalent)	rat	100
Cobalt	Oral: --		
Copper	Oral: local GI irritation	human	NA
Cyanide, total	Oral: weight loss, thyroid effects and myelin degeneration	rat	500
Iron	Oral: --		
Lead	Oral: --		
Manganese	Oral: no adverse effects observed	human	1
Mercury	Oral: kidney effects	rat	1000
Nickel	Oral: reduced body and organ weight	rat	300
Selenium	Oral: --		

TABLE 5-6 (CONTINUED)			
Chemical	**Critical Effect**	**Species**	**Uncert. Factor**
Silver	Oral: argyria	human	2
Thallium	Oral: increased SGOT and serum LDH, alopecia	rat	300
Vanadium	Oral: no adverse effects observed	rat	100
Zinc	Oral: anemia	human	10

Note: All subchronic information is from the *Health Effects Assessment Summary Tables*, FY 1991.
NA Not Available
-- No data or "data inadequate for quantitative risk assessment."

5.3.2 Chemical-Specific Considerations

PAHs

Noncarcinogenic PAHs were detected in storm drain sediments. Toxicity values exist for all noncarcinogenic PAH COCs except 2-methylnaphthalene and phenanthrene. The lack of toxicological data precludes the derivation of a systemic toxicity value. Per EPA guidance, surrogate RfDs for 2-methylnaphthalene and phenanthrene were not used due to the uncertainties associated with assuming noncarcinogenic PAHs cause similar effects (EPA, 1991d).

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.

Per EPA Region II guidance (EPA, 1992a), 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

Chromium

Total chromium was detected in most media sampled at the Tronic site. Per EPA Region II guidance, total chromium was assumed to consist of seven parts trivalent

chromium and one part hexavalent chromium (EPA, 1992a). In order to assess the risk presented by total chromium at the site, a weighted average of the toxicity values for trivalent and hexavalent chromium was calculated based on the assumed ratio of the species in total chromium. For example, the chronic oral RfDs for the chromium species are:

trivalent: 1×10^{0} mg/kg/day
hexavalent: 5×10^{-3} mg/kg/day (see Table 5-1)

A weighted-average chronic oral RfD was calculated as follows:

1×10^{0} mg/kg/day x 7 parts trivalent

$= 7 \times 10^{0}$ mg/kg/day

5×10^{-3} mg/kg/day x 1 part hexavalent

$= 5 \times 10^{-3}$ mg/kg/day

7×10^{0} mg/kg/day + 5×10^{-3} mg/kg/day = 7.005×10^{0} mg/kg/day

$$\frac{7.005 \times 10^{0} \text{ mg/kg/day}}{8 \text{ parts total (7tri + 1hex)}}$$

$= 8.76 \times 10^{-1}$ mg/kg/day

The weighted-average toxicity value (RfD) for total chromium was applied in oral risk calculations as discussed in Section 5.3.1.

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 to assist in evaluating whether remedial action at the Tronic site is necessary.

The present and potential future public health risks attributable to the Tronic 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 were used to represent exposure.

Exposure and risk calculation worksheets are presented in Appendix D 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 Tronic 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 herein 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 (from Table 5-1). The dose represents the amount of contaminant to which a receptor is exposed, as described in Section 4.0. For present scenarios, the dose is the estimated daily intake averaged over a 70-year lifetime for a utility worker (25 years exposure). The future scenarios for a general worker/utility worker and an excavation worker also estimate a daily intake averaged over a 70-year lifetime, with 25 years exposure for a general worker/utility worker and 3 months exposure for an excavation worker.

Incremental CA risk was calculated for each COC and exposure pathway. 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. In order to conservatively reflect EPA's action levels, any carcinogenic risk values exceeding 1×10^{-6} are considered to be of potential concern in this report.

6.2.2 Noncarcinogenic Effects

Potential noncarcinogenic effects were evaluated based on a comparison of chemical-specific acute, subchronic, and chronic exposure doses with corresponding protective doses derived from health criteria, as described in Section 5.0. Acute protective doses were based on one-day HAs and subchronic and chronic protective doses were based on the RfD. 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 acute, 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. Separate calculations were performed for acute, subchronic, and chronic effects. The HQs for each contaminant were then summed to produce a rough estimate of the exposure pathway-specific risk, the Hazard Index (HI). In estimating total noncarcinogenic risk, response additivity was assumed. However, all COCs do not have the same or similar toxic endpoints. Therefore, in those cases where the HI exceeded one, further analyses were undertaken to evaluate which specific chemicals might exhibit toxic effects.

Summation across age groups is not appropriate for the noncarcinogenic evaluation because the estimated doses for the noncarcinogens are based on less than lifetime exposures and do not reflect an average lifetime dose.

6.3 Risk Summary

An overall summary of Tronic 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. Chemical-specific risk values are presented in Appendix D.

In accordance with current EPA Region II guidance (EPA, 1992a) the quantitative risk assessment for dermal contact exposures was limited. 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 Tronic 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 to contaminants for which no toxicity values are currently available. Lead is evaluated in relation to a OSWER Directive (EPA, 1989d) on soil cleanup levels and the drinking water action level; cobalt is evaluated in relation to dietary intake levels.

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

Scenario	Receptor	Present/Future	Incremental Risk
Ground Water			
Ingestion	General Worker	F	8.7×10^{-5}*
Subsurface Soil			
Group A			
Ingestion	Excavation Worker	F	5.1×10^{-8}
Ingestion	Utility Worker	P/F	4.1×10^{-8}
Group B			
Ingestion	Excavation Worker	F	5.0×10^{-8}
Ingestion	Utility Worker	P/F	4.0×10^{-8}
Group C			
Ingestion	Excavation Worker	F	2.3×10^{-7}
Ingestion	Utility Worker	P/F	1.9×10^{-7}
Storm Drain Sediments			
Ingestion	Utility Worker	P/F	9.8×10^{-8}

*Exceeds 10^{-6} risk

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

Scenario	Receptor	Present/Future	Acute HI	Chronic HI
Ground Water				
Ingestion	General Worker	F	3.9×10^{-1}	2.4×10^{0}*
Subsurface Soil				
Group A				
Ingestion	Excavation Worker	F	4.2×10^{-2}	1.9×10^{-1}a
Dermal Contact**	Excavation Worker	F	--	2.5×10^{-3}a
Ingestion	Utility Worker	P/F	8.8×10^{-3}	1.7×10^{-3}
Dermal Contact**	Utility Worker	P/F	--	9.5×10^{-5}
Group B				
Ingestion	Excavation Worker	F	4.1×10^{-2}	1.8×10^{-1}
Dermal Contact**	Excavation Worker	F	--	4.0×10^{-3}
Ingestion	Utility Worker	P/F	8.6×10^{-3}	1.7×10^{-3}
Dermal Contact**	Utility Worker	P/F	--	1.5×10^{-4}
Group C				
Ingestion	Excavation Worker	F	1.8×10^{-1}	7.0×10^{-1}
Dermal Contact**	Excavation Worker	F	--	1.2×10^{-1}
Ingestion	Utility Worker	P/F	3.8×10^{-2}	5.9×10^{-3}
Dermal Contact**	Utility Worker	P/F	--	4.5×10^{-3}
Storm Drain Sediments				
Ingestion	Utility Worker	P/F	4.4×10^{-1}	1.1×10^{-2}
Dermal Contact**	Utility Worker	P/F	--	1.7×10^{-2}
Storm Drain Water				
Dermal Contact**	Utility Worker	P/F	--	1.4×10^{-1}

*Hazard Index exceeds one (1).
**Pathway evaluated for cadmium only, per EPA guidance.
a - Subchronic HIs were calculated for this scenario.

The greatest carcinogenic risk value is associated with the highly conservative ground water ingestion scenario (8.7×10^{-5}). No other exposure scenarios produced carcinogenic risk in excess of 10^{-6}. Noncarcinogenic HIs, summed across chemicals for each exposure route, exceeded one only for ground water ingestion.

6.3.1 Ground Water

Carcinogenic Risks

Carcinogenic risks attributable to ground water ingestion fell within the NCP acceptable risk range of 10^{-6} to 10^{-4}. Five contaminants showed risks that exceeded 10^{-6}: 1,1-DCE, PCE, TCE, arsenic, and beryllium. The maximum concentrations of these contaminants were detected in wells at different areas of the site. The highest concentrations of TCE (490 µg/l) and beryllium (5.7 µg/l) were detected at location MW1. This area was thought to be upgradient of the site; however, samples were included in the risk assessment because they showed contamination with organic compounds. Arsenic is a Class A carcinogen; PCE, TCE, and beryllium are Class B2 carcinogens; 1,1-DCE is a Class C carcinogen. PCE (21/24) and TCE (23/24) were detected frequently. 1,1-DCE (4/24), arsenic (3/24), and beryllium (1/24) were detected relatively infrequently.

Noncarcinogenic Risks

Only the chronic HI exceeded one. Although none of the chemical-specific HQs exceeded one, the risk was driven by TCE (HQ = 4.7×10^{-1}), antimony (HQ = 6.7×10^{-1}) and thallium (HQ = 3.9×10^{-1}). Five other inorganics also contributed to the risk including aluminum, arsenic, cadmium, chromium VI, and manganese. As mentioned above, the maximum concentration of TCE (490 µg/l) was detected in an area thought to be upgradient of the site. The one detected concentration (from 24 samples) of thallium (5.6 µg/l) was also from this area as was the maximum

concentration of manganese (3180 µg/l). Antimony, arsenic, and cadmium were detected relatively infrequently (2/24, 3/24 and 1/24, respectively); however, aluminum (20/24) and chromium VI (13/14) were detected frequently throughout the site. Of the contaminants contributing to noncarcinogenic risk, only cadmium has an RfD with a high EPA level of confidence. Aluminum, arsenic, and manganese have RfDs with medium EPA levels of confidence, while TCE, antimony, and chromium VI have low EPA levels of confidence. Thallium has no EPA level of confidence. The RfD for TCE is an interim value provided by ECAO (EPA, 1992e).

Other Potential Ground Water Risks

Lead concentrations measured in well MW1 (75.5 µg/l), which is possibly upgradient of the site, exceeded the current ground water action level of 15 µg/l. The maximum dose calculated for cobalt (1.0×10^{-4} mg/kg/day) is well below the range of average daily intakes for children (0.01 - 0.06 mg/kg/clay) and adults (0.002 - 0.008 mg/kg/day) (EPA, 1992d).

6.3.2 Subsurface Soils

Carcinogenic Risks

Total carcinogenic risk for both the excavation and utility worker scenarios were below 10^{-6} for all three groups of subsurface soils and are therefore assumed to be insignificant.

Noncarcinogenic Risk

All HI values for subsurface soil scenarios were below one. This indicates that noncarcinogenic risks associated with subsurface soils are insignificant.

6.3.3 Storm Drain Sediments

Carcinogenic Risks

Incidental ingestion of sediments by a utility worker produced risks below 10^{-6}. Carcinogenic risks are therefore assumed to be insignificant.

Noncarcinogenic Risks

All sediment exposure scenarios resulted in noncarcinogenic HIs below one. Noncarcinogenic risks associated with sediments are therefore assumed to be insignificant.

Other Potential Risks

Lead concentrations in sediments at two storm drain locations exceeded the OSWER target soil cleanup level of 500-1000 mg/kg. Concentrations measured at locations SD1 and SD2 were 1190 mg/kg and 2290 mg/kg, respectively.

Cobalt concentrations resulted in a maximum dose of 9.6×10^{-8} mg/kg/day, which is well below the range of average daily intakes for children (0.01 - 0.06 mg/kg/day) and adults (0.002 - 0.008 mg/kg/day) (EPA, 1992d).

6.3.4 Storm Drain Water

Dermal contact was the only exposure evaluated for storm drain water. Based on current EPA guidance, only cadmium was included in the quantitative assessment. The cadmium HQ was below one; risk is therefore assumed to be insignificant.

Lead concentrations in storm drain water were elevated in both samples analyzed. Concentrations of 138 µg/l (SD1) and 14,000 µg/l (SD2) exceeded the federal drinking water action level of 15 µg/l.

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 in the area of the Tronic 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 leads 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, RAGS 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 has the advantage of providing some standardization of approach across different risk assessments and 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 Region II guidance (EPA, 1992a) indicates 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). This approach leads to an underestimation of total medium-specific risk. However, 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 Tronic 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 Tronic 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.

Surface soils were not chemically characterized by the 1991 RI, so risks associated with this medium could not be assessed. These risks are predicted to be minimal given that past disposal did not involve discharge to surface soils or soils immediately beneath the pavement and because the remote location of detected contaminants in the subsurface renders them inaccessible during assumed surface exposure scenarios. Though exposures to surface soils are expected to be negligible, the lack of chemical data for this medium introduces a source of uncertainty to the risk assessment.

Certain media (e.g., storm drain sediments) were characterized with very few 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 very conservative methodology. A strong justification was needed for excluding contaminants from the risk assessment rather than including them. Therefore, it is highly unlikely that significant contributors to risk were excluded. For example, although thallium was retained as a COC and is one of the contaminants driving noncarcinogenic risk for ground water, it was only detected in one of twenty-four samples. In addition, the area in which thallium was detected may be upgradient of the site.

Certain COCs lacked toxicity values 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 action level for drinking water. Cobalt was evaluated in relation to dietary intake levels. For other COCs and/or pathways, groups of chemicals were evaluated together using the same toxicity values. As recommended by RAGS, PAHs evaluated for oral exposures were grouped in this manner. When available, toxicity values for the more toxic species of PAHs were used to evaluate other species. While this approach may be a source of uncertainty, it can only result in conservative risk estimates.

Other COCs were evaluated using interim toxicity values obtained from ECAO. The RfD for TCE is an interim value and may lead to additional uncertainty in the risk estimates.

Future land use and future use of 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 in the vicinity of the site will reach wells without attenuation. Future use of the site property was assumed to be residential commercial/light industrial. Continued commercial/industrial use is not likely to alter exposures from

those evaluated in this risk assessment. It assumed the site would be accessible to current facility workers, utility workers, and excavation workers.

Exposure parameters for the Tronic 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, limiting the quantitative risk assessment for dermal contact to cadmium only will underestimate risk from this pathway.

Finally, one of the purposes of this risk assessment is to characterize the incremental risk associated with the Tronic 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. However, the "background" ground water samples collected at the Tronic site were contaminated, indicating they may represent an as yet unidentified source of contamination. These samples could not be used in the risk assessment to compare site contamination with background levels.

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 risk (from Section 4) as well as central tendency exposure parameters obtained from EPA guidance and the Bureau of Labor Statistics. 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 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 D) results in the following changes in risk values:

- carcinogenic risk decreases by a factor of 8.5, 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" chemicals of concern. The total pathway carcinogenic risk and noncarcinogenic HI for all COCs is also listed.

TABLE 7-1. COMPARISON OF WORKER 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 Worker	70	Average	RAGS Suppl.	70	Average	RAGS Suppl.
Duration of Exposure (years)						
Adult Worker	25	95th Percentile	RAGS Suppl.	4.2	50th Percentile	Bureau of Labor Stat.
Exposure Frequency (days/year)	250	Average	RAGS Suppl.	250	Average	RAGS Suppl.
Ingestion Rate (l/day)						
Adult Worker	1	0.5 x 95th Percentile Residential	RAGS Suppl.	0.7	0.5 x Average Residential	RAGS Suppl.
Averaging Time (days)						
noncarcinogenic	9125	25 x 365	N/A	1533	4.2 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.
Bureau of Labor Statistics: News Release, U.S. Department of Labor, October 22, 1987.

TABLE 7-2. COMPARISON OF RME RISK AND CENTRAL TENDENCY RISK FOR WORKER GROUND WATER INGESTION		
	RME Risk[a]	**Central Tendency Risk[b]**
Total Carcinogenic Risk[c]	8.7×10^{-5}	1.0×10^{-5}
Driver Chemicals		
1,1-Dichloroethylene	7.6×10^{-6}	8.9×10^{-7}
Tetrachloroethylene	1.5×10^{-6}	1.8×10^{-7}
Trichloroethylene	1.1×10^{-5}	1.3×10^{-6}
Arsenic	2.9×10^{-5}	3.4×10^{-6}
Beryllium	3.8×10^{-5}	4.5×10^{-6}
Total Noncarcinogenic HI[c]	$2.4 \times 10^{+0}$	$1.7 \times 10^{+0}$
Driver Chemicals		
Trichloroethylene	4.7×10^{-1}	3.4×10^{-1}
Aluminum	2.1×10^{-1}	1.5×10^{-1}
Antimony	6.7×10^{-1}	4.8×10^{-1}
Arsenic	1.6×10^{-1}	1.1×10^{-1}
Cadmium	1.1×10^{-1}	7.9×10^{-2}
Chromium VI	1.3×10^{-1}	9.3×10^{-2}
Manganese	1.1×10^{-1}	7.9×10^{-2}
Thallium	3.9×10^{-1}	2.8×10^{-1}

[a] From Appendix D.
[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 habitats and species that have been noted or are expected to be present at or in the immediate vicinity of the Tronic site. No aquatic habitats are present at the site or in its immediate vicinity. An evaluation of the potential risks associated with the exposure of biota to contaminants detected during the RI is also included in this risk assessment. Quantitative measures to evaluate ecological risks (e.g., wildlife inventories, biota sampling, bioassays, and modelling) were not within the scope of this risk assessment, as agreed with EPA, nor were sufficient data available in the RI.

8.1.2 General Methodology

This analysis is divided into two parts. The first part (Habitat and Species Characterization) describes regional ecological characteristics including habitats and species expected to be present in the vicinity of the site. Regional descriptions are based on information gathered from state and local agencies. Site visits were not part of the scope of work for this risk assessment.

The second part of the ecological risk assessment forms a qualitative evaluation of potential risks to biota at and near the Tronic site based on reported levels of contamination. The discussion includes a brief summary of potential exposures and a risk characterization. It should be reiterated that the area in which the site is located has been extensively modified for industrial purposes. Due to the lack of potential habitats in the area, and because known contamination at the site is subsurficial, the potential exposure of biota to contaminants from the site is considered unlikely.

Because of this, certain sections of this ecological risk assessment have been abbreviated.

8.2 Habitat and Species Characterization

8.2.1 Onsite Habitats

The majority of the Tronic site is developed. With the exception of a 3,750 square foot cultivated lawn, the entire site is covered by structures and pavement. The only potential onsite habitat is the cultivated lawn. NYSDEC characterizes cultivated lawns as areas which are dominated by clipped grass and are covered by trees on less than 30 percent of their area (NYSDEC, 1990). Species expected to inhabit the cultivated lawn are listed in Table 8-1. No aquatic habitats are present at the site.

8.2.2 Offsite Habitats

The area in the vicinity of the Tronic site is dominated by light industrial property and residential "A" property, which is zoned for use as cemeteries only. No aquatic habitats are known to be present in the immediate vicinity of the site. The only significant terrestrial habitat in the vicinity of the site is a woodland located approximately 300 feet south of the former Tronic facility. The woodlands are part of the Pinelawn Cemetery and are currently being cleared for the construction of burial plots (Britts, 1992). According to the cemetery's supervisor, the woodlands will be completely cleared within the next ten or fifteen years. The woodland can best be characterized as a pitch pine-oak forest (Britts, 1992; NYSDEC, 1990). The cemetery consists primarily of cultivated lawns with scattered trees and shrubs. Species that may potentially utilize these habitats have been identified in Table 8-1.

TABLE 8-1. SPECIES EXPECTED TO INHABIT THE VICINITY OF THE TRONIC SITE

Common Name	Scientific Name
CULTIVATED LAWN	
Plants:	
Introduced grasses	*Graminue spp.*
Wildlife:	
American robin	*Turdus migratorius*
American crow	*Corvus brachyrhynchos*
Rock dove	*Columba livia*
House sparrow	*Passer domesticus*
European starling	*Sturnus vulgaris*
WOODLAND/CEMETERY	
Plants (Woodland Habitat):	
Pitch pine	*Pinus rigida*
Scarlet oak	*Quercus coccinea*
White oak	*Q. alba*
Red oak	*Q. rubra*
Black oak	*Q. veluntina*
Scrub oak	*Q. licifolia*
Lowbush blueberry	*Vaccinium pallidum, V. angustifolium*
Black huckleberry	*Gaylussacia baccata*
Bracken fern	*Pteridium aquilinum*
Wintergreen	*Gaultheria procumbens*
Pennsylvania sedge	*Carex pensylvania*
Wildlife:	
Rufous-sided towhee	*Pipilo erythrophthalmus*
Common yellowthroat	*Geothlypis trichas*
Field sparrow	*Spizella pusilla*
Prairie warbler	*Dendroica discolor*
Pine warbler	*D. pinus*
Blue jay	*Cyanocitta cristata*

TABLE 8-1. (CONTINUED)

Common Name	Scientific Name
Whip-poor-will	*Caprimulgus vociferus*
American robin	*Turdus migratorius*
Northern mockingbird	*Mimus polyglottos*
Northern cardinal	*Cardinalis cardinalis*
Black-capped chickadee	*Parus atricapillus*
Tufted titmouse	*P. bicolor*
Downy woodpecker	*Picoides pubescens*
European starling	*Sturnus vulgaris*
Mourning dove	*Zenaida macroura*
Raccoon	*Procyon lotor*
Short-tailed shrew	*Blarina brevicauda*
Gray squirrel	*Sciurus carolinensis*
Eastern cottontail	*Sylviliagus floridanus*
Eastern chipmunk	*Tamias striatus*
White-footed mouse	*Peromyseus leucopus*
Garter snake	*Thamnophis sirtalis sirtalis*
Brown snake	*Storeria dekayi*
American toad	*Bufo americanus*
Woodhouse's toad	*Bufo woodhousei*
Redbacked salamander	*Plethodon cinereus*
Red-spotted newt	*Notophthalmus viridescens*

Modified from NYSDEC, 1990 and Britts, 1992.

8.2.3 Regionally Significant Habitats

The National Wetlands Inventory has recorded six small wetland areas within one mile of the site. Five of these areas are classified as semi-permanent, palustrine open water wetlands, resulting from excavation (USFWS, 1981a; USFWS, 1981b). One wetland is classified as a temporary, flat palustrine area. Nineteen other wetland areas, all of which are described as temporary or semi-permanent palustrine wetlands created by excavations, are located within two miles of the site (USFWS, 1981a; USFWS, 1981b).

NYSDEC Significant Habitat Unit files indicate that one community of concern, a pitch pine-scrub oak barrens, is located within two miles of the site. The community is classified as Unprotected by the state, but is also described as being "imperiled globally because of rarity (few remaining acres) or very vulnerable to extinction throughout its range because of other factors" and "typically few remaining acres or some factor of its biology making it especially vulnerable in New York State" (Buffington, 1992). Rare and endangered species are discussed in the subsequent section.

8.2.4 Regional Species Profile

The information in this section regarding species expected to inhabit the vicinity of the site is compiled primarily from NYSDEC's Ecological Communities of New York State (NYSDEC, 1990). The description of species inhabiting the pitch pine-oak woodlands located south of the site was compiled from communications with the supervisor of Pinelawn Cemetery and from a description provided by Alliance field oversight personnel. Information concerning rare and endangered species was obtained from NYSDEC's Significant Habitat Unit and Natural Heritage Program. Species noted or expected to inhabit the vicinity of the site are listed in Table 8-1.

Plants

The cultivated lawn is dominated by numerous introduced grasses and herbs. The grass areas of the cemetery are also dominated by various grasses and herbs and are interspersed with species of ornamental trees and shrubs. The woodland portion of the cemetery is dominated by pitch pines and several species of oak (Britts, 1992). Pitch pine-oak forests typically support shrublayers dominated by scrub oak, lowbush blueberry, and black huckleberry (NYSDEC, 1990). The herbaceous layer in this type of forest is characteristically dominated by species such as the bracken fern, wintergreen, and Pennsylvania sedge (NYSDEC, 1990).

Birds

The species of birds that are expected to frequent the cultivated lawn are those that typically inhabit developed areas. Representative species include robins, starlings, crows, rock doves, and sparrows (NYSDEC, 1990). These species are expected to utilize the lawn as foraging habitat only. Because of the lawn's small size, frequent level of human disturbance, and lack of woody overstory vegetation, it is highly unlikely that avian species nest in the area.

Bird species known to frequent pitch pine-oak forests on Long Island include rufous-sided towhees, common yellowthroats, field sparrows, prairie warblers, pine warblers, blue jays, and whip-poor-wills (NYSDEC, 1990). These species would be expected to inhabit the wooded area of the cemetery. The remaining grass areas of the cemetery containing scattered trees and shrubs would provide suitable habitat for common bird species such as the American robin, northern mockingbird, and mourning dove (See Table 8-1).

Mammals

Because of the extensive development in the area of the Tronic site, the habitats in the vicinity of the site probably support species tolerant of highly developed areas or species with small home ranges. Squirrels, rabbits, and various small mammals inhabit the woodlands and the adjacent cemetery (Britts, 1992). It is unlikely that the Tronic site itself provides suitable habitat for mammalian species.

Amphibians/Reptiles

Several species of reptiles may inhabit the cemetery and woodland south of the site. The more common species expected to be present include the garter snake and brown-snake. Several species of amphibians, including salamanders and toads, may also utilize the pitch pine-oak woodland and cemetery.

Species of Concern

Information concerning endangered species was obtained from NYSDEC's Significant Habitat Unit and Natural Heritage Program data bases. The data bases record six occurrences of rare plants and one occurrence of a rare invertebrate within two miles of the Tronic site. Table 8-2 lists these species and includes their scientific name, state and federal status, and date last observed within two miles of the site. Information regarding the precise locations of these plant and invertebrate species is confidential and cannot be released to the public in order to protect these species from possible human disturbance.

One federally listed endangered species, sandplain gerardia, has been noted to occur within two miles of the site. Federally endangered species are considered to be critically imperiled globally with typically five or fewer individuals known to exist worldwide. They can also be characterized as being extremely vulnerable to extinction because of some factor of its biology (Buffington, 1992). Sandplain gerardia is also

TABLE 8-2. RARE AND ENDANGERED SPECIES AND COMMUNITIES IN THE VICINITY OF THE TRONIC SITE

Common Name	Scientific Name	State/Federal Status	Date Last Observed
Sandplain gerardia	*Agalinia acuta*	E/LE	1921
Coastal barrens buckmoth	*Hemieuca maia*	SC/-	1985
Collins sedge	*Carex collinsii*	R/-	1927
Pitch pine-scrub oak barrens	---	U/-	1985
Dwarf plantain	*Plantago pusilla*	U/-	1987
St. Andrew's Cross	*Hypericum hypericoides*	E/-	1987
Woodland agrimony	*Agrimonia rostellata*	R/-	1924
Tick-trefoil	*Desmodium ciliare*	T/-	1925

Modified from Buffington, 1992.
E - Endangered species
LE - Formally listed as endangered species
SC - Special concern species
R - Rare species
U - Unprotected within the state
T - Threatened species

listed as a state endangered species. State endangered species are considered to be in imminent danger of extinction in New York State or are listed as endangered by the U.S. Department of the Interior. Sandplain gerardia was last observed in the vicinity of the site in 1921 (Buffington, 1992).

St. Andrew's Cross is also listed as state endangered and was recently recorded as occurring within two miles of the site. This species, however, holds no federal status because it is demonstrably secure globally (Buffington, 1992).

One state threatened plant species, tick-trefoil, was formerly known to occur within two miles of the site. State threatened species are characterized as being in imminent danger of extirpation or extinction in New York State. The tick-trefoil is described as

being demonstrably secure globally and was last noted in the site vicinity in 1925 (Buffington, 1992).

Two plant species, the Collins sedge and the woodland agrimony, are listed by New York State as rare species and have been noted within two miles of the site. State rare species are characterized as having 20 to 35 extant locations or 3,000 to 5,000 individuals statewide. Both of these plants are known to be secure globally, but have not been observed in the site vicinity since 1927, in the case of the Collins sedge, and 1924, in the case of the woodland agrimony (Buffington, 1992).

One plant species, dwarf plantain, has recently been noted within two miles of the site. This species is listed as unprotected by the state but is ranked by the Heritage program as occurring five or fewer times in New York. The dwarf plantain is considered to be demonstrably secure globally (Buffington, 1992).

One invertebrate species, the coastal barrens buckmoth, is listed as a state species of special concern. Species of concern are characterized as those species that are not yet recognized as endangered or threatened but for which concern exists for their continued welfare in New York. The coastal barrens buckmoth is considered to be demonstrably secure globally and was observed within two miles of the site as recently as 1985 (Buffington, 1992).

8.3 Hazard Identification

Contamination at the Tronic site is summarized in Section 1 of this report. Chemical-specific summary statistics for ground water, soils, storm drain sediments, and storm drain water are presented in Appendix A. Contaminants of concern were not selected for the ecological risk assessment based on negligible exposure potential, as described below.

8.4 Exposure Assessment

Because the source areas (leaching pools and storm drains) at the Tronic site are subsurficial, the media sampled during the RI were limited to ground water, subsurface soils, storm drain sediments, and storm drain water. Contamination was most evident in the storm drain sediments and subsurface soils. It is highly unlikely that wildlife would be exposed to these contaminant sources due to their depth. As a result, the likelihood that local wildlife populations will be exposed to contaminants at the site is assumed to be negligible. It should be noted that surface soils at the site were not sampled during the RI. However, based on the history of the site, surficial contamination would not be expected.

Contaminants present in the ground water could conceivably be discharged into surface water bodies downgradient of the site. Once discharged into surface water, they could also accumulate in sediments. However, there are no natural surface water bodies located within two miles of the site (USFWS, 1981a; USFWS, 1981b). As a result, it appears highly unlikely that contaminants present in the ground water could impact aquatic species in the vicinity of the site. Furthermore, the storm drains sampled at the Tronic site recharge directly to ground water and are between 12 and 14 feet in depth. Exposure of wildlife to contaminants in storm drain water or sediments is assumed to be negligible for reasons discussed above.

The area of Farmingdale in which the Tronic site is located is zoned for industrial and residential A (cemeteries) use only. According to the Babylon town planner, there is little to no possibility that the zoning classification will change in the future (Butera, 1992). Therefore, it can be assumed that future exposures of wildlife to onsite subsurface soil contamination will be similar to present exposures, i.e., negligible.

8.5 Risk Characterization

The Tronic site and its vicinity have been heavily modified for industrial use. The only potential habitats include the site's lawn area and the wooded area 300 feet south of the site. The wooded area will most likely be cleared by the owners of Pinelawn Cemetery within fifteen years. In addition, known contamination at the site is limited to the subsurface. Based on these observations, the potential risk of exposure of wildlife to site contaminants is assumed to be negligible.

8.6 Uncertainties

This ecological risk assessment is based on the assumption that site contamination is completely subsurficial. Based on the site history, there is little reason to suspect that surficial contamination has occurred. If any has occurred, because the site is extensively paved and otherwise developed, exposure would still be minimal.

Characterization of local habitats and species was not provided in the RI, hence most of the site specific and regional species and habitat descriptions are based on information available in the literature.

9.0 SUMMARY AND CONCLUSIONS

Results from RI sampling at the Tronic site indicate that ground water, subsurface soils, storm drain sediments, and storm drain water are contaminated primarily with VOCs and metals. Most surface soils at the site are not currently exposed due to the presence of pavement and buildings. However, disposal history suggests that surface contamination is not of primary concern. Air impacts are also assumed to be minimal.

On the basis of current and future land use information, residential development of the site or its surroundings is not expected. Therefore, the risk assessment evaluated exposures to general area workers, as well as onsite utility and excavation workers. Exposure scenarios evaluated in the quantitative risk assessment included:

- ingestion of ground water by a worker,
- ingestion of and dermal contact with subsurface soils by utility and excavation workers,
- ingestion of and dermal contact with storm drain sediments by a utility worker, and
- dermal contact with storm drain water by a utility worker.

Dermal contact scenarios evaluated only cadmium quantitatively (EPA, 1992a).

All carcinogenic risk estimates fell within EPA's acceptable risk range of 10^{-6} to 10^{-4}. Ground water ingestion risks were the highest (8.7×10^{-5}), attributable to chlorinated VOCs (1,1-DCE, PCE, TCE) and metals (arsenic, beryllium). Noncarcinogenic hazard indices exceeded one only for the worker ground water ingestion scenario. The ground water ingestion chronic hazard index was 2.4; however, none of the chemical-specific hazard quotients exceeded one. TCE, antimony, and thallium contributed

most to noncarcinogenic risk, but only TCE was detected frequently (23 of 24 samples). Antimony and thallium were detected in two and one samples (of 24 samples total), respectively.

The uncertainty analysis included calculation of "central tendency" risks associated with worker ground water ingestion. The central tendency risk calculations generally incorporated average or 50th percentile exposure inputs in place of certain "upper bound" assumptions in the RME risk estimates. Total ground water ingestion carcinogenic risk calculated by this method decreased to 1.0×10^{-5}. The noncarcinogenic HI decreased to 1.7.

Lead could not be evaluated in the quantitative risk assessment in the absence of EPA-approved toxicity values. Instead, lead concentrations were compared to current EPA action levels. The maximum ground water concentration (75.5 µg/l), measured in a well potentially upgradient of the site, exceeded the drinking water action level of 15 µg/l. Storm drain water sample concentrations (138 µg/l; 14,000 µg/l) also exceeded this action level. Storm drain sediment lead concentrations (1,190 mg/kg; 2,290 mg/kg) exceeded the OSWER target cleanup range of 500 to 1,000 mg/kg.

As agreed with EPA, the ecological risk assessment was a qualitative screening evaluation based on previously published information and discussions with state and local officials. Results of the analysis suggest that the highly industrialized/modified nature of the Tronic site and its vicinity severely limits the occurrence of significant terrestrial habitats and species. In addition, no surface water bodies are located at or in the vicinity of the site. Limited ecological receptors and the remote nature of contamination at the Tronic site (subsurface soils, storm drains, ground water) indicate that risks to ecological receptors are minimal.

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APPENDIX A

CONCENTRATION DATA SUMMARIES

SUMMARY STATISTICS FOR THE TRONIC SITE.

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Ground Water

Chem. Class	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
VOCs	Methylene Chloride	1	24	3.00	3.0	MWSP1-R2	2.52	3.42	1.00	25.00
	Acetone	4	23	6.00	79.0	MW1D-R2	7.96	24.97	10.00	640.00
	1,1-Dichloroethylene	4	24	2.00	7.0	MW2S-R1	2.88	3.64	5.00	25.00
	1,1-Dichloroethane	2	24	2.00	2.0	MW3I-B	2.62	3.16	5.00	25.00
	Chloroform	1	24	3.00	3.0	PLCWW1	2.52	3.42	1.00	25.00
	1,1,1-Trichloroethane	18	24	2.00	42.0	MW3I-B	5.85	14.22	5.00	5.00
	1,2-Dichloropropane	1	24	1.00	1.0	MW3I-R2	2.57	3.22	5.00	25.00
	Trichloroethylene	23	24	1.00	490.0	MW1D-R2	37.63	288.76	18.00	18.00
	Benzene	1	24	4.00	4.0	MW2S-R1	2.55	3.49	1.00	25.00
	Tetrachloroethylene	21	24	1.00	41.0	MW3I-R2	4.24	10.22	5.00	5.00
	Toluene	2	24	3.00	4.0	MW2S-R1	2.57	3.52	1.00	25.00
	1,2-Dichloroethylene (total)	11	24	1.00	13.0	MWP5-R2	3.01	4.35	5.00	5.00
	Freon-113	5	13	1.00	61.0	MW3I-R2	4.28	22.05	5.00	5.00
BNAs	bis(2-Ethylhexyl)phthalate	1	6	11.00	11.0	MW1D-R1	6.10	9.04	10.00	15.00
Inor.	Aluminum	20	24	40.70	21400.0	MW1S-R1	382.21	33270.67	36.00	100.00
	Antimony	2	24	50.00	58.2	MW2S-R1	22.97	27.24	35.00	50.00
	Arsenic	3	24	8.00	20.0	MW2S-R1	1.80	4.76	1.40	5.00
	Barium	19	24	25.75	259.0	MW1S-R1	57.31	107.45	50.00	50.00
	Beryllium	1	24	5.70	5.7	MW1S-R1	1.77	2.50	2.00	5.00
	Cadmium	1	24	93.30	93.3	MW5S-B	2.91	5.36	5.00	5.00
	Calcium	23	23	9190.00	35550.0	MW3I-R2	15568.95	18702.13	.	.
	Chromium, total	8	18	8.20	84.1	PLCWW1	8.36	26.35	5.00	10.00
	Cobalt	4	24	11.70	48.5	MW1S-R1	6.49	10.46	10.00	10.00
	Copper	8	18	23.20	89.6	MW2S-R2	12.35	72.51	6.00	37.50
	Iron	7	7	22.00	931.0	MW5S-B	120.93	5540.44	.	.
	Lead	17	18	4.00	75.5	MW1D-R2	14.72	37.85	5.00	5.00
	Magnesium	23	23	1470.00	6650.0	MW1S-R1	3519.42	4447.86	.	.
	Manganese	23	24	7.30	3180.0	MW1S-R1	82.79	1130.50	10.00	10.00
	Nickel	10	24	31.20	114.0	MW5S-B	15.87	39.62	13.00	20.00
	Potassium	24	24	862.00	60500.0	MW3I-R1	5676.07	13987.61	.	.
	Selenium	2	24	1.40	5.0	MW2I-R1	1.37	2.85	1.00	5.00
	Silver	2	24	31.60	64.0	MW3I-R2	4.50	8.94	5.00	10.00
	Sodium	24	24	8710.00	30500.0	MW3I-R1	13802.90	15837.97	.	.
	Thallium	1	24	5.60	5.6	MW1S-R2	1.38	2.80	1.10	5.00
	Vanadium	8	24	10.10	43.8	MW1S-R1	7.52	14.76	8.00	12.60
	Zinc	3	3	41.60	90.7	MW2D-B	55.39	330.07	.	.
	Cyanide	1	24	60.00	60.0	MWSP1-R2	5.04	8.89	1.00	10.00
	Chromium, VI	13	14	7.00	68.5	MW3I-R2	22.72	84.51	5.00	5.00

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Subsurface Soils

Chem. Class	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
VOCs	Methylene Chloride	2	12	11.00	14.0	DW1P5(5-7)	3.61	7.32	5.00	16.50
	Acetone	7	12	10.00	61.0	LP1(6-10)	10.70	29.84	6.00	23.50
	Styrene	6	12	5.00	5.0	LP1(6-10)	3.54	4.69	5.00	5.00
	Total Xylenes	1	7	5.00	5.0	LP2(14-16)	2.76	3.57	5.00	5.00
	Freon-113	2	2	7.90	46.0	SP1(7-9)	19.06	46.00	.	.
BNAs	Benzyl alcohol	1	12	340.00	340.0	DW1P5(5-7)	183.38	208.34	340.00	360.00
	3-Nitroaniline	1	12	1600.00	1600.0	SP1(7-9)	886.74	1002.21	1600.00	1800.00
	Di-n-butylphthalate	4	12	94.00	2600.0	MW2I(10-12.5)	232.75	1597.14	68.00	4100.00
	bis(2-Ethylhexyl)phthalate	5	12	160.00	2800.0	LP2(8-10)	388.68	1483.92	340.00	1800.00
	Indeno(1,2,3-cd)pyrene	1	12	340.00	340.0	SP1(7-9)	183.38	208.34	340.00	360.00
P/PCBs	4,4-DDE	1	12	72.00	72.0	LP2(14-16)	9.95	18.65	16.00	17.00
	4,4-DDT	1	12	37.00	37.0	LP2(14-16)	9.42	13.52	16.00	17.00
Inor.	Aluminum	12	12	682000.00	3780000.0	LP3(5-9)	1159220.80	2074577.13	.	.
	Antimony	1	12	10900.00	10900.0	MW2D(10-12)	5485.94	6340.38	10100.00	10600.00
	Arsenic	4	17	970.00	1700.0	LP3(5-9)	457.33	945.42	290.00	1000.00
	Barium	8	18	2900.00	15600.0	R4(1-3)	5527.84	7577.83	10200.00	10500.00
	Cadmium	4	18	1300.00	3650.0	MW2D(10-12)	719.29	1299.67	1000.00	1120.00
	Calcium	12	12	5170.00	749000.0	LP3(5-9)	361320.42	3805348.43	.	.
	Chromium, total	15	18	1600.00	12000.0	LP2(14-16)	2943.56	5660.38	2060.00	2100.00
	Cobalt	3	12	2400.00	3900.0	LP3(5-9)	1347.50	2113.73	2000.00	2100.00
	Copper	16	18	1300.00	30900.0	LP2(14-16)	4439.99	15608.78	1250.00	1260.00
	Iron	12	12	1120000.00	6040000.0	LP3(5-9)	3065443.20	5130241.76	.	.
	Lead	14	18	520.00	47100.0	MW3I(10-12)	1525.81	7009.08	1000.00	1000.00
	Magnesium	12	12	177000.00	466000.0	LP2(14-16)	297304.95	363638.78	.	.
	Manganese	12	12	12200.00	102000.0	DW1P5(5-7)	43100.19	83638.54	.	.
	Nickel	3	18	6050.00	6300.0	LP2(14-16)	2148.88	3179.78	2660.00	4200.00
	Silver	4	7	1100.00	3600.0	LP2(14-16)	967.57	2891.81	1020.00	1050.00
	Sodium	12	12	51800.00	99000.0	MW2I(10-12.5)	74313.81	84451.03	.	.
	Vanadium	9	12	2300.00	9800.0	LP3(5-9)	2996.48	7737.69	2000.00	2000.00
	Zinc	14	16	4400.00	21200.0	LP3(5-9)	8482.46	15099.08	4000.00	4700.00
	Cyanide	4	18	430.00	46900.0	LP2(14-16)	393.98	7790.27	200.00	1120.00
	Chromium, VI	12	12	60.00	22600.0	LP2(8-10)	1915.16	1396902.04	.	.

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Subsurface Soils/Sediments (1-16')

Chem. Class	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
VOCs	Methylene Chloride	2	14	11.00	14.0	DW1P5(5-7)	3.48	6.24	5.00	16.50
	Acetone	7	14	10.00	61.0	LP1(6-10)	9.75	23.72	6.00	23.50
	Styrene	6	14	5.00	5.0	LP1(6-10)	3.42	4.36	5.00	5.70
	Total Xylenes	1	9	5.00	5.0	LP2(14-16)	2.77	3.32	5.00	5.70
	Freon-113	2	4	7.90	46.0	SP1(7-9)	7.29	13791.63	5.45	5.70
BNAs	Benzyl alcohol	1	12	340.00	340.0	DW1P5(5-7)	183.38	208.34	340.00	360.00
	3-Nitroaniline	1	12	1600.00	1600.0	SP1(7-9)	886.74	1002.21	1600.00	1800.00
	Di-n-butylphthalate	4	12	94.00	2600.0	MW2I(10-12.5)	232.75	1597.14	68.00	4100.00
	bis(2-Ethylhexyl)phthalate	5	12	160.00	2800.0	LP2(8-10)	388.68	1483.92	340.00	1800.00
	Indeno(1,2,3-cd)pyrene	1	12	340.00	340.0	SP1(7-9)	183.38	208.34	340.00	360.00
P/PCBs	4,4-DDE	1	12	72.00	72.0	LP2(14-16)	9.95	18.65	16.00	17.00
	4,4-DDT	1	12	37.00	37.0	LP2(14-16)	9.42	13.52	16.00	17.00
Inor.	Aluminum	14	14	595000.00	3780000.0	LP3(5-9)	1055251.48	1814767.44	.	.
	Antimony	1	13	10900.00	10900.0	MW2D(10-12)	5351.66	6193.50	7950.00	10600.00
	Arsenic	5	19	780.00	1700.0	LP3(5-9)	471.82	899.34	290.00	1000.00
	Barium	10	20	2900.00	15600.0	R4(1-3)	5586.41	7409.18	10200.00	10500.00
	Cadmium	6	20	1300.00	6400.0	SD2(14-16)	870.83	2071.24	1000.00	1120.00
	Calcium	14	14	5170.00	6700000.0	SD1(14-16)	419008.28	5778942.38	.	.
	Chromium, total	17	20	1600.00	12000.0	LP2(14-16)	3162.58	5889.70	2060.00	2100.00
	Cobalt	3	14	2400.00	3900.0	LP3(5-9)	1311.50	1907.38	2000.00	2270.00
	Copper	18	20	1300.00	30900.0	LP2(14-16)	4876.25	16068.30	1250.00	1260.00
	Iron	14	14	1120000.00	6040000.0	LP3(5-9)	2754141.53	4611941.46	.	.
	Lead	16	20	520.00	54000.0	SD1(14-16)	1976.11	14883.11	1000.00	1000.00
	Magnesium	14	14	177000.00	2710000.0	SD1(14-16)	338782.36	639484.72	.	.
	Manganese	14	14	12200.00	102000.0	DW1P5(5-7)	41698.59	72168.96	.	.
	Nickel	5	20	5800.00	10600.0	SD1(14-16)	2445.84	4109.47	2660.00	4200.00
	Potassium	1	7	110000.00	110000.0	SD1(14-16)	49996.59	75930.81	80800.00	122000.00
	Silver	5	9	1100.00	3600.0	LP2(14-16)	978.47	2331.81	1020.00	1090.00
	Sodium	13	14	51800.00	99000.0	MW2I(10-12.5)	68430.00	88791.96	46100.00	46100.00
	Vanadium	10	14	2300.00	9800.0	LP3(5-9)	2949.59	6362.46	2000.00	6000.00
	Zinc	16	18	4400.00	69900.0	SD1(14-16)	9810.01	20970.01	4000.00	4700.00
	Cyanide	5	20	430.00	46900.0	LP2(14-16)	422.46	5868.94	200.00	1140.00
	Chromium, VI	12	12	60.00	22600.0	LP2(8-10)	1915.16	1396902.04	.	.

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Subsurface Soils/Sediments (0-16')

Chem. Class	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
VOCs	Chloromethane	1	22	8.00	8.0	SD3(BOTTOM)	6.62	9.40	10.00	91.00
	Vinyl Chloride	1	22	20.00	20.0	SD5(BOTTOM)	6.82	10.01	10.00	91.00
	Methylene Chloride	5	22	5.00	14.0	DW1P5(5-7)	4.10	6.67	5.00	16.95
	Acetone	12	22	10.00	140.0	SD2(BOTTOM)-R1	12.86	35.05	6.00	23.50
	2-Butanone (MEK)	1	8	3.00	3.0	SD6(BOTTOM)	6.41	10.90	10.90	33.90
	Trichloroethylene	1	22	180.00	180.0	SD5(BOTTOM)	3.78	10.74	5.00	45.00
	Tetrachloroethylene	1	22	2.00	2.0	SD3(BOTTOM)	3.13	4.52	5.00	45.00
	1,1,2,2-Tetrachloroethane	1	22	4.00	4.0	SD3(BOTTOM)	3.23	4.62	5.00	45.00
	Toluene	3	22	4.00	36.0	SD5(BOTTOM)	3.56	6.46	5.00	45.00
	Ethylbenzene	3	22	4.00	30.0	SD2(BOTTOM)-R1	3.41	5.81	5.00	7.80
	Styrene	6	21	5.00	5.0	LP1(6-10)	3.89	5.69	5.00	45.00
	Total Xylenes	5	17	1.00	140.0	SD2(BOTTOM)-R1	4.62	28.09	5.00	7.80
	1,2-Dichloroethylene (total)	1	22	38.00	38.0	SD5(BOTTOM)	3.53	6.50	5.00	45.00
	Freon-113	2	10	7.90	46.0	SP1(7-9)	5.43	17.64	5.45	16.95
BNAs	Benzyl alcohol	1	20	340.00	340.0	DW1P5(5-7)	389.25	1081.66	340.00	3121.80
	Naphthalene	2	20	910.00	5600.0	SD2(BOTTOM)	396.50	1447.18	340.00	2781.90
	2-Methylnaphthalene	4	20	1500.00	20000.0	SD2(BOTTOM)	483.86	3989.92	340.00	2781.90
	Dimethylphthalate	1	20	2500.00	2500.0	SD2(BOTTOM)	384.96	1194.73	340.00	2781.90
	3-Nitroaniline	1	20	1600.00	1600.0	SP1(7-9)	1907.62	5458.52	1600.00	15609.00
	Acenaphthene	1	20	1700.00	1700.0	SD2(BOTTOM)	377.60	1097.63	340.00	2781.90
	Dibenzofuran	1	20	1200.00	1200.0	SD2(BOTTOM)	371.08	1029.15	340.00	2781.90
	Fluorene	2	20	1900.00	2950.0	SD2(BOTTOM)	399.91	1383.69	340.00	2781.90
	Phenanthrene	4	20	1600.00	6900.0	SD2(BOTTOM)	455.68	2532.58	340.00	2781.90
	Di-n-butylphthalate	4	20	94.00	2600.0	MW2I(10-12.5)	449.12	2222.04	68.00	4100.00
	Fluoranthene	4	20	1400.00	2300.0	SD2(BOTTOM)	402.60	1384.97	340.00	2781.90
	Pyrene	4	18	930.00	4200.0	SD2(BOTTOM)	353.09	1305.09	340.00	2399.10
	Chrysene	2	18	720.00	1000.0	SD7(BOTTOM)	317.40	811.03	340.00	3121.80
	bis(2-Ethylhexyl)phthalate	12	19	160.00	43000.0	SD6(BOTTOM)	1341.12	58310.51	340.00	1800.00
	Indeno(1,2,3-cd)pyrene	1	18	340.00	340.0	SP1(7-9)	338.31	888.13	340.00	2983.20
P/PCBs	4,4-DDE	1	12	72.00	72.0	LP2(14-16)	9.95	18.65	16.00	17.00
	4,4-DDT	1	12	37.00	37.0	LP2(14-16)	9.42	13.52	16.00	17.00
Inor.	Aluminum	22	22	595000.00	21600000.0	SD2(BOTTOM)-R1	1674015.56	4276105.72	.	.
	Antimony	1	15	10900.00	10900.0	MW2D(10-12)	5536.63	6358.64	7950.00	14900.00
	Arsenic	7	21	780.00	9800.0	SD2(BOTTOM)-R1	576.01	1617.00	290.00	1000.00
	Barium	10	20	2900.00	15600.0	R4(1-3)	5586.41	7409.18	10200.00	10500.00
	Beryllium	2	22	1300.00	3500.0	SD2(BOTTOM)-R1	451.05	724.65	440.00	1100.00
	Cadmium	14	28	1300.00	1130000.0	SD2(BOTTOM)-R1	2159.76	61094.49	1000.00	1120.00
	Calcium	22	22	5170.00	57700000.0	SD2(BOTTOM)-R1	1614460.39	173586954.45	.	.
	Chromium, total	25	28	1600.00	1580000.0	SD2(BOTTOM)-R1	7766.63	110547.99	2060.00	2100.00
	Cobalt	11	22	2400.00	23400.0	SD2(BOTTOM)-R1	2200.06	5040.93	2000.00	2270.00
	Copper	24	26	1300.00	4560000.0	SD3(BOTTOM)	13953.01	1176142.41	1250.00	1260.00
		20	2	11	00	100	6(B	338	42.	

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Subsurface Soils/Sediments (0-16')
(continued)

Chem. Class	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	18	22	520.00	2290000.0	SD2(BOTTOM)-R1	3642.81	762975.29	1000.00	1000.00
	Magnesium	22	22	177000.00	29200000.0	SD4(BOTTOM)	1148309.60	24840254.88	.	.
	Manganese	20	20	12200.00	102000.0	DW1P5(5-7)	43366.92	63853.88	.	.
	Mercury	1	22	310.00	310.0	SD2(BOTTOM)-R1	17.18	41.16	20.00	300.00
	Nickel	11	26	5800.00	138000.0	SD3(BOTTOM)	4362.22	20672.04	2660.00	4200.00
	Potassium	3	12	110000.00	1440000.0	SD2(BOTTOM)-R1	88160.19	420599.45	80800.00	196000.00
	Selenium	1	22	2400.00	2400.0	SD2(BOTTOM)-R1	311.31	739.03	200.00	1300.00
	Silver	5	9	1100.00	3600.0	LP2(14-16)	978.47	2331.81	1020.00	1090.00
	Sodium	15	22	51800.00	735000.0	SD2(BOTTOM)-R1	84157.45	145795.92	46100.00	166000.00
	Vanadium	16	20	2300.00	46000.0	SD6(BOTTOM)	5765.75	28342.07	2000.00	6000.00
	Zinc	22	24	4400.00	3200000.0	SD3(BOTTOM)	26078.41	777162.66	4000.00	4700.00
	Cyanide	9	28	430.00	46900.0	LP2(14-16)	638.82	8161.15	200.00	1450.00
	Chromium, VI	14	14	60.00	22600.0	LP2(8-10)	2001.92	354006.29	.	.

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Storm Drain Sediments

Chem. Class	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
VOCs	Chloromethane	1	8	8.00	8.0	SD3(BOTTOM)	9.82	25.99	11.90	91.00
	Vinyl Chloride	1	8	20.00	20.0	SD5(BOTTOM)	10.62	29.31	11.90	91.00
	Methylene Chloride	3	8	5.00	13.0	SD2(BOTTOM)-R1	5.47	10.72	5.95	16.95
	Acetone	5	8	21.00	140.0	SD2(BOTTOM)-R1	20.88	170.24	11.90	14.60
	2-Butanone (MEK)	1	6	3.00	3.0	SD6(BOTTOM)	6.71	15.60	11.90	33.90
	Trichloroethylene	1	8	180.00	180.0	SD5(BOTTOM)	7.61	262.96	5.95	45.00
	Tetrachloroethylene	1	8	2.00	2.0	SD3(BOTTOM)	4.50	13.90	5.95	45.00
	1,1,2,2-Tetrachloroethane	1	8	4.00	4.0	SD3(BOTTOM)	4.90	12.88	5.95	45.00
	Toluene	3	8	4.00	36.0	SD5(BOTTOM)	6.41	32.95	5.95	45.00
	Ethylbenzene	3	8	4.00	30.0	SD2(BOTTOM)-R1	5.69	27.30	5.95	7.80
	Total Xylenes	4	8	1.00	140.0	SD2(BOTTOM)-R1	8.22	1146.23	5.95	7.80
	1,2-Dichloroethylene (total)	1	8	38.00	38.0	SD5(BOTTOM)	6.26	36.45	5.95	45.00
BNAs	Naphthalene	2	8	910.00	5600.0	SD2(BOTTOM)	1374.76	2839.28	1943.70	2781.90
	2-Methylnaphthalene	4	8	1500.00	20000.0	SD2(BOTTOM)	2261.51	15894.99	1943.70	2781.90
	Dimethylphthalate	1	8	2500.00	2500.0	SD2(BOTTOM)	1276.83	1675.00	1943.70	2781.90
	Acenaphthene	1	8	1700.00	1700.0	SD2(BOTTOM)	1216.74	1404.68	1943.70	2781.90
	Dibenzofuran	1	8	1200.00	1200.0	SD2(BOTTOM)	1164.90	1270.63	1943.70	2781.90
	Fluorene	2	8	1900.00	2950.0	SD2(BOTTOM)	1404.48	2001.38	1943.70	2781.90
	Phenanthrene	4	8	1600.00	6900.0	SD2(BOTTOM)	1946.49	5005.87	1943.70	2781.90
	Fluoranthene	4	8	1400.00	2300.0	SD2(BOTTOM)	1628.21	1785.83	1943.70	2781.90
	Pyrene	4	6	930.00	4200.0	SD2(BOTTOM)	1469.34	3442.91	1943.70	2399.10
	Chrysene	2	6	720.00	1000.0	SD7(BOTTOM)	1067.36	1412.42	1943.70	3121.80
	bis(2-Ethylhexyl)phthalate	7	7	2400.00	43000.0	SD6(BOTTOM)	11208.41	115215.71	.	.
Inor.	Aluminum	8	8	951000.00	21600000.0	SD2(BOTTOM)-R1	3753752.68	16026472.39	.	.
	Arsenic	2	2	1500.00	9800.0	SD2(BOTTOM)-R1	3834.06	9800.00	.	.
	Beryllium	2	8	1300.00	3500.0	SD2(BOTTOM)-R1	455.67	2670.91	480.00	620.00
	Cadmium	8	8	2000.00	1130000.0	SD2(BOTTOM)-R1	20921.28	30428840.49	.	.
	Calcium	8	8	4210000.00	57700000.0	SD2(BOTTOM)-R1	17107469.30	77169792.25	.	.
	Chromium, total	8	8	16700.00	1580000.0	SD2(BOTTOM)-R1	73402.58	2593755.85	.	.
	Cobalt	8	8	3200.00	23400.0	SD2(BOTTOM)-R1	5440.04	12199.63	.	.
	Copper	6	6	87900.00	4560000.0	SD3(BOTTOM)	464096.57	19776822.05	.	.
	Iron	6	6	3570000.00	7750000.0	SD6(BOTTOM)	5522562.97	7814504.91	.	.
	Lead	2	2	1190000.00	2290000.0	SD2(BOTTOM)-R1	1650787.69	2290000.00	.	.
	Magnesium	8	8	2850000.00	29200000.0	SD4(BOTTOM)	9723012.79	32634607.05	.	.
	Manganese	6	6	25500.00	68200.0	SD4(BOTTOM)	47523.91	71109.58	.	.
	Mercury	1	2	310.00	310.0	SD2(BOTTOM)-R1	215.64	310.00	300.00	300.00
	Nickel	6	6	11300.00	138000.0	SD3(BOTTOM)	30012.82	234904.37	.	.
	Potassium	2	5	320000.00	1440000.0	SD2(BOTTOM)-R1	195046.25	20229292.12	133000.00	196000.00
	Selenium	1	2	2400.00	2400.0	SD2(BOTTOM)-R1	1249.00	2400.00	1300.00	1300.00
	Sodium	2	8	403000.00	735000.0	SD2(BOTTOM)-R1	120871.25	622995.37	111000.00	166000.00
	Vanadium	6	6	13300.00	46000.0	SD6(BOTTOM)	27546.92	47003.47	.	.
		1		1 00	000	03(B	999(	848		

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Storm Drain Sediments
(continued)

Chem. Class	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
	Cyanide	4	8	1100.00	92000.0	SD2(BOTTOM)-R1	1981.23	304970.09	1190.00	1450.00
	Chromium, VI	2	2	2200.00	3100.0	SD2(BOTTOM)-R1	2611.51	3100.00	.	.

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Storm Drain Water

Chem. Class	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
Inor.	Aluminum	2	2	3140.00	162000.0	SD2-R1	22553.94	162000.00	.	.
	Antimony	1	2	73.50	73.5	SD2-R1	42.87	73.50	50.00	50.00
	Arsenic	1	2	28.40	28.4	SD2-R1	8.43	28.40	5.00	5.00
	Barium	1	2	2350.00	2350.0	SD2-R1	242.38	2350.00	50.00	50.00
	Beryllium	1	2	27.50	27.5	SD2-R1	8.29	27.50	5.00	5.00
	Cadmium	2	2	24.70	8270.0	SD2-R1	451.96	8270.00	.	.
	Calcium	2	2	20400.00	296000.0	SD2-R1	77707.14	296000.00	.	.
	Cobalt	1	2	163.00	163.0	SD2-R1	28.55	163.00	10.00	10.00
	Lead	2	2	138.00	14100.0	SD2-R1	1394.92	14100.00	.	.
	Magnesium	2	2	4780.00	132000.0	SD2-R1	25118.92	132000.00	.	.
	Manganese	2	2	86.30	2520.0	SD2-R1	466.34	2520.00	.	.
	Mercury	1	2	1.10	1.1	SD2-R1	0.33	1.10	0.20	0.20
	Nickel	2	2	32.00	11900.0	SD2-R1	617.09	11900.00	.	.
	Potassium	2	2	1140.00	10900.0	SD2-R1	3525.05	10900.00	.	.
	Selenium	1	2	9.00	9.0	SD2-R1	4.74	9.00	5.00	5.00
	Silver	1	2	759.00	759.0	SD2-R1	61.60	759.00	10.00	10.00
	Sodium	2	2	1700.00	5310.0	SD2-R1	3004.50	5310.00	.	.
	Vanadium	2	2	15.80	797.0	SD2-R1	112.22	797.00	.	.
	Cyanide	1	2	1.30	1.3	SD2-R1	2.55	1.30	10.00	10.00
	Chromium, VI	2	2	13.00	22.0	SD1-R1	16.91	22.00	.	.

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Background Soils

Chem. Class	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
VOCs	Acetone	1	3	74.00	74.0	MW1D(10-12)	13.47	22237626495.25	11.00	12.00
	Chloroform	2	3	8.00	9.0	MW1D(10-12)	6.00	204.61	6.00	6.00
BNAs	Diethylphthalate	1	3	150.00	150.0	MW1D(37-40)	164.64	150.00	340.00	350.00
	Di-n-butylphthalate	3	3	5200.00	6000.0	MW1D(25-27)	5454.05	6000.00	.	.
	Benzylbutylphthalate	3	3	47.00	820.0	MW1D(37-40)	206.96	98924894467.73	.	.
Inor.	Aluminum	3	3	651000.00	1140000.0	MW1D(10-12)	861983.35	1948614.75	.	.
	Calcium	3	3	493000.00	538000.0	MW1D(37-40)	514338.29	538000.00	.	.
	Chromium, total	3	3	3700.00	6900.0	MW1D(25-27)	5133.84	13765.15	.	.
	Cobalt	2	3	2600.00	3300.0	MW1D(37-40)	2047.21	98596.19	2000.00	2000.00
	Copper	3	3	4000.00	7200.0	MW1D(10-12)	5691.03	15105.02	.	.
	Iron	3	3	1740000.00	3790000.0	MW1D(25-27)	2531480.12	11149003.44	.	.
	Lead	1	3	1100.00	1100.0	MW1D(25-27)	650.30	4845.14	1000.00	1000.00
	Magnesium	3	3	247000.00	322000.0	MW1D(10-12)	291365.24	398015.20	.	.
	Manganese	3	3	52600.00	74500.0	MW1D(37-40)	62599.68	93405.92	.	.
	Sodium	3	3	65800.00	85600.0	MW1D(10-12)	75133.14	99202.29	.	.
	Vanadium	3	3	2500.00	4500.0	MW1D(25-27)	3586.29	9785.45	.	.
	Zinc	3	3	8300.00	12500.0	MW1D(10-12)	9737.77	16944.06	.	.
	Chromium, VI	3	3	120.00	450.0	MW1D(25-27)	213.41	18316.73	.	.

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Deep Soils

Chem. Class	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
VOCs	Methylene Chloride	2	23	8.00	20.0	DW1P5(19-21)	3.51	5.20	5.00	20.00
	Acetone	8	22	26.00	150.0	MW2I(22.5-25)	15.01	58.21	10.00	110.00
	Styrene	2	23	5.00	5.0	LP1(14-19)	2.88	3.16	5.00	6.20
	Freon-113	5	8	15.00	51.0	SP1(19-21)	12.65	212.90	5.50	6.20
BNAs	Benzyl alcohol	1	20	340.00	340.0	DW1P5(19-21)	185.83	199.74	340.00	410.00
	3-Nitroaniline	4	20	1600.00	1800.0	SP1(37-39)	1000.38	1154.52	1600.00	2000.00
	4-Nitrophenol	1	20	1600.00	1600.0	SP1(19-21)	901.59	966.89	1600.00	2000.00
	4-Nitroaniline	1	20	1600.00	1600.0	SP1(19-21)	901.59	966.89	1600.00	2000.00
	Di-n-butylphthalate	12	20	130.00	5100.0	LP1(38-40)	638.06	3979.93	340.00	2300.00
	bis(2-Ethylhexyl)phthalate	8	20	130.00	2600.0	MW2D(37-40)	358.39	1390.41	70.00	4800.00
	Di-n-octylphthalate	1	20	77.00	77.0	SP1(19-21)	172.53	190.58	340.00	410.00
	Indeno(1,2,3-cd)pyrene	1	20	360.00	360.0	SP1(37-39)	185.83	201.28	340.00	410.00
Inor.	Aluminum	22	22	137000.00	2010000.0	LP3(18-20)	497561.58	763110.40	.	.
	Arsenic	7	31	320.00	2100.0	MW3I(75-77.5)	421.84	634.82	290.00	1200.00
	Barium	10	31	1400.00	4700.0	R5(19-21)	4265.89	5159.68	3500.00	11500.00
	Cadmium	7	32	1400.00	8200.0	LP2(18-20)	775.94	1380.16	1000.00	1240.00
	Calcium	21	22	4300.00	685000.0	MW2D(20-22)	262157.27	2340398.95	9800.00	9800.00
	Chromium, total	28	32	2200.00	62600.0	SP1(37-39)	4889.34	14469.08	1100.00	2470.00
	Copper	29	32	1500.00	38000.0	LP1(14-19)	4387.93	9832.04	1230.00	6300.00
	Iron	22	22	41900.00	4680000.0	MW3I(75-77.5)	1424100.67	4331533.53	.	.
	Lead	20	32	300.00	69200.0	MW3I(32-35)	1056.22	3098.93	1000.00	1200.00
	Magnesium	22	22	18300.00	444000.0	LP3(18-20)	156898.75	292421.64	.	.
	Manganese	22	22	2900.00	60500.0	LP3(18-20)	17550.99	34458.34	.	.
	Nickel	4	32	2900.00	13800.0	LP1(14-19)	2071.05	2728.64	2660.00	4600.00
	Silver	1	14	1100.00	1100.0	R3(29-31)	612.58	751.25	1020.00	2100.00
	Sodium	19	22	51500.00	109000.0	LP4(38-40)	65228.21	97144.47	17200.00	167000.00
	Thallium	1	21	270.00	270.0	MW5S(39-41)	481.02	594.26	240.00	1200.00
	Vanadium	10	22	2400.00	6100.0	MW2I(22.5-25)	1818.34	3114.59	1790.00	2300.00
	Zinc	19	28	4600.00	15600.0	LP1(14-19)	5587.86	8797.93	2600.00	8300.00
	Cyanide	12	32	320.00	15000.0	LP1(14-19)	445.77	1912.69	200.00	1240.00
	Chromium, VI	17	19	100.00	23440.0	LP1(14-19)	1634.28	44268.20	7300.00	10000.00

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=Deep Sediments

Chem. Class	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
VOCs	Acetone	1	9	8.00	8.0	SD2(21-23)	5.72	6.27	10.60	11.40
	Carbon Disulfide	1	9	22.00	22.0	SD2(21-23)	3.46	8.34	5.30	5.70
Inor.	Aluminum	9	9	211000.00	1130000.0	SD2(31-33)	458268.24	811373.51	.	.
	Arsenic	6	9	360.00	990.0	SD2(31-33)	444.71	1054.22	300.00	970.00
	Barium	7	9	2800.00	9300.0	SD2(31-33)	4431.16	9301.13	3300.00	3700.00
	Cadmium	5	9	1300.00	8150.0	SD2(21-23)	1564.68	12226.81	1060.00	1110.00
	Calcium	9	9	30400.00	6700000.0	SD1(14-16)	140247.94	6501961.36	.	.
	Chromium, total	7	9	2200.00	12300.0	SD2(21-23)	3296.56	10464.77	2160.00	2175.00
	Copper	8	9	1900.00	17600.0	SD1(14-16)	3612.49	15677.35	1300.00	1300.00
	Iron	9	9	414000.00	2670000.0	SD3(17-19)	1006928.29	2347199.02	.	.
	Lead	8	9	320.00	54000.0	SD1(14-16)	2136.62	733534.93	220.00	220.00
	Magnesium	9	9	57400.00	2710000.0	SD1(14-16)	184017.00	1871602.96	.	.
	Manganese	9	9	7400.00	38500.0	SD2(21-23)	21409.44	45482.77	.	.
	Mercury	1	9	50.00	50.0	SD2(21-23)	22.70	29.56	40.00	50.00
	Nickel	4	9	2900.00	10600.0	SD1(14-16)	2690.14	8723.71	2750.00	2920.00
	Potassium	4	9	62700.00	339000.0	SD2(31-33)	70265.46	170281.66	77700.00	122000.00
	Silver	1	9	1900.00	1900.0	SD1(14-16)	627.33	939.01	1060.00	1120.00
	Sodium	3	9	45600.00	75500.0	SD1(14-16)	33758.56	53408.99	38500.00	64850.00
	Vanadium	5	9	2200.00	4600.0	SD2(31-33)	1994.30	4132.99	1730.00	6000.00
	Zinc	9	9	4300.00	69900.0	SD1(14-16)	14964.24	63641.23	.	.
	Cyanide	3	9	1100.00	1450.0	SD2(21-23)	714.05	1048.43	1060.00	1140.00

SUMMARY STATISTICS FOR THE TRONIC SITE. (continued).

SUMMARY STATISTICS FOR SITE, BY CHEMICAL AND MEDIUM/AREA
all in units of ppb

TYPE=USGS Well

Chem. Class	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
VOCs	1,1,1-Trichloroethane	2	2	21.00	28.0	MWUG-1806-R1	24.25	28.00	.	.
	Trichloroethylene	2	2	14.00	61.0	MWUG-1806-R1	29.22	61.00	.	.
	Freon-113	1	1	120.00	120.0	MWUG-1806-R1	120.00	120.00	.	.
Inor.	Aluminum	2	2	8800.00	80900.0	MWUG-1806-R1	26681.83	80900.00	.	.
	Arsenic	1	2	9.00	9.0	MWUG-1806-R1	4.74	9.00	5.00	5.00
	Barium	2	2	80.40	392.0	MWUG-1806-R1	177.53	392.00	.	.
	Beryllium	1	2	7.20	7.2	MWUG-1806-R1	4.24	7.20	5.00	5.00
	Cadmium	2	2	23.90	151.0	MWUG-1806-R1	60.07	151.00	.	.
	Calcium	2	2	15400.00	20300.0	MWUG-1806-R1	17681.06	20300.00	.	.
	Chromium, total	1	1	36.50	36.5	MWUG-1806-R2	36.50	36.50	.	.
	Cobalt	1	2	32.30	32.3	MWUG-1806-R1	12.71	32.30	10.00	10.00
	Copper	1	1	65.10	65.1	MWUG-1806-R2	65.10	65.10	.	.
	Lead	2	2	312.00	2550.0	MWUG-1806-R1	891.96	2550.00	.	.
	Magnesium	2	2	4110.00	11000.0	MWUG-1806-R1	6723.84	11000.00	.	.
	Manganese	2	2	246.00	3230.0	MWUG-1806-R1	891.39	3230.00	.	.
	Mercury	1	2	1.10	1.1	MWUG-1806-R1	0.33	1.10	0.20	0.20
	Nickel	2	2	22.10	74.0	MWUG-1806-R1	40.44	74.00	.	.
	Potassium	2	2	3560.00	6190.0	MWUG-1806-R1	4694.29	6190.00	.	.
	Sodium	2	2	9060.00	9120.0	MWUG-1806-R1	9089.95	9120.00	.	.
	Vanadium	2	2	16.80	150.0	MWUG-1806-R1	50.20	150.00	.	.
	Chromium, VI	2	2	38.00	80.0	MWUG-1806-R2	55.14	80.00	.	.

APPENDIX B

TOXICITY VALUES FOR ALL DETECTED CONTAMINANTS

TOXICITY VALUES FOR ALL CONTAMINANTS DETECTED AT THE TRONIC SITE.

Chemical	CARCINOGENIC Oral Slope Factor (mg/kg/day)-1	CHRONIC Chronic Oral RfD (mg/kg/day)	SUBCHRONIC Subchronic Oral RfD (mg/kg/day)	ACUTE Acute Oral "RfD" [1-Day HA/10] (mg/kg/day)
Volatiles				
Acetone		1.00E-01 a	1.00E+00 b	
Benzene	2.90E-02 a			2.00E-02 c
2-Butanone (MEK)		5.00E-02 b	5.00E-01 b	8.00E+00 c
Carbon disulfide		1.00E-01 a	1.00E-01 b	
Chloroform	6.10E-03 a	1.00E-02 a	1.00E-02 b	4.00E-01 c
Chloromethane (methyl chloride)	1.30E-02 b			9.00E-01 c
1,1-Dichloroethane		1.00E-01 b	1.00E+00 b	
1,1-Dichloroethylene	6.00E-01 a	9.00E-03 a	9.00E-03 b	2.00E-01 c
1,2-Dichloroethylene (total)		1.00E-02 k	1.00E-01 k	4.00E-01 k
1,2-Dichloropropane	6.80E-02 b			
Ethylbenzene		1.00E-01 a	1.00E+00 b	3.20E+00 a
Methylene chloride	7.50E-03 a	6.00E-02 a	6.00E-02 b	1.33E+00 a
Styrene	3.00E-02 b	2.00E-01 a	2.00E+00 b	2.00E+00 a
1,1,2,2-Tetrachloroethane	2.00E-01 a			
Tetrachloroethylene	5.10E-02 b	1.00E-02 a	1.00E-01 b	2.00E-01 a
Toluene		2.00E-01 b	2.00E+00 b	2.00E+00 c
1,1,1-Trichloroethane		9.00E-02 b	9.00E-01 b	1.00E+01 a
Trichloroethylene	1.10E-02 b	6.00E-03 d	6.00E-03 j	
Trichlorotrifluoroethane (Freon-113)		3.00E+01 b	3.00E+00 b	
Vinyl chloride (chloroethylene)	1.90E+00 b			3.00E-01 c
Xylenes		2.00E+00 a	4.00E+00 b	4.00E+00 c
Semivolatiles				
Acenaphthene		6.00E-02 a	6.00E-01 b	
Benzyl alcohol		3.00E-01 b	1.00E+00 b	
Benzylbutylphthalate		2.00E-01 a	2.00E+00 b	
Bis(2-ethylhexyl)phthalate	1.40E-02 a	2.00E-02 a	2.00E-02 b	
Chrysene	5.79E+00 e			
Dibenzofuran		4.00E-03 d	4.00E-03 j	
Di-n-butyl phthalate		1.00E-01 a	1.00E+00 b	
Dimethylphthalate		1.00E+00 b	1.00E+00 b	
Fluoranthene		4.00E-02 a	4.00E-01 b	
Fluorene		4.00E-02 a	4.00E-01 b	
Indeno(1,2,3-cd)pyrene	5.79E+00 e			
2-Methylnaphthalene				
Naphthalene		4.00E-03 b	4.00E-02 b	5.00E-02 c
3-Nitroaniline	4.00E-02 d	3.00E-04 d	3.00E-04 j	
Phenanthrene				
Pyrene		3.00E-02 a	3.00E-01 b	
Inorganics				
Aluminum		1.00E+00 d	1.00E+00 j	
Antimony		4.00E-04 a	4.00E-04 b	1.50E-03 c
Arsenic	1.75E+00 f	3.00E-04 a	1.00E-03 b	
Barium		5.00E-02 b	5.00E-02 b	
Beryllium	4.30E+00 a	5.00E-03 a	5.00E-03 b	3.00E+00 c
Cadmium (l)		5.00E-04 a,g	5.00E-04 j	4.00E-03 c
Calcium				
Chromium, total		8.76E-01 i	8.75E+00 i	1.40E-01 a
* Chromium, III		1.00E+00 a	1.00E+01 b	
Chromium, VI		5.00E-03 a	2.00E-02 b	
Cobalt		d		
Copper		4.00E-02 d	4.00E-02 j	

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

Chemical	CARCINOGENIC Oral Slope Factor (mg/kg/day)-1	CHRONIC Chronic Oral RfD (mg/kg/day)	SUBCHRONIC Subchronic Oral RfD (mg/kg/day)	ACUTE Acute Oral "RfD" [1-Day HA/10] (mg/kg/day)
Cyanide		2.00E-02 a	2.00E-02 b	2.00E-02 a
Iron		5.00E-01 d	5.00E-01 j	
Lead				
Magnesium				
Manganese		1.00E-01 a	1.00E-01 b	
Mercury		3.00E-04 b	3.00E-04 b	
Nickel		2.00E-02 a,h	2.00E-02 b	1.00E-01 c
Potassium				
Selenium		5.00E-03 a	5.00E-03 j	
Silver		5.00E-03 a	3.00E-03 b	2.00E-02 c
Sodium				
Thallium		7.00E-05 b	7.00E-04 b	7.00E-04 c
Vanadium		7.00E-03 b	7.00E-03 b	8.00E-03 c
Zinc		2.00E-01 b	2.00E-01 b	4.00E-01 c

* Not analyzed for, used in derivation of Total Chromium toxicity values.

a. From Integrated Risk Information System (IRIS) 4/01/92.

b. From Health Effects Assessment Summary Tables (HEAST) FY 1991.

c. From Drinking Water Regulations and Health Advisories, November 1991.

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.

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 0.10 (10%) per EPA guidance (see text for specific reference). The dermal values are:

Chronic Dermal RfD: 5.00E-05 mg/kg/day
Subchronic Dermal RfD: 5.00E-05 mg/kg/day

APPENDIX C

PHYSICAL/CHEMICAL PROPERTIES FOR ORGANIC CONTAMINANTS

PHYSICAL AND CHEMICAL PROPERTIES OF DETECTED ORGANIC COMPOUNDS										
Detected Compounds	Chem. Class	Water Solubility (mg/L)	Koc Organic Carbon Partition Coeff. (mL/g)	Log Kow Octanol Water Partition Coeff.	Fish Bioconc. Factor (L/Kg)	Vapor Pressure (mm Hg)	Henry's Law Constant (atm*m3/mol)	Soil Half-Life (days)	Surface Water Half-Life (days)	Ref
Acenaphthene	PAH	3.42	4600	4	242	1.55E-03	9.20E-05			A
Acetone	VOC	1000000	2.2	-0.24		2.70E+02	2.06E-05		1.9	A
Benzene	VOC	1750	83	2.12	5.2	9.52E+01	5.59E-03		1-6	A
Benzyl alcohol	BN	800		1.10		1.10E-01	1.95E-05			H
Bis(2-ethylhexyl)phthalate	BN	0.4		8.73		2.00E-07 (F)	2.57E-07 *			E
Butanone (2-)	VOC	268000	4.5	0.26	0	7.75E+01	2.74E-05		10	A
Butylbenzyl phthalate	BN	2.9		4.8						E
Carbon disulfide	VOC	2940	54	2	0	3.60E+02	1.23E-02			A
Chloroform	VOC	8200	31	1.97	3.75	1.51E+02	2.87E-03		0.3-30	A
Chloromethane	VOC	6500	35	0.95		3.31E+03	4.40E-02			H
Chrysene	PAH	0.0018	200000	5.61		6.30E-09	1.05E-06		0.2	A
Dibenzofuran	BN			4.12						H
Dichloroethane (1,1-)	VOC	5500	30	1.79		1.82E+02	4.31E-03		1-5	A
Dichloroethylene (1,1-)	VOC	2250	65	1.84	5.6	6.00E+02	3.40E-02		1-6	A
Dichloroethylene (1,2-) **	VOC	3500	49	0.7	1.6	2.08E+02	7.58E-03		1-6	A
Dichloropropane (1,2-)	VOC	2700	51	2.00		4.21E+01	2.31E-03		1.4-7.7	A
Dimethylphthalate	BN	4320		2.12		<1.00E-02				H
Di-n-butyl phthalate	BN	13	170000	5.6		1.00E-05	2.82E-07			A
Ethylbenzene	VOC	152	1100	3.15	37.5	7.00E+00	6.43E-03		1.5-7.5	A
Fluoranthene	PAH	0.206	38000	4.9	1150	5.00E-06	6.46E-06		1-2	A
Fluorene	PAH	1.69	7300	4.2	1300	7.10E-04	6.42E-05			A
Freon-113	VOC	10		2		2.70E+02				H
Indeno(1,2,3-cd)pyrene	PAH	0.00053	1600000	6.5		1.00E-10	6.86E-08		0.02-2.08	A
Methylene chloride	VOC	20000	8.8	1.3	5	3.62E+02	2.03E-03		1.2-5.8	A
Methylnaphthalene (2-)	PAH	25.596		3.86			4.81E-04 (D)			C
Naphthalene	PAH	30.6		3.35		2.30E-01 (H)	4.48E-04 (D)			C
Nitroaniline (3-)	BN	890		1.37						H

PHYSICAL AND CHEMICAL PROPERTIES OF DETECTED ORGANIC COMPOUNDS										
Detected Compounds	Chem. Class	Water Solubility (mg/L)	Koc Organic Carbon Partition Coeff. (mL/g)	Log Kow Octanol Water Partition Coeff.	Fish Bioconc. Factor (L/Kg)	Vapor Pressure (mm Hg)	Henry's Law Constant (atm*m3/mol)	Soil Half-Life (days)	Surface Water Half-Life (days)	Ref
Phenanthrene	PAH	1	14000	4.46	2630	6.80E-04	1.59E-04		0.38-2.00	A
Pyrene	PAH	0.132	38000	4.88		2.50E-06	5.04E-06			A
Styrene	VOC	300				4.50E+00	2.05E-03			H
Tetrachloroethane (1,1,2,2-)	VOC	2900	118	2.39		5.00E+00	3.81E-04		0.04	H
Tetrachloroethylene	VOC	150	364	2.6	31	1.78E+01	2.59E-02		1.0-30.0	A
Toluene	VOC	535	300	2.73	10.7	2.81E+01	6.37E-03		0.2	A
Trichloroethane (1,1,1-)	VOC	1500	152	2.5	5.6	1.23E+02	1.44E-02		0.1-7.0	A
Trichloroethylene	VOC	1100	126	2.38	10.6	5.79E+01	9.10E-03		1.0-90.0	A
Vinyl chloride	VOC	2670	57	1.38	1.17	2.66E+03	8.19E-02		1.0-5.0	A
Xylene (total)	VOC	198	240	3.26		1.00E+01	7.04E-03		1.5-9.0	A

LEGEND:

VOC - Volatile Organic Compound
A - Acid Extractable Organic Compound
BN - Base/Neutral Extractable Organic Compound
PAH - Polycyclic Aromatic Hydrocarbon
P/PCB - Pesticide/Polychlorinated Biphenyl
* - Estimated Value
** - Properties are reported for cis-1,2-dichloroethylene

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: Treatablility Data, September, 1981.
G - Handbook of Environmental Data on Organic Chemicals, Verschueren, 1977.
H - EPA Basics of Pump-and-Treat Ground-Water Remediation Technology, March, 1990

APPENDIX D

RISK SPREADSHEETS

TABLE : D-1
SITE : TRONIC PLATING
ENV. MEDIUM : GROUND WATER
EXPOSURE TYPE : INGESTION/WORKER
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 -1 (mg/kg/day)	Increased CA Risk
1,1,1-Trichloroethane	0.014	1	250	25	70	25550	4.97E-05		0.00E+00
1,1-Dichloroethane	0.002	1	250	25	70	25550	6.99E-06		0.00E+00
1,1-Dichloroethylene	0.004	1	250	25	70	25550	1.27E-05	6.00E-01	7.64E-06
1,2-Dichloroethylene (total)	0.004	1	250	25	70	25550	1.52E-05		0.00E+00
Acetone	0.025	1	250	25	70	25550	8.73E-05		0.00E+00
Tetrachloroethylene	0.008	1	250	25	70	25550	2.84E-05	5.10E-02	1.45E-06
Toluene	0.004	1	250	25	70	25550	1.23E-05		0.00E+00
Trichloroethylene	0.289	1	250	25	70	25550	1.01E-03	1.10E-02	1.11E-05
Freon-113	0.022	1	250	25	70	25550	7.70E-05		0.00E+00
bis(2-Ethylhexyl)phthalate	0.009	1	250	25	70	25550	3.16E-05	1.40E-02	4.42E-07
Aluminum	21.400	1	250	25	70	25550	7.48E-02		0.00E+00
Antimony	0.027	1	250	25	70	25550	9.52E-05		0.00E+00
Arsenic	0.005	1	250	25	70	25550	1.66E-05	1.75E+00	2.91E-05
Barium	0.107	1	250	25	70	25550	3.75E-04		0.00E+00
Beryllium	0.003	1	250	25	70	25550	8.74E-06	4.30E+00	3.76E-05
Cadmium	0.005	1	250	25	70	25550	1.87E-05		0.00E+00
Chromium, VI	0.068	1	250	25	70	25550	2.39E-04		0.00E+00
Chromium, total	0.026	1	250	25	70	25550	9.21E-05		0.00E+00
Cobalt	0.010	1	250	25	70	25550	3.66E-05		0.00E+00
Copper	0.073	1	250	25	70	25550	2.53E-04		0.00E+00
Cyanide	0.009	1	250	25	70	25550	3.11E-05		0.00E+00
Iron	0.931	1	250	25	70	25550	3.25E-03		0.00E+00
Lead	0.038	1	250	25	70	25550	1.32E-04		0.00E+00
Manganese	1.131	1	250	25	70	25550	3.95E-03		0.00E+00
Nickel	0.040	1	250	25	70	25550	1.38E-04		0.00E+00
Selenium	0.003	1	250	25	70	25550	9.96E-06		0.00E+00
Silver	0.009	1	250	25	70	25550	3.12E-05		0.00E+00
Thallium	0.003	1	250	25	70	25550	9.78E-06		0.00E+00
Vanadium	0.015	1	250	25	70	25550	5.16E-05		0.00E+00
Zinc	0.091	1	250	25	70	25550	3.17E-04		0.00E+00

TOTAL RISK: 8.73E-05

TABLE : D-2
SITE : TRONIC PLATING
ENV. MEDIUM : GROUND WATER
EXPOSURE TYPE : INGESTION/FUTURE/WORKER
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)	Acute Body Dose (mg/kg/day)	Chronic Body Dose (mg/kg/day)	Acute Protective Body Dose (mg/kg/day)	Chronic Protective Body Dose (mg/kg/day)	Acute Hazard Quotient	Chronic Hazard Quotient
1,1,1-Trichloroethane	0.014	1	250	25	70	9125	2.03E-04	1.39E-04	1.00E+01	9.00E-02	2.03E-05	1.55E-03
1,1-Dichloroethane	0.002	1	250	25	70	9125	2.86E-05	1.96E-05		1.00E-01		1.96E-04
1,1-Dichloroethylene	0.004	1	250	25	70	9125	5.21E-05	3.57E-05	2.00E-01	9.00E-03	2.60E-04	3.96E-03
1,2-Dichloroethylene (total)	0.004	1	250	25	70	9125	6.21E-05	4.26E-05	4.00E-01	1.00E-02	1.55E-04	4.26E-03
Acetone	0.025	1	250	25	70	9125	3.57E-04	2.44E-04		1.00E-01		2.44E-03
Tetrachloroethylene	0.008	1	250	25	70	9125	1.16E-04	7.94E-05	2.00E-01	1.00E-02	5.80E-04	7.94E-03
Toluene	0.004	1	250	25	70	9125	5.03E-05	3.45E-05	2.00E+00	2.00E-01	2.52E-05	1.72E-04
Trichloroethylene	0.289	1	250	25	70	9125	4.13E-03	2.83E-03		6.00E-03		4.71E-01
Freon-113	0.022	1	250	25	70	9125	3.15E-04	2.16E-04		3.00E+01		7.19E-06
bis(2-Ethylhexyl)phthalate	0.009	1	250	25	70	9125	1.29E-04	8.84E-05		2.00E-02		4.42E-03
Aluminum	21.400	1	250	25	70	9125	3.06E-01	2.09E-01		1.00E+00		2.09E-01
Antimony	0.027	1	250	25	70	9125	3.89E-04	2.67E-04	1.50E-03	4.00E-04	2.59E-01	6.66E-01
Arsenic	0.005	1	250	25	70	9125	6.80E-05	4.66E-05		3.00E-04		1.55E-01
Barium	0.107	1	250	25	70	9125	1.54E-03	1.05E-03		5.00E-02		2.10E-02
Beryllium	0.003	1	250	25	70	9125	3.57E-05	2.45E-05	3.00E+00	5.00E-03	1.19E-05	4.90E-03
Cadmium	0.005	1	250	25	70	9125	7.66E-05	5.24E-05	4.00E-03	5.00E-04	1.91E-02	1.05E-01
Chromium, VI	0.068	1	250	25	70	9125	9.79E-04	6.70E-04		5.00E-03		1.34E-01
Chromium, total	0.026	1	250	25	70	9125	3.76E-04	2.58E-04	1.40E-01	8.76E-01	2.69E-03	2.94E-04
Cobalt	0.010	1	250	25	70	9125	1.49E-04	1.02E-04				
Copper	0.073	1	250	25	70	9125	1.04E-03	7.09E-04		4.00E-02		1.77E-02
Cyanide	0.009	1	250	25	70	9125	1.27E-04	8.70E-05	2.00E-02	2.00E-02	6.35E-03	4.35E-03
Iron	0.931	1	250	25	70	9125	1.33E-02	9.11E-03		5.00E-01		1.82E-02
Lead	0.038	1	250	25	70	9125	5.41E-04	3.70E-04				
Manganese	1.131	1	250	25	70	9125	1.62E-02	1.11E-02		1.00E-01		1.11E-01
Nickel	0.040	1	250	25	70	9125	5.66E-04	3.88E-04	1.00E-01	2.00E-02	5.66E-03	1.94E-02
Selenium	0.003	1	250	25	70	9125	4.07E-05	2.79E-05		5.00E-03		5.58E-03
Silver	0.009	1	250	25	70	9125	1.28E-04	8.75E-05	2.00E-02	5.00E-03	6.39E-03	1.75E-02
Thallium	0.003	1	250	25	70	9125	4.00E-05	2.74E-05	7.00E-04	7.00E-05	5.71E-02	3.91E-01
Vanadium	0.015	1	250	25	70	9125	2.11E-04	1.44E-04	8.00E-03	7.00E-03	2.64E-02	2.06E-02
Zinc	0.091	1	250	25	70	9125	1.30E-03	8.87E-04	4.00E-01	2.00E-01	3.24E-03	4.44E-03
										HAZARD INDEX:	3.87E-01	2.40E+00

TABLE : D-3
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP A)
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 -1 (mg/kg/day)	Increased CA Risk
Acetone	0.030	480	1.0E-06	1	65	1	70	25550	5.21E-10		0.00E+00
Styrene	0.005	480	1.0E-06	1	65	1	70	25550	8.18E-11	3.00E-02	2.45E-12
Freon-113	0.046	480	1.0E-06	1	65	1	70	25550	8.02E-10		0.00E+00
Total Xylenes	0.004	480	1.0E-06	1	65	1	70	25550	6.22E-11		0.00E+00
3-Nitroaniline	1.002	480	1.0E-06	1	65	1	70	25550	1.75E-08	4.00E-02	6.99E-10
bis(2-Ethylhexyl)phthalate	1.484	480	1.0E-06	1	65	1	70	25550	2.59E-08	1.40E-02	3.62E-10
Di-n-butylphthalate	1.597	480	1.0E-06	1	65	1	70	25550	2.79E-08		0.00E+00
Indeno(1,2,3-cd)pyrene	0.208	480	1.0E-06	1	65	1	70	25550	3.63E-09	5.79E+00	2.10E-08
Aluminum	2074.577	480	1.0E-06	1	65	1	70	25550	3.62E-05		0.00E+00
Antimony	6.340	480	1.0E-06	1	65	1	70	25550	1.11E-07		0.00E+00
Arsenic	0.945	480	1.0E-06	1	65	1	70	25550	1.65E-08	1.75E+00	2.89E-08
Barium	7.578	480	1.0E-06	1	65	1	70	25550	1.32E-07		0.00E+00
Cadmium	1.300	480	1.0E-06	1	65	1	70	25550	2.27E-08		0.00E+00
Chromium, VI	22.600	480	1.0E-06	1	65	1	70	25550	3.94E-07		0.00E+00
Chromium, total	5.660	480	1.0E-06	1	65	1	70	25550	9.87E-08		0.00E+00
Cobalt	2.114	480	1.0E-06	1	65	1	70	25550	3.69E-08		0.00E+00
Copper	15.609	480	1.0E-06	1	65	1	70	25550	2.72E-07		0.00E+00
Cyanide	7.790	480	1.0E-06	1	65	1	70	25550	1.36E-07		0.00E+00
Iron	5130.242	480	1.0E-06	1	65	1	70	25550	8.95E-05		0.00E+00
Lead	7.009	480	1.0E-06	1	65	1	70	25550	1.22E-07		0.00E+00
Manganese	83.639	480	1.0E-06	1	65	1	70	25550	1.46E-06		0.00E+00
Nickel	3.180	480	1.0E-06	1	65	1	70	25550	5.55E-08		0.00E+00
Silver	2.892	480	1.0E-06	1	65	1	70	25550	5.04E-08		0.00E+00
Vanadium	7.738	480	1.0E-06	1	65	1	70	25550	1.35E-07		0.00E+00
Zinc	15.099	480	1.0E-06	1	65	1	70	25550	2.63E-07		0.00E+00
								TOTAL RISK			5.10E-08

TABLE : D-4
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP A)
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)	Acute Body Dose (mg/kg/day)	Subchronic Body Dose (mg/kg/day)	Acute Protective Body Dose (mg/kg/day)	Subchronic Protective Body Dose (mg/kg/day)	Acute Hazard Quotient	Subchronic Hazard Quotient
Acetone	0.030	480	1.0E-06	1	65	1	70	91	2.05E-07	1.46E-07		1.00E+00		1.46E-07
Styrene	0.005	480	1.0E-06	1	65	1	70	91	3.22E-08	2.30E-08	2.00E+00	2.00E+00	1.61E-08	1.15E-08
Freon-113	0.046	480	1.0E-06	1	65	1	70	91	3.15E-07	2.25E-07		3.00E+00		7.51E-08
Total Xylenes	0.004	480	1.0E-06	1	65	1	70	91	2.44E-08	1.75E-08	4.00E+00	4.00E+00	6.11E-09	4.37E-09
3-Nitroaniline	1.002	480	1.0E-06	1	65	1	70	91	6.87E-06	4.91E-06		3.00E-04		1.64E-02
bis(2-Ethylhexyl)phthalate	1.484	480	1.0E-06	1	65	1	70	91	1.02E-05	7.27E-06		2.00E-02		3.63E-04
Di-n-butylphthalate	1.597	480	1.0E-06	1	65	1	70	91	1.10E-05	7.82E-06		1.00E+00		7.82E-06
Indeno(1,2,3-cd)pyrene	0.208	480	1.0E-06	1	65	1	70	91	1.43E-06	1.02E-06				
Aluminum	2074.577	480	1.0E-06	1	65	1	70	91	1.42E-02	1.02E-02		1.00E+00		1.02E-02
Antimony	6.340	480	1.0E-06	1	65	1	70	91	4.35E-05	3.11E-05	1.50E-03	4.00E-04	2.90E-02	7.76E-02
Arsenic	0.945	480	1.0E-06	1	65	1	70	91	6.48E-06	4.63E-06		1.00E-03		4.63E-03
Barium	7.578	480	1.0E-06	1	65	1	70	91	5.20E-05	3.71E-05		5.00E-02		7.42E-04
Cadmium	1.300	480	1.0E-06	1	65	1	70	91	8.91E-06	6.37E-06	4.00E-03	1.00E-03	2.23E-03	6.37E-03
Chromium, VI	22.600	480	1.0E-06	1	65	1	70	91	1.55E-04	1.11E-04		2.00E-02		5.53E-03
Chromium, total	5.660	480	1.0E-06	1	65	1	70	91	3.88E-05	2.77E-05	1.40E-01	8.75E+00	2.77E-04	3.17E-06
Cobalt	2.114	480	1.0E-06	1	65	1	70	91	1.45E-05	1.04E-05				
Copper	15.609	480	1.0E-06	1	65	1	70	91	1.07E-04	7.65E-05		4.00E-02		1.91E-03
Cyanide	7.790	480	1.0E-06	1	65	1	70	91	5.34E-05	3.82E-05	2.00E-02	2.00E-02	2.67E-03	1.91E-03
Iron	5130.242	480	1.0E-06	1	65	1	70	91	3.52E-02	2.51E-02		5.00E-01		5.03E-02
Lead	7.009	480	1.0E-06	1	65	1	70	91	4.81E-05	3.43E-05				
Manganese	83.639	480	1.0E-06	1	65	1	70	91	5.74E-04	4.10E-04		1.00E-01		4.10E-03
Nickel	3.180	480	1.0E-06	1	65	1	70	91	2.18E-05	1.56E-05	1.00E-01	2.00E-02	2.18E-04	7.79E-04
Silver	2.892	480	1.0E-06	1	65	1	70	91	1.98E-05	1.42E-05	2.00E-02	3.00E-03	9.91E-04	4.72E-03
Vanadium	7.738	480	1.0E-06	1	65	1	70	91	5.31E-05	3.79E-05	8.00E-03	7.00E-03	6.63E-03	5.41E-03
Zinc	15.099	480	1.0E-06	1	65	1	70	91	1.04E-04	7.40E-05	4.00E-01	2.00E-01	2.59E-04	3.70E-04
										HAZARD INDEX			4.23E-02	1.91E-01

TABLE : D-5
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP A)
EXPOSURE TYPE : INGESTION/PRESENT & FUTURE/UTILITY 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 -1 (mg/kg/day)	Increased CA Risk
Acetone	0.030	100	1.0E-06	1	10	25	70	25550	4.17E-10		0.00E+00
Styrene	0.005	100	1.0E-06	1	10	25	70	25550	6.55E-11	3.00E-02	1.97E-12
Freon-113	0.046	100	1.0E-06	1	10	25	70	25550	6.43E-10		0.00E+00
Total Xylenes	0.004	100	1.0E-06	1	10	25	70	25550	4.98E-11		0.00E+00
3-Nitroaniline	1.002	100	1.0E-06	1	10	25	70	25550	1.40E-08	4.00E-02	5.60E-10
bis(2-Ethylhexyl)phthalate	1.484	100	1.0E-06	1	10	25	70	25550	2.07E-08	1.40E-02	2.90E-10
Di-n-butylphthalate	1.597	100	1.0E-06	1	10	25	70	25550	2.23E-08		0.00E+00
Indeno(1,2,3-cd)pyrene	0.208	100	1.0E-06	1	10	25	70	25550	2.91E-09	5.79E+00	1.69E-08
Aluminum	2074.577	100	1.0E-06	1	10	25	70	25550	2.90E-05		0.00E+00
Antimony	6.340	100	1.0E-06	1	10	25	70	25550	8.86E-08		0.00E+00
Arsenic	0.945	100	1.0E-06	1	10	25	70	25550	1.32E-08	1.75E+00	2.31E-08
Barium	7.578	100	1.0E-06	1	10	25	70	25550	1.06E-07		0.00E+00
Cadmium	1.300	100	1.0E-06	1	10	25	70	25550	1.82E-08		0.00E+00
Chromium, VI	22.600	100	1.0E-06	1	10	25	70	25550	3.16E-07		0.00E+00
Chromium, total	5.660	100	1.0E-06	1	10	25	70	25550	7.91E-08		0.00E+00
Cobalt	2.114	100	1.0E-06	1	10	25	70	25550	2.95E-08		0.00E+00
Copper	15.609	100	1.0E-06	1	10	25	70	25550	2.18E-07		0.00E+00
Cyanide	7.790	100	1.0E-06	1	10	25	70	25550	1.09E-07		0.00E+00
Iron	5130.242	100	1.0E-06	1	10	25	70	25550	7.17E-05		0.00E+00
Lead	7.009	100	1.0E-06	1	10	25	70	25550	9.80E-08		0.00E+00
Manganese	83.639	100	1.0E-06	1	10	25	70	25550	1.17E-06		0.00E+00
Nickel	3.180	100	1.0E-06	1	10	25	70	25550	4.44E-08		0.00E+00
Silver	2.892	100	1.0E-06	1	10	25	70	25550	4.04E-08		0.00E+00
Vanadium	7.738	100	1.0E-06	1	10	25	70	25550	1.08E-07		0.00E+00
Zinc	15.099	100	1.0E-06	1	10	25	70	25550	2.11E-07		0.00E+00

TOTAL RISK	4.08E-08

TABLE : D-6
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP A)
EXPOSURE TYPE : INGESTION/PRESENT & FUTURE/UTILITY 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)	Acute Body Dose (mg/kg/day)	Chronic Body Dose (mg/kg/day)	Acute Protective Body Dose (mg/kg/day)	Chronic Protective Body Dose (mg/kg/day)	Acute Hazard Quotient	Chronic Hazard Quotient
Acetone	0.030	100	1.0E-06	1	10	25	70	9125	4.26E-08	1.17E-09		1.00E-01		1.17E-08
Styrene	0.005	100	1.0E-06	1	10	25	70	9125	6.70E-09	1.84E-10	2.00E+00	2.00E-01	3.35E-09	9.18E-10
Freon-113	0.046	100	1.0E-06	1	10	25	70	9125	6.57E-08	1.80E-09		3.00E+01		6.00E-11
Total Xylenes	0.004	100	1.0E-06	1	10	25	70	9125	5.09E-09	1.40E-10	4.00E+00	2.00E+00	1.27E-09	6.98E-11
3-Nitroaniline	1.002	100	1.0E-06	1	10	25	70	9125	1.43E-06	3.92E-08		3.00E-04		1.31E-04
bis(2-Ethylhexyl)phthalate	1.484	100	1.0E-06	1	10	25	70	9125	2.12E-06	5.81E-08		2.00E-02		2.90E-06
Di-n-butylphthalate	1.597	100	1.0E-06	1	10	25	70	9125	2.28E-06	6.25E-08		1.00E-01		6.25E-07
Indeno(1,2,3-cd)pyrene	0.208	100	1.0E-06	1	10	25	70	9125	2.98E-07	8.15E-09				
Aluminum	2074.577	100	1.0E-06	1	10	25	70	9125	2.96E-03	8.12E-05		1.00E+00		8.12E-05
Antimony	6.340	100	1.0E-06	1	10	25	70	9125	9.06E-06	2.48E-07	1.50E-03	4.00E-04	6.04E-03	6.20E-04
Arsenic	0.945	100	1.0E-06	1	10	25	70	9125	1.35E-06	3.70E-08		3.00E-04		1.23E-04
Barium	7.578	100	1.0E-06	1	10	25	70	9125	1.08E-05	2.97E-07		5.00E-02		5.93E-06
Cadmium	1.300	100	1.0E-06	1	10	25	70	9125	1.86E-06	5.09E-08	4.00E-03	1.00E-03	4.64E-04	5.09E-05
Chromium, VI	22.600	100	1.0E-06	1	10	25	70	9125	3.23E-05	8.85E-07		5.00E-03		1.77E-04
Chromium, total	5.660	100	1.0E-06	1	10	25	70	9125	8.09E-06	2.22E-07	1.40E-01	8.76E-01	5.78E-05	2.53E-07
Cobalt	2.114	100	1.0E-06	1	10	25	70	9125	3.02E-06	8.27E-08				
Copper	15.609	100	1.0E-06	1	10	25	70	9125	2.23E-05	6.11E-07		4.00E-02		1.53E-05
Cyanide	7.790	100	1.0E-06	1	10	25	70	9125	1.11E-05	3.05E-07	2.00E-02	2.00E-02	5.56E-04	1.52E-05
Iron	5130.242	100	1.0E-06	1	10	25	70	9125	7.33E-03	2.01E-04		5.00E-01		4.02E-04
Lead	7.009	100	1.0E-06	1	10	25	70	9125	1.00E-05	2.74E-07				
Manganese	83.639	100	1.0E-06	1	10	25	70	9125	1.19E-04	3.27E-06		1.00E-01		3.27E-05
Nickel	3.180	100	1.0E-06	1	10	25	70	9125	4.54E-06	1.24E-07	1.00E-01	2.00E-02	4.54E-05	6.22E-06
Silver	2.892	100	1.0E-06	1	10	25	70	9125	4.13E-06	1.13E-07	2.00E-02	5.00E-03	2.07E-04	2.26E-05
Vanadium	7.738	100	1.0E-06	1	10	25	70	9125	1.11E-05	3.03E-07	8.00E-03	7.00E-03	1.38E-03	4.33E-05
Zinc	15.099	100	1.0E-06	1	10	25	70	9125	2.16E-05	5.91E-07	4.00E-01	2.00E-01	5.39E-05	2.95E-06
												HAZARD INDEX	8.80E-03	1.73E-03

TABLE : D-7
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP A)
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)	Absorption 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	1.300	1.0E-06	3120	0.6	0.005	65	1	70	. 91	1.24E-07	5.00E-05	2.48E-03

TABLE : D-8
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP A)
EXPOSURE TYPE : DERMAL CONTACT/PRESENT & FUTURE/UTILITY WORKER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	Concentration (mg/kg)	kg/mg	Skin Surface Area Contacted (cm2/day)	Soil Skin Adherence Factor (mg/cm2)	Absorption 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	1.300	1.0E-06	3120	0.6	0.005	10	25	70	9125	4.76E-09	5.00E-05	9.52E-05

TABLE : D-9
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP B)
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 -1 (mg/kg/day)	Increased CA Risk
Acetone	0.024	480	1.0E-06	1	65	1	70	25550	4.14E-10		0.00E+00
Methylene Chloride	0.006	480	1.0E-06	1	65	1	70	25550	1.09E-10	7.50E-03	8.16E-13
Styrene	0.004	480	1.0E-06	1	65	1	70	25550	7.61E-11	3.00E-02	2.28E-12
Freon-113	0.046	480	1.0E-06	1	65	1	70	25550	8.02E-10		0.00E+00
Total Xylenes	0.003	480	1.0E-06	1	65	1	70	25550	5.78E-11		0.00E+00
3-Nitroaniline	1.002	480	1.0E-06	1	65	1	70	25550	1.75E-08	4.00E-02	6.99E-10
bis(2-Ethylhexyl)phthalate	1.484	480	1.0E-06	1	65	1	70	25550	2.59E-08	1.40E-02	3.62E-10
Di-n-butylphthalate	1.597	480	1.0E-06	1	65	1	70	25550	2.79E-08		0.00E+00
Indeno(1,2,3-cd)pyrene	0.208	480	1.0E-06	1	65	1	70	25550	3.63E-09	5.79E+00	2.10E-08
Aluminum	1814.767	480	1.0E-06	1	65	1	70	25550	3.17E-05		0.00E+00
Antimony	6.193	480	1.0E-06	1	65	1	70	25550	1.08E-07		0.00E+00
Arsenic	0.899	480	1.0E-06	1	65	1	70	25550	1.57E-08	1.75E+00	2.75E-08
Barium	7.409	480	1.0E-06	1	65	1	70	25550	1.29E-07		0.00E+00
Cadmium	2.071	480	1.0E-06	1	65	1	70	25550	3.61E-08		0.00E+00
Chromium, VI	22.600	480	1.0E-06	1	65	1	70	25550	3.94E-07		0.00E+00
Chromium, total	5.890	480	1.0E-06	1	65	1	70	25550	1.03E-07		0.00E+00
Cobalt	1.907	480	1.0E-06	1	65	1	70	25550	3.33E-08		0.00E+00
Copper	16.068	480	1.0E-06	1	65	1	70	25550	2.80E-07		0.00E+00
Cyanide	5.869	480	1.0E-06	1	65	1	70	25550	1.02E-07		0.00E+00
Iron	4611.941	480	1.0E-06	1	65	1	70	25550	8.05E-05		0.00E+00
Lead	14.883	480	1.0E-06	1	65	1	70	25550	2.60E-07		0.00E+00
Manganese	72.169	480	1.0E-06	1	65	1	70	25550	1.26E-06		0.00E+00
Nickel	4.109	480	1.0E-06	1	65	1	70	25550	7.17E-08		0.00E+00
Silver	2.332	480	1.0E-06	1	65	1	70	25550	4.07E-08		0.00E+00
Vanadium	6.362	480	1.0E-06	1	65	1	70	25550	1.11E-07		0.00E+00
Zinc	20.970	480	1.0E-06	1	65	1	70	25550	3.66E-07		0.00E+00
								TOTAL RISK			4.96E-08

TABLE : D-10
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP B)
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)	Acute Body Dose (mg/kg/day)	Subchronic Body Dose (mg/kg/day)	Acute Protective Body Dose (mg/kg/day)	Subchronic Protective Body Dose (mg/kg/day)	Acute Hazard Quotient	Subchronic Hazard Quotient
Acetone	0.024	480	1.0E-06	1	65	1	70	91	1.63E-07	1.16E-07		1.00E+00		1.16E-07
Methylene Chloride	0.006	480	1.0E-06	1	65	1	70	91	4.28E-08	3.05E-08	1.33E+00	6.00E-02	3.21E-08	5.09E-07
Styrene	0.004	480	1.0E-06	1	65	1	70	91	2.99E-08	2.14E-08	2.00E+00	2.00E+00	1.49E-08	1.07E-08
Freon-113	0.046	480	1.0E-06	1	65	1	70	91	3.15E-07	2.25E-07		3.00E+00		7.51E-08
Total Xylenes	0.003	480	1.0E-06	1	65	1	70	91	2.27E-08	1.62E-08	4.00E+00	4.00E+00	5.68E-09	4.06E-09
3-Nitroaniline	1.002	480	1.0E-06	1	65	1	70	91	6.87E-06	4.91E-06		3.00E-04		1.64E-02
bis(2-Ethylhexyl)phthalate	1.484	480	1.0E-06	1	65	1	70	91	1.02E-05	7.27E-06		2.00E-02		3.63E-04
Di-n-butylphthalate	1.597	480	1.0E-06	1	65	1	70	91	1.10E-05	7.82E-06		1.00E+00		7.82E-06
Indeno(1,2,3-cd)pyrene	0.208	480	1.0E-06	1	65	1	70	91	1.43E-06	1.02E-06				
Aluminum	1814.767	480	1.0E-06	1	65	1	70	91	1.24E-02	8.89E-03		1.00E+00		8.89E-03
Antimony	6.193	480	1.0E-06	1	65	1	70	91	4.25E-05	3.03E-05	1.50E-03	4.00E-04	2.83E-02	7.58E-02
Arsenic	0.899	480	1.0E-06	1	65	1	70	91	6.17E-06	4.40E-06		1.00E-03		4.40E-03
Barium	7.409	480	1.0E-06	1	65	1	70	91	5.08E-05	3.63E-05		5.00E-02		7.26E-04
Cadmium	2.071	480	1.0E-06	1	65	1	70	91	1.42E-05	1.01E-05	4.00E-03	1.00E-03	3.55E-03	1.01E-02
Chromium, VI	22.600	480	1.0E-06	1	65	1	70	91	1.55E-04	1.11E-04		2.00E-02		5.53E-03
Chromium, total	5.890	480	1.0E-06	1	65	1	70	91	4.04E-05	2.88E-05	1.40E-01	8.75E+00	2.88E-04	3.30E-06
Cobalt	1.907	480	1.0E-06	1	65	1	70	91	1.31E-05	9.34E-06				
Copper	16.068	480	1.0E-06	1	65	1	70	91	1.10E-04	7.87E-05		4.00E-02		1.97E-03
Cyanide	5.869	480	1.0E-06	1	65	1	70	91	4.02E-05	2.87E-05	2.00E-02	2.00E-02	2.01E-03	1.44E-03
Iron	4611.941	480	1.0E-06	1	65	1	70	91	3.16E-02	2.26E-02		5.00E-01		4.52E-02
Lead	14.883	480	1.0E-06	1	65	1	70	91	1.02E-04	7.29E-05				
Manganese	72.169	480	1.0E-06	1	65	1	70	91	4.95E-04	3.53E-04		1.00E-01		3.53E-03
Nickel	4.109	480	1.0E-06	1	65	1	70	91	2.82E-05	2.01E-05	1.00E-01	2.00E-02	2.82E-04	1.01E-03
Silver	2.332	480	1.0E-06	1	65	1	70	91	1.60E-05	1.14E-05	2.00E-02	3.00E-03	7.99E-04	3.81E-03
Vanadium	6.362	480	1.0E-06	1	65	1	70	91	4.36E-05	3.12E-05	8.00E-03	7.00E-03	5.45E-03	4.45E-03
Zinc	20.970	480	1.0E-06	1	65	1	70	91	1.44E-04	1.03E-04	4.00E-01	2.00E-01	3.59E-04	5.14E-04
											HAZARD INDEX		4.11E-02	1.84E-01

TABLE : D-11
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP B)
EXPOSURE TYPE : INGESTION/PRESENT & FUTURE/UTILITY 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 -1 (mg/kg/day)	Increased CA Risk
Acetone	0.024	100	1.0E-06	1	10	25	70	25550	3.32E-10		0.00E+00
Methylene Chloride	0.006	100	1.0E-06	1	10	25	70	25550	8.72E-11	7.50E-03	6.54E-13
Styrene	0.004	100	1.0E-06	1	10	25	70	25550	6.09E-11	3.00E-02	1.83E-12
Freon-113	0.046	100	1.0E-06	1	10	25	70	25550	6.43E-10		0.00E+00
Total Xylenes	0.003	100	1.0E-06	1	10	25	70	25550	4.63E-11		0.00E+00
3-Nitroaniline	1.002	100	1.0E-06	1	10	25	70	25550	1.40E-08	4.00E-02	5.60E-10
bis(2-Ethylhexyl)phthalate	1.484	100	1.0E-06	1	10	25	70	25550	2.07E-08	1.40E-02	2.90E-10
Di-n-butylphthalate	1.597	100	1.0E-06	1	10	25	70	25550	2.23E-08		0.00E+00
Indeno(1,2,3-cd)pyrene	0.208	100	1.0E-06	1	10	25	70	25550	2.91E-09	5.79E+00	1.69E-08
Aluminum	1814.767	100	1.0E-06	1	10	25	70	25550	2.54E-05		0.00E+00
Antimony	6.193	100	1.0E-06	1	10	25	70	25550	8.66E-08		0.00E+00
Arsenic	0.899	100	1.0E-06	1	10	25	70	25550	1.26E-08	1.75E+00	2.20E-08
Barium	7.409	100	1.0E-06	1	10	25	70	25550	1.04E-07		0.00E+00
Cadmium	2.071	100	1.0E-06	1	10	25	70	25550	2.90E-08		0.00E+00
Chromium, VI	22.600	100	1.0E-06	1	10	25	70	25550	3.16E-07		0.00E+00
Chromium, total	5.890	100	1.0E-06	1	10	25	70	25550	8.23E-08		0.00E+00
Cobalt	1.907	100	1.0E-06	1	10	25	70	25550	2.67E-08		0.00E+00
Copper	16.068	100	1.0E-06	1	10	25	70	25550	2.25E-07		0.00E+00
Cyanide	5.869	100	1.0E-06	1	10	25	70	25550	8.20E-08		0.00E+00
Iron	4611.941	100	1.0E-06	1	10	25	70	25550	6.45E-05		0.00E+00
Lead	14.883	100	1.0E-06	1	10	25	70	25550	2.08E-07		0.00E+00
Manganese	72.169	100	1.0E-06	1	10	25	70	25550	1.01E-06		0.00E+00
Nickel	4.109	100	1.0E-06	1	10	25	70	25550	5.74E-08		0.00E+00
Silver	2.332	100	1.0E-06	1	10	25	70	25550	3.26E-08		0.00E+00
Vanadium	6.362	100	1.0E-06	1	10	25	70	25550	8.89E-08		0.00E+00
Zinc	20.970	100	1.0E-06	1	10	25	70	25550	2.93E-07		0.00E+00

TOTAL RISK	3.97E-08

TABLE : D-12
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP B)
EXPOSURE TYPE : INGESTION/PRESENT & FUTURE/UTILITY 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)	Acute Body Dose (mg/kg/day)	Chronic Body Dose (mg/kg/day)	Acute Protective Body Dose (mg/kg/day)	Chronic Protective Body Dose (mg/kg/day)	Acute Hazard Quotient	Chronic Hazard Quotient
Acetone	0.024	100	1.0E-06	1	10	25	70	9125	3.39E-08	9.28E-10		1.00E-01		9.28E-09
Methylene Chloride	0.006	100	1.0E-06	1	10	25	70	9125	8.91E-09	2.44E-10	1.33E+00	6.00E-02	6.70E-09	4.07E-09
Styrene	0.004	100	1.0E-06	1	10	25	70	9125	6.23E-09	1.71E-10	2.00E+00	2.00E-01	3.11E-09	8.53E-10
Freon-113	0.046	100	1.0E-06	1	10	25	70	9125	6.57E-08	1.80E-09		3.00E+01		6.00E-11
Total Xylenes	0.003	100	1.0E-06	1	10	25	70	9125	4.74E-09	1.30E-10	4.00E+00	2.00E+00	1.18E-09	6.49E-11
3-Nitroaniline	1.002	100	1.0E-06	1	10	25	70	9125	1.43E-06	3.92E-08		3.00E-04		1.31E-04
bis(2-Ethylhexyl)phthalate	1.484	100	1.0E-06	1	10	25	70	9125	2.12E-06	5.81E-08		2.00E-02		2.90E-06
Di-n-butylphthalate	1.597	100	1.0E-06	1	10	25	70	9125	2.28E-06	6.25E-08		1.00E-01		6.25E-07
Indeno(1,2,3-cd)pyrene	0.208	100	1.0E-06	1	10	25	70	9125	2.98E-07	8.15E-09				
Aluminum	1814.767	100	1.0E-06	1	10	25	70	9125	2.59E-03	7.10E-05		1.00E+00		7.10E-05
Antimony	6.193	100	1.0E-06	1	10	25	70	9125	8.85E-06	2.42E-07	1.50E-03	4.00E-04	5.90E-03	6.06E-04
Arsenic	0.899	100	1.0E-06	1	10	25	70	9125	1.28E-06	3.52E-08		3.00E-04		1.17E-04
Barium	7.409	100	1.0E-06	1	10	25	70	9125	1.06E-05	2.90E-07		5.00E-02		5.80E-06
Cadmium	2.071	100	1.0E-06	1	10	25	70	9125	2.96E-06	8.11E-08	4.00E-03	1.00E-03	7.40E-04	8.11E-05
Chromium, VI	22.600	100	1.0E-06	1	10	25	70	9125	3.23E-05	8.85E-07		5.00E-03		1.77E-04
Chromium, total	5.890	100	1.0E-06	1	10	25	70	9125	8.41E-06	2.31E-07	1.40E-01	8.76E-01	6.01E-05	2.63E-07
Cobalt	1.907	100	1.0E-06	1	10	25	70	9125	2.72E-06	7.47E-08				
Copper	16.068	100	1.0E-06	1	10	25	70	9125	2.30E-05	6.29E-07		4.00E-02		1.57E-05
Cyanide	5.869	100	1.0E-06	1	10	25	70	9125	8.38E-06	2.30E-07	2.00E-02	2.00E-02	4.19E-04	1.15E-05
Iron	4611.941	100	1.0E-06	1	10	25	70	9125	6.59E-03	1.81E-04		5.00E-01		3.61E-04
Lead	14.883	100	1.0E-06	1	10	25	70	9125	2.13E-05	5.83E-07				
Manganese	72.169	100	1.0E-06	1	10	25	70	9125	1.03E-04	2.82E-06		1.00E-01		2.82E-05
Nickel	4.109	100	1.0E-06	1	10	25	70	9125	5.87E-06	1.61E-07	1.00E-01	2.00E-02	5.87E-05	8.04E-06
Silver	2.332	100	1.0E-06	1	10	25	70	9125	3.33E-06	9.13E-08	2.00E-02	5.00E-03	1.67E-04	1.83E-05
Vanadium	6.362	100	1.0E-06	1	10	25	70	9125	9.09E-06	2.49E-07	8.00E-03	7.00E-03	1.14E-03	3.56E-05
Zinc	20.970	100	1.0E-06	1	10	25	70	9125	3.00E-05	8.21E-07	4.00E-01	2.00E-01	7.49E-05	4.10E-06
												HAZARD INDEX	8.55E-03	1.68E-03

TABLE : D-13
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP B)
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)	Absorption 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	2.071	1.0E-06	3120	0.6	0.005	65	1	70	91	1.98E-07	5.00E-05	3.96E-03

TABLE : D-14
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP B)
EXPOSURE TYPE : DERMAL CONTACT/PRESENT & FUTURE/UTILITY WORKER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	Concentration (mg/kg)	kg/mg	Skin Surface Area Contacted (cm2/day)	Soil Skin Adherence Factor (mg/cm2)	Absorption 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	2.071	1.0E-06	3120	0.6	0.005	10	25	70	9125	7.59E-09	5.00E-05	1.52E-04

TABLE : D-15
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP C)
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 -1 (mg/kg/day)	Increased CA Risk
1,1,2,2-Tetrachloroethane	0.004	480	1.0E-06	1	65	1	70	25550	6.98E-11	2.00E-01	1.40E-11
1,2-Dichloroethylene (total)	0.006	480	1.0E-06	1	65	1	70	25550	1.13E-10		0.00E+00
Acetone	0.035	480	1.0E-06	1	65	1	70	25550	6.11E-10		0.00E+00
Chloromethane	0.008	480	1.0E-06	1	65	1	70	25550	1.40E-10	1.30E-02	1.81E-12
Ethylbenzene	0.006	480	1.0E-06	1	65	1	70	25550	1.01E-10		0.00E+00
Methylene Chloride	0.007	480	1.0E-06	1	65	1	70	25550	1.16E-10	7.50E-03	8.73E-13
Styrene	0.005	480	1.0E-06	1	65	1	70	25550	8.72E-11	3.00E-02	2.62E-12
Tetrachloroethylene	0.002	480	1.0E-06	1	65	1	70	25550	3.49E-11	5.10E-02	1.78E-12
Toluene	0.006	480	1.0E-06	1	65	1	70	25550	1.13E-10		0.00E+00
Trichloroethylene	0.011	480	1.0E-06	1	65	1	70	25550	1.87E-10	1.10E-02	2.06E-12
Freon-113	0.018	480	1.0E-06	1	65	1	70	25550	3.08E-10		0.00E+00
Vinyl Chloride	0.010	480	1.0E-06	1	65	1	70	25550	1.75E-10	1.90E+00	3.32E-10
Total Xylenes	0.028	480	1.0E-06	1	65	1	70	25550	4.90E-10		0.00E+00
2-Methylnaphthalene	3.990	480	1.0E-06	1	65	1	70	25550	6.96E-08		0.00E+00
3-Nitroaniline	1.600	480	1.0E-06	1	65	1	70	25550	2.79E-08	4.00E-02	1.12E-09
Acenaphthylene	1.098	480	1.0E-06	1	65	1	70	25550	1.91E-08		0.00E+00
bis(2-Ethylhexyl)phthalate	43.000	480	1.0E-06	1	65	1	70	25550	7.50E-07	1.40E-02	1.05E-08
Chrysene	0.811	480	1.0E-06	1	65	1	70	25550	1.41E-08	5.79E+00	8.19E-08
Di-n-butylphthalate	2.222	480	1.0E-06	1	65	1	70	25550	3.88E-08		0.00E+00
Dibenzofuran	1.029	480	1.0E-06	1	65	1	70	25550	1.80E-08		0.00E+00
Dimethylphthalate	1.195	480	1.0E-06	1	65	1	70	25550	2.08E-08		0.00E+00
Fluoranthene	1.385	480	1.0E-06	1	65	1	70	25550	2.42E-08		0.00E+00
Fluorene	1.384	480	1.0E-06	1	65	1	70	25550	2.41E-08		0.00E+00
Indeno(1,2,3-cd)pyrene	0.340	480	1.0E-06	1	65	1	70	25550	5.93E-09	5.79E+00	3.43E-08
Naphthalene	1.447	480	1.0E-06	1	65	1	70	25550	2.52E-08		0.00E+00
Phenanthrene	2.533	480	1.0E-06	1	65	1	70	25550	4.42E-08		0.00E+00
Pyrene	1.305	480	1.0E-06	1	65	1	70	25550	2.28E-08		0.00E+00
Aluminum	4276.106	480	1.0E-06	1	65	1	70	25550	7.46E-05		0.00E+00
Antimony	6.359	480	1.0E-06	1	65	1	70	25550	1.11E-07		0.00E+00
Arsenic	1.617	480	1.0E-06	1	65	1	70	25550	2.82E-08	1.75E+00	4.94E-08
Barium	7.409	480	1.0E-06	1	65	1	70	25550	1.29E-07		0.00E+00
Beryllium	0.725	480	1.0E-06	1	65	1	70	25550	1.26E-08	4.30E+00	5.44E-08
Cadmium	61.094	480	1.0E-06	1	65	1	70	25550	1.07E-06		0.00E+00
Chromium, VI	22.600	480	1.0E-06	1	65	1	70	25550	3.94E-07		0.00E+00
Chromium, total	110.548	480	1.0E-06	1	65	1	70	25550	1.93E-06		0.00E+00
Cobalt	5.041	480	1.0E-06	1	65	1	70	25550	8.79E-08		0.00E+00
Copper	1176.142	480	1.0E-06	1	65	1	70	25550	2.05E-05		0.00E+00
Cyanide	8.161	480	1.0E-06	1	65	1	70	25550	1.42E-07		0.00E+00
Iron	5417.243	480	1.0E-06	1	65	1	70	25550	9.45E-05		0.00E+00
Lead	762.975	480	1.0E-06	1	65	1	70	25550	1.33E-05		0.00E+00

TABLE : D-15
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP C)
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 -1 (mg/kg/day)	Increased CA Risk
Manganese	63.854	480	1.0E-06	1	65	1	70	25550	1.11E-06		0.00E+00
Mercury	0.041	480	1.0E-06	1	65	1	70	25550	7.18E-10		0.00E+00
Nickel	20.672	480	1.0E-06	1	65	1	70	25550	3.61E-07		0.00E+00
Selenium	0.739	480	1.0E-06	1	65	1	70	25550	1.29E-08		0.00E+00
Silver	2.332	480	1.0E-06	1	65	1	70	25550	4.07E-08		0.00E+00
Vanadium	28.342	480	1.0E-06	1	65	1	70	25550	4.94E-07		0.00E+00
Zinc	777.163	480	1.0E-06	1	65	1	70	25550	1.36E-05		0.00E+00
									TOTAL RISK		2.32E-07

TABLE : D-16
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP C)
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)	Acute Body Dose (mg/kg/day)	Subchronic Body Dose (mg/kg/day)	Acute Protective Body Dose (mg/kg/day)	Subchronic Protective Body Dose (mg/kg/day)	Acute Hazard Quotient	Subchronic Hazard Quotient
1,1,2,2-Tetrachloroethane	0.004	480	1.0E-06	1	65	1	70	91	2.74E-08	1.96E-08				
1,2-Dichloroethylene (total)	0.006	480	1.0E-06	1	65	1	70	91	4.46E-08	3.18E-08	4.00E-01	1.00E-01	1.11E-07	3.18E-07
Acetone	0.035	480	1.0E-06	1	65	1	70	91	2.40E-07	1.72E-07		1.00E+00		1.72E-07
Chloromethane	0.008	480	1.0E-06	1	65	1	70	91	5.49E-08	3.92E-08	9.00E-01		6.10E-08	
Ethylbenzene	0.006	480	1.0E-06	1	65	1	70	91	3.99E-08	2.85E-08	3.20E+00	1.00E+00	1.25E-08	2.85E-08
Methylene Chloride	0.007	480	1.0E-06	1	65	1	70	91	4.57E-08	3.27E-08	1.33E+00	6.00E-02	3.44E-08	5.45E-07
Styrene	0.005	480	1.0E-06	1	65	1	70	91	3.43E-08	2.45E-08	2.00E+00	2.00E+00	1.71E-08	1.22E-08
Tetrachloroethylene	0.002	480	1.0E-06	1	65	1	70	91	1.37E-08	9.80E-09	2.00E-01	1.00E-01	6.86E-08	9.80E-08
Toluene	0.006	480	1.0E-06	1	65	1	70	91	4.43E-08	3.16E-08	2.00E+00	2.00E+00	2.21E-08	1.58E-08
Trichloroethylene	0.011	480	1.0E-06	1	65	1	70	91	7.37E-08	5.26E-08		6.00E-03		8.77E-06
Freon-113	0.018	480	1.0E-06	1	65	1	70	91	1.21E-07	8.64E-08		3.00E+00		2.88E-08
Vinyl Chloride	0.010	480	1.0E-06	1	65	1	70	91	6.86E-08	4.90E-08	3.00E-01		2.29E-07	
Total Xylenes	0.028	480	1.0E-06	1	65	1	70	91	1.93E-07	1.38E-07	4.00E+00	4.00E+00	4.81E-08	3.44E-08
2-Methylnaphthalene	3.990	480	1.0E-06	1	65	1	70	91	2.74E-05	1.95E-05				
3-Nitroaniline	1.600	480	1.0E-06	1	65	1	70	91	1.10E-05	7.84E-06		3.00E-04		2.61E-02
Acenaphthylene	1.098	480	1.0E-06	1	65	1	70	91	7.53E-06	5.38E-06				
bis(2-Ethylhexyl)phthalate	43.000	480	1.0E-06	1	65	1	70	91	2.95E-04	2.11E-04		2.00E-02		1.05E-02
Chrysene	0.811	480	1.0E-06	1	65	1	70	91	5.56E-06	3.97E-06				
Di-n-butylphthalate	2.222	480	1.0E-06	1	65	1	70	91	1.52E-05	1.09E-05		1.00E+00		1.09E-05
Dibenzofuran	1.029	480	1.0E-06	1	65	1	70	91	7.06E-06	5.04E-06		4.00E-03		1.26E-03
Dimethylphthalate	1.195	480	1.0E-06	1	65	1	70	91	8.19E-06	5.85E-06		1.00E+00		5.85E-06
Fluoranthene	1.385	480	1.0E-06	1	65	1	70	91	9.50E-06	6.78E-06		4.00E-01		1.70E-05
Fluorene	1.384	480	1.0E-06	1	65	1	70	91	9.49E-06	6.78E-06		4.00E-01		1.69E-05
Indeno(1,2,3-cd)pyrene	0.340	480	1.0E-06	1	65	1	70	91	2.33E-06	1.67E-06				
Naphthalene	1.447	480	1.0E-06	1	65	1	70	91	9.92E-06	7.09E-06	5.00E-02	4.00E-02	1.98E-04	1.77E-04
Phenanthrene	2.533	480	1.0E-06	1	65	1	70	91	1.74E-05	1.24E-05				
Pyrene	1.305	480	1.0E-06	1	65	1	70	91	8.95E-06	6.39E-06		3.00E-01		2.13E-05
Aluminum	4276.106	480	1.0E-06	1	65	1	70	91	2.93E-02	2.09E-02		1.00E+00		2.09E-02
Antimony	6.359	480	1.0E-06	1	65	1	70	91	4.36E-05	3.11E-05	1.50E-03	4.00E-04	2.91E-02	7.79E-02
Arsenic	1.617	480	1.0E-06	1	65	1	70	91	1.11E-05	7.92E-06		1.00E-03		7.92E-03
Barium	7.409	480	1.0E-06	1	65	1	70	91	5.08E-05	3.63E-05		5.00E-02		7.26E-04
Beryllium	0.725	480	1.0E-06	1	65	1	70	91	4.97E-06	3.55E-06	3.00E+00	5.00E-03	1.66E-06	7.10E-04
Cadmium	61.094	480	1.0E-06	1	65	1	70	91	4.19E-04	2.99E-04	4.00E-03	1.00E-03	1.05E-01	2.99E-01
Chromium, VI	22.600	480	1.0E-06	1	65	1	70	91	1.55E-04	1.11E-04		2.00E-02		5.53E-03
Chromium, total	110.548	480	1.0E-06	1	65	1	70	91	7.58E-04	5.41E-04	1.40E-01	8.75E+00	5.41E-03	6.19E-05
Cobalt	5.041	480	1.0E-06	1	65	1	70	91	3.46E-05	2.47E-05				
Copper	1176.142	480	1.0E-06	1	65	1	70	91	8.06E-03	5.76E-03		4.00E-02		1.44E-01
Cyanide	8.161	480	1.0E-06	1	65	1	70	91	5.60E-05	4.00E-05	2.00E-02	2.00E-02	2.80E-03	2.00E-03
Iron	5417.243	480	1.0E-06	1	65	1	70	91	3.71E-02	2.65E-02		5.00E-01		5.31E-02
Lead	762.975	480	1.0E-06	1	65	1	70	91	5.23E-03	3.74E-03				
Manganese	63.854	480	1.0E-06	1	65	1	70	91	4.38E-04	3.13E-04		1.00E-01		3.13E-03
Mercury	0.041	480	1.0E-06	1	65	1	70	91	2.82E-07	2.02E-07		3.00E-04		6.72E-04

TABLE : D-16
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP C)
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)	Acute Body Dose (mg/kg/day)	Subchronic Body Dose (mg/kg/day)	Acute Protective Body Dose (mg/kg/day)	Subchronic Protective Body Dose (mg/kg/day)	Acute Hazard Quotient	Subchronic Hazard Quotient
Nickel	20.672	480	1.0E-06	1	65	1	70	91	1.42E-04	1.01E-04	1.00E-01	2.00E-02	1.42E-03	5.06E-03
Selenium	0.739	480	1.0E-06	1	65	1	70	91	5.07E-06	3.62E-06		5.00E-03		7.24E-04
Silver	2.332	480	1.0E-06	1	65	1	70	91	1.60E-05	1.14E-05	2.00E-02	3.00E-03	7.99E-04	3.81E-03
Vanadium	28.342	480	1.0E-06	1	65	1	70	91	1.94E-04	1.39E-04	8.00E-03	7.00E-03	2.43E-02	1.98E-02
Zinc	777.163	480	1.0E-06	1	65	1	70	91	5.33E-03	3.81E-03	4.00E-01	2.00E-01	1.33E-02	1.90E-02
										HAZARD INDEX			1.82E-01	7.03E-01

TABLE : D-17
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP C)
EXPOSURE TYPE : INGESTION/PRESENT & FUTURE/UTILITY 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 -1 (mg/kg/day)	Increased CA Risk
1,1,2,2-Tetrachloroethane	0.004	100	1.0E-06	1	10	25	70	25550	5.59E-11	2.00E-01	1.12E-11
1,2-Dichloroethylene (total)	0.006	100	1.0E-06	1	10	25	70	25550	9.08E-11		0.00E+00
Acetone	0.035	100	1.0E-06	1	10	25	70	25550	4.90E-10		0.00E+00
Chloromethane	0.008	100	1.0E-06	1	10	25	70	25550	1.12E-10	1.30E-02	1.45E-12
Ethylbenzene	0.006	100	1.0E-06	1	10	25	70	25550	8.13E-11		0.00E+00
Methylene Chloride	0.007	100	1.0E-06	1	10	25	70	25550	9.33E-11	7.50E-03	6.99E-13
Styrene	0.005	100	1.0E-06	1	10	25	70	25550	6.99E-11	3.00E-02	2.10E-12
Tetrachloroethylene	0.002	100	1.0E-06	1	10	25	70	25550	2.80E-11	5.10E-02	1.43E-12
Toluene	0.006	100	1.0E-06	1	10	25	70	25550	9.02E-11		0.00E+00
Trichloroethylene	0.011	100	1.0E-06	1	10	25	70	25550	1.50E-10	1.10E-02	1.65E-12
Freon-113	0.018	100	1.0E-06	1	10	25	70	25550	2.47E-10		0.00E+00
Vinyl Chloride	0.010	100	1.0E-06	1	10	25	70	25550	1.40E-10	1.90E+00	2.66E-10
Total Xylenes	0.028	100	1.0E-06	1	10	25	70	25550	3.93E-10		0.00E+00
2-Methylnaphthalene	3.990	100	1.0E-06	1	10	25	70	25550	5.58E-08		0.00E+00
3-Nitroaniline	1.600	100	1.0E-06	1	10	25	70	25550	2.24E-08	4.00E-02	8.95E-10
Acenaphthylene	1.098	100	1.0E-06	1	10	25	70	25550	1.53E-08		0.00E+00
bis(2-Ethylhexyl)phthalate	43.000	100	1.0E-06	1	10	25	70	25550	6.01E-07	1.40E-02	8.41E-09
Chrysene	0.811	100	1.0E-06	1	10	25	70	25550	1.13E-08	5.79E+00	6.56E-08
Di-n-butylphthalate	2.222	100	1.0E-06	1	10	25	70	25550	3.11E-08		0.00E+00
Dibenzofuran	1.029	100	1.0E-06	1	10	25	70	25550	1.44E-08		0.00E+00
Dimethylphthalate	1.195	100	1.0E-06	1	10	25	70	25550	1.67E-08		0.00E+00
Fluoranthene	1.385	100	1.0E-06	1	10	25	70	25550	1.94E-08		0.00E+00
Fluorene	1.384	100	1.0E-06	1	10	25	70	25550	1.93E-08		0.00E+00
Indeno(1,2,3-cd)pyrene	0.340	100	1.0E-06	1	10	25	70	25550	4.75E-09	5.79E+00	2.75E-08
Naphthalene	1.447	100	1.0E-06	1	10	25	70	25550	2.02E-08		0.00E+00
Phenanthrene	2.533	100	1.0E-06	1	10	25	70	25550	3.54E-08		0.00E+00
Pyrene	1.305	100	1.0E-06	1	10	25	70	25550	1.82E-08		0.00E+00
Aluminum	4276.106	100	1.0E-06	1	10	25	70	25550	5.98E-05		0.00E+00
Antimony	6.359	100	1.0E-06	1	10	25	70	25550	8.89E-08		0.00E+00
Arsenic	1.617	100	1.0E-06	1	10	25	70	25550	2.26E-08	1.75E+00	3.96E-08
Barium	7.409	100	1.0E-06	1	10	25	70	25550	1.04E-07		0.00E+00
Beryllium	0.725	100	1.0E-06	1	10	25	70	25550	1.01E-08	4.30E+00	4.36E-08
Cadmium	61.094	100	1.0E-06	1	10	25	70	25550	8.54E-07		0.00E+00
Chromium, VI	22.600	100	1.0E-06	1	10	25	70	25550	3.16E-07		0.00E+00
Chromium, total	110.548	100	1.0E-06	1	10	25	70	25550	1.55E-06		0.00E+00
Cobalt	5.041	100	1.0E-06	1	10	25	70	25550	7.05E-08		0.00E+00
Copper	1176.142	100	1.0E-06	1	10	25	70	25550	1.64E-05		0.00E+00
Cyanide	8.161	100	1.0E-06	1	10	25	70	25550	1.14E-07		0.00E+00
Iron	5417.243	100	1.0E-06	1	10	25	70	25550	7.57E-05		0.00E+00

TABLE : D-17
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP C)
EXPOSURE TYPE : INGESTION/PRESENT & FUTURE/UTILITY 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 -1 (mg/kg/day)	Increased CA Risk
Lead	762.975	100	1.0E-06	1	10	25	70	25550	1.07E-05		0.00E+00
Manganese	63.854	100	1.0E-06	1	10	25	70	25550	8.93E-07		0.00E+00
Mercury	0.041	100	1.0E-06	1	10	25	70	25550	5.75E-10		0.00E+00
Nickel	20.672	100	1.0E-06	1	10	25	70	25550	2.89E-07		0.00E+00
Selenium	0.739	100	1.0E-06	1	10	25	70	25550	1.03E-08		0.00E+00
Silver	2.332	100	1.0E-06	1	10	25	70	25550	3.26E-08		0.00E+00
Vanadium	28.342	100	1.0E-06	1	10	25	70	25550	3.96E-07		0.00E+00
Zinc	777.163	100	1.0E-06	1	10	25	70	25550	1.09E-05		0.00E+00
								TOTAL RISK			1.86E-07

TABLE : D-18
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP C)
EXPOSURE TYPE : INGESTION/PRESENT & FUTURE/UTILITY 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)	Acute Body Dose (mg/kg/day)	Chronic Body Dose (mg/kg/day)	Acute Protective Body Dose (mg/kg/day)	Chronic Protective Body Dose (mg/kg/day)	Acute Hazard Quotient	Chronic Hazard Quotient
1,1,2,2-Tetrachloroethane	0.004	100	1.0E-06	1	10	25	70	9125	5.71E-09	1.57E-10				
1,2-Dichloroethylene (total)	0.006	100	1.0E-06	1	10	25	70	9125	9.28E-09	2.54E-10	4.00E-01	1.00E-02	2.32E-08	2.54E-08
Acetone	0.035	100	1.0E-06	1	10	25	70	9125	5.01E-08	1.37E-09		1.00E-01		1.37E-08
Chloromethane	0.008	100	1.0E-06	1	10	25	70	9125	1.14E-08	3.13E-10	9.00E-01		1.27E-08	
Ethylbenzene	0.006	100	1.0E-06	1	10	25	70	9125	8.31E-09	2.28E-10	3.20E+00	1.00E-01	2.60E-09	2.28E-09
Methylene Chloride	0.007	100	1.0E-06	1	10	25	70	9125	9.53E-09	2.61E-10	1.33E+00	6.00E-02	7.17E-09	4.35E-09
Styrene	0.005	100	1.0E-06	1	10	25	70	9125	7.14E-09	1.96E-10	2.00E+00	2.00E-01	3.57E-09	9.78E-10
Tetrachloroethylene	0.002	100	1.0E-06	1	10	25	70	9125	2.86E-09	7.83E-11	2.00E-01	1.00E-02	1.43E-08	7.83E-09
Toluene	0.006	100	1.0E-06	1	10	25	70	9125	9.22E-09	2.53E-10	2.00E+00	2.00E-01	4.61E-09	1.26E-09
Trichloroethylene	0.011	100	1.0E-06	1	10	25	70	9125	1.53E-08	4.20E-10		6.00E-03		7.01E-08
Freon-113	0.018	100	1.0E-06	1	10	25	70	9125	2.52E-08	6.90E-10		3.00E+01		2.30E-11
Vinyl Chloride	0.010	100	1.0E-06	1	10	25	70	9125	1.43E-08	3.92E-10	3.00E-01		4.77E-08	
Total Xylenes	0.028	100	1.0E-06	1	10	25	70	9125	4.01E-08	1.10E-09	4.00E+00	2.00E+00	1.00E-08	5.50E-10
2-Methylnaphthalene	3.990	100	1.0E-06	1	10	25	70	9125	5.70E-06	1.56E-07				
3-Nitroaniline	1.600	100	1.0E-06	1	10	25	70	9125	2.29E-06	6.26E-08		3.00E-04		2.09E-04
Acenaphthylene	1.098	100	1.0E-06	1	10	25	70	9125	1.57E-06	4.30E-08				
bis(2-Ethylhexyl)phthalate	43.000	100	1.0E-06	1	10	25	70	9125	6.14E-05	1.68E-06		2.00E-02		8.41E-05
Chrysene	0.811	100	1.0E-06	1	10	25	70	9125	1.16E-06	3.17E-08				
Di-n-butylphthalate	2.222	100	1.0E-06	1	10	25	70	9125	3.17E-06	8.70E-08		1.00E-01		8.70E-07
Dibenzofuran	1.029	100	1.0E-06	1	10	25	70	9125	1.47E-06	4.03E-08		4.00E-03		1.01E-05
Dimethylphthalate	1.195	100	1.0E-06	1	10	25	70	9125	1.71E-06	4.68E-08		1.00E+00		4.68E-08
Fluoranthene	1.385	100	1.0E-06	1	10	25	70	9125	1.98E-06	5.42E-08		4.00E-02		1.36E-06
Fluorene	1.384	100	1.0E-06	1	10	25	70	9125	1.98E-06	5.42E-08		4.00E-02		1.35E-06
Indeno(1,2,3-cd)pyrene	0.340	100	1.0E-06	1	10	25	70	9125	4.86E-07	1.33E-08				
Naphthalene	1.447	100	1.0E-06	1	10	25	70	9125	2.07E-06	5.66E-08	5.00E-02	4.00E-03	4.13E-05	1.42E-05
Phenanthrene	2.533	100	1.0E-06	1	10	25	70	9125	3.62E-06	9.91E-08				
Pyrene	1.305	100	1.0E-06	1	10	25	70	9125	1.86E-06	5.11E-08		3.00E-02		1.70E-06
Aluminum	4276.106	100	1.0E-06	1	10	25	70	9125	6.11E-03	1.67E-04		1.00E+00		1.67E-04
Antimony	6.359	100	1.0E-06	1	10	25	70	9125	9.08E-06	2.49E-07	1.50E-03	4.00E-04	6.06E-03	6.22E-04
Arsenic	1.617	100	1.0E-06	1	10	25	70	9125	2.31E-06	6.33E-08		3.00E-04		2.11E-04
Barium	7.409	100	1.0E-06	1	10	25	70	9125	1.06E-05	2.90E-07		5.00E-02		5.80E-06
Beryllium	0.725	100	1.0E-06	1	10	25	70	9125	1.04E-06	2.84E-08	3.00E+00	5.00E-03	3.45E-07	5.67E-06
Cadmium	61.094	100	1.0E-06	1	10	25	70	9125	8.73E-05	2.39E-06	4.00E-03	1.00E-03	2.18E-02	2.39E-03
Chromium, VI	22.600	100	1.0E-06	1	10	25	70	9125	3.23E-05	8.85E-07		5.00E-03		1.77E-04
Chromium, total	110.548	100	1.0E-06	1	10	25	70	9125	1.58E-04	4.33E-06	1.40E-01	8.76E-01	1.13E-03	4.94E-06
Cobalt	5.041	100	1.0E-06	1	10	25	70	9125	7.20E-06	1.97E-07				
Copper	1176.142	100	1.0E-06	1	10	25	70	9125	1.68E-03	4.60E-05		4.00E-02		1.15E-03
Cyanide	8.161	100	1.0E-06	1	10	25	70	9125	1.17E-05	3.19E-07	2.00E-02	2.00E-02	5.83E-04	1.60E-05
Iron	5417.243	100	1.0E-06	1	10	25	70	9125	7.74E-03	2.12E-04		5.00E-01		4.24E-04
Lead	762.975	100	1.0E-06	1	10	25	70	9125	1.09E-03	2.99E-05				
Manganese	63.854	100	1.0E-06	1	10	25	70	9125	9.12E-05	2.50E-06		1.00E-01		2.50E-05

TABLE : D-18
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP C)
EXPOSURE TYPE : INGESTION/PRESENT & FUTURE/UTILITY 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)	Acute Body Dose (mg/kg/day)	Chronic Body Dose (mg/kg/day)	Acute Protective Body Dose (mg/kg/day)	Chronic Protective Body Dose (mg/kg/day)	Acute Hazard Quotient	Chronic Hazard Quotient
Mercury	0.041	100	1.0E-06	1	10	25	70	9125	5.88E-08	1.61E-09		3.00E-04		5.37E-06
Nickel	20.672	100	1.0E-06	1	10	25	70	9125	2.95E-05	8.09E-07	1.00E-01	2.00E-02	2.95E-04	4.05E-05
Selenium	0.739	100	1.0E-06	1	10	25	70	9125	1.06E-06	2.89E-08		5.00E-03		5.78E-06
Silver	2.332	100	1.0E-06	1	10	25	70	9125	3.33E-06	9.13E-08	2.00E-02	5.00E-03	1.67E-04	1.83E-05
Vanadium	28.342	100	1.0E-06	1	10	25	70	9125	4.05E-05	1.11E-06	8.00E-03	7.00E-03	5.06E-03	1.58E-04
Zinc	777.163	100	1.0E-06	1	10	25	70	9125	1.11E-03	3.04E-05	4.00E-01	2.00E-01	2.78E-03	1.52E-04
												HAZARD INDEX	3.79E-02	5.90E-03

TABLE : D-19
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP C)
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)	Absorption 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	61.094	1.0E-06	3120	0.6	0.005	65	1	70	91	5.84E-06	5.00E-05	1.17E-01

TABLE : D-20
SITE : TRONIC PLATING
ENV. MEDIUM : SUBSURFACE SOIL (GROUP C)
EXPOSURE TYPE : DERMAL CONTACT/PRESENT & FUTURE/UTILITY WORKER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	Concentration (mg/kg)	kg/mg	Skin Surface Area Contacted (cm2/day)	Soil Skin Adherence Factor (mg/cm2)	Absorption 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	61.094	1.0E-06	3120	0.6	0.005	10	25	70	9125	2.24E-07	5.00E-05	4.48E-03

TABLE : D-21
SITE : TRONIC PLATING
ENV. MEDIUM : STORM DRAIN SEDIMENTS
EXPOSURE TYPE : INGESTION/PRESENT & FUTURE/UTILITY 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 -1 (mg/kg/day)	Increased CA Risk
1,1,2,2-Tetrachloroethane	0.004	100	1.0E-06	1	2	25	70	25550	1.12E-11	2.00E-01	2.24E-12
1,2-Dichloroethylene (total)	0.036	100	1.0E-06	1	2	25	70	25550	1.02E-10		0.00E+00
Acetone	0.140	100	1.0E-06	1	2	25	70	25550	3.91E-10		0.00E+00
Chloromethane	0.008	100	1.0E-06	1	2	25	70	25550	2.24E-11	1.30E-02	2.91E-13
Ethylbenzene	0.027	100	1.0E-06	1	2	25	70	25550	7.63E-11		0.00E+00
Methylene Chloride	0.011	100	1.0E-06	1	2	25	70	25550	3.00E-11	7.50E-03	2.25E-13
Tetrachloroethylene	0.002	100	1.0E-06	1	2	25	70	25550	5.59E-12	5.10E-02	2.85E-13
Toluene	0.033	100	1.0E-06	1	2	25	70	25550	9.21E-11		0.00E+00
Trichloroethylene	0.180	100	1.0E-06	1	2	25	70	25550	5.03E-10	1.10E-02	5.54E-12
Vinyl Chloride	0.020	100	1.0E-06	1	2	25	70	25550	5.59E-11	1.90E+00	1.06E-10
Total Xylenes	0.140	100	1.0E-06	1	2	25	70	25550	3.91E-10		0.00E+00
Acenaphthene	1.405	100	1.0E-06	1	2	25	70	25550	3.93E-09		0.00E+00
bis(2-Ethylhexyl)phthalate	43.000	100	1.0E-06	1	2	25	70	25550	1.20E-07	1.40E-02	1.68E-09
Chrysene	1.000	100	1.0E-06	1	2	25	70	25550	2.80E-09	5.79E+00	1.62E-08
Dibenzofuran	1.200	100	1.0E-06	1	2	25	70	25550	3.35E-09		0.00E+00
Dimethylphthalate	1.675	100	1.0E-06	1	2	25	70	25550	4.68E-09		0.00E+00
Fluoranthene	1.786	100	1.0E-06	1	2	25	70	25550	4.99E-09		0.00E+00
Fluorene	2.001	100	1.0E-06	1	2	25	70	25550	5.60E-09		0.00E+00
2-Methylnaphthalene	15.895	100	1.0E-06	1	2	25	70	25550	4.44E-08		0.00E+00
Naphthalene	2.839	100	1.0E-06	1	2	25	70	25550	7.94E-09		0.00E+00
Phenanthrene	5.006	100	1.0E-06	1	2	25	70	25550	1.40E-08		0.00E+00
Pyrene	3.443	100	1.0E-06	1	2	25	70	25550	9.63E-09		0.00E+00
Aluminum	16026.472	100	1.0E-06	1	2	25	70	25550	4.48E-05		0.00E+00
Arsenic	9.800	100	1.0E-06	1	2	25	70	25550	2.74E-08	1.75E+00	4.79E-08
Beryllium	2.671	100	1.0E-06	1	2	25	70	25550	7.47E-09	4.30E+00	3.21E-08
Cadmium	1130.000	100	1.0E-06	1	2	25	70	25550	3.16E-06		0.00E+00
Chromium, VI	3.100	100	1.0E-06	1	2	25	70	25550	8.67E-09		0.00E+00
Chromium, total	1580.000	100	1.0E-06	1	2	25	70	25550	4.42E-06		0.00E+00
Cobalt	12.200	100	1.0E-06	1	2	25	70	25550	3.41E-08		0.00E+00
Copper	4560.000	100	1.0E-06	1	2	25	70	25550	1.27E-05		0.00E+00
Cyanide	42.000	100	1.0E-06	1	2	25	70	25550	1.17E-07		0.00E+00
Iron	7750.000	100	1.0E-06	1	2	25	70	25550	2.17E-05		0.00E+00
Lead	2290.000	100	1.0E-06	1	2	25	70	25550	6.40E-06		0.00E+00
Manganese	68.200	100	1.0E-06	1	2	25	70	25550	1.91E-07		0.00E+00
Mercury	0.310	100	1.0E-06	1	2	25	70	25550	8.67E-10		0.00E+00
Nickel	138.000	100	1.0E-06	1	2	25	70	25550	3.86E-07		0.00E+00
Selenium	2.400	100	1.0E-06	1	2	25	70	25550	6.71E-09		0.00E+00
Vanadium	46.000	100	1.0E-06	1	2	25	70	25550	1.29E-07		0.00E+00
Zinc	3200.000	100	1.0E-06	1	2	25	70	25550	8.95E-06		0.00E+00

TOTAL RISK 9.80E-08

TABLE : D-22
SITE : TRONIC PLATING
ENV. MEDIUM : STORM DRAIN SEDIMENTS
EXPOSURE TYPE : INGESTION/PRESENT & FUTURE/UTILITY 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)	Acute Body Dose (mg/kg/day)	Chronic Body Dose (mg/kg/day)	Acute Protective Body Dose (mg/kg/day)	Chronic Protective Body Dose (mg/kg/day)	Acute Hazard Quotient	Chronic Hazard Quotient
1,1,2,2-Tetrachloroethane	0.004	100	1.0E-06	1	2	25	70	9125	5.71E-09	3.13E-11				
1,2-Dichloroethylene (total)	0.036	100	1.0E-06	1	2	25	70	9125	5.21E-08	2.85E-10	4.00E-01	1.00E-02	1.30E-07	2.85E-08
Acetone	0.140	100	1.0E-06	1	2	25	70	9125	2.00E-07	1.10E-09		1.00E-01		1.10E-08
Chloromethane	0.008	100	1.0E-06	1	2	25	70	9125	1.14E-08	6.26E-11	9.00E-01		1.27E-08	
Ethylbenzene	0.027	100	1.0E-06	1	2	25	70	9125	3.90E-08	2.14E-10	3.20E+00	1.00E-01	1.22E-08	2.14E-09
Methylene Chloride	0.011	100	1.0E-06	1	2	25	70	9125	1.53E-08	8.39E-11	1.33E+00	6.00E-02	1.15E-08	1.40E-09
Tetrachloroethylene	0.002	100	1.0E-06	1	2	25	70	9125	2.86E-09	1.57E-11	2.00E-01	1.00E-02	1.43E-08	1.57E-09
Toluene	0.033	100	1.0E-06	1	2	25	70	9125	4.71E-08	2.58E-10	2.00E+00	2.00E-01	2.35E-08	1.29E-09
Trichloroethylene	0.180	100	1.0E-06	1	2	25	70	9125	2.57E-07	1.41E-09		6.00E-03		2.35E-07
Vinyl Chloride	0.020	100	1.0E-06	1	2	25	70	9125	2.86E-08	1.57E-10	3.00E-01		9.52E-08	
Total Xylenes	0.140	100	1.0E-06	1	2	25	70	9125	2.00E-07	1.10E-09	4.00E+00	2.00E+00	5.00E-08	5.48E-10
Acenaphthene	1.405	100	1.0E-06	1	2	25	70	9125	2.01E-06	1.10E-08		6.00E-02		1.83E-07
bis(2-Ethylhexyl)phthalate	43.000	100	1.0E-06	1	2	25	70	9125	6.14E-05	3.37E-07		2.00E-02		1.68E-05
Chrysene	1.000	100	1.0E-06	1	2	25	70	9125	1.43E-06	7.83E-09				
Dibenzofuran	1.200	100	1.0E-06	1	2	25	70	9125	1.71E-06	9.39E-09		4.00E-03		2.35E-06
Dimethylphthalate	1.675	100	1.0E-06	1	2	25	70	9125	2.39E-06	1.31E-08		1.00E+00		1.31E-08
Fluoranthene	1.786	100	1.0E-06	1	2	25	70	9125	2.55E-06	1.40E-08		4.00E-02		3.49E-07
Fluorene	2.001	100	1.0E-06	1	2	25	70	9125	2.86E-06	1.57E-08		4.00E-02		3.92E-07
2-Methylnaphthalene	15.895	100	1.0E-06	1	2	25	70	9125	2.27E-05	1.24E-07				
Naphthalene	2.839	100	1.0E-06	1	2	25	70	9125	4.06E-06	2.22E-08	5.00E-02	4.00E-03	8.11E-05	5.56E-06
Phenanthrene	5.006	100	1.0E-06	1	2	25	70	9125	7.15E-06	3.92E-08				
Pyrene	3.443	100	1.0E-06	1	2	25	70	9125	4.92E-06	2.70E-08		3.00E-02		8.98E-07
Aluminum	16026.472	100	1.0E-06	1	2	25	70	9125	2.29E-02	1.25E-04		1.00E+00		1.25E-04
Arsenic	9.800	100	1.0E-06	1	2	25	70	9125	1.40E-05	7.67E-08		3.00E-04		2.56E-04
Beryllium	2.671	100	1.0E-06	1	2	25	70	9125	3.82E-06	2.09E-08	3.00E+00	5.00E-03	1.27E-06	4.18E-06
Cadmium	1130.000	100	1.0E-06	1	2	25	70	9125	1.61E-03	8.85E-06	4.00E-03	1.00E-03	4.04E-01	8.85E-03
Chromium, VI	3.100	100	1.0E-06	1	2	25	70	9125	4.43E-06	2.43E-08		5.00E-03		4.85E-06
Chromium, total	1580.000	100	1.0E-06	1	2	25	70	9125	2.26E-03	1.24E-05	1.40E-01	8.76E-01	1.61E-02	1.41E-05
Cobalt	12.200	100	1.0E-06	1	2	25	70	9125	1.74E-05	9.55E-08				
Copper	4560.000	100	1.0E-06	1	2	25	70	9125	6.51E-03	3.57E-05		4.00E-02		8.92E-04
Cyanide	42.000	100	1.0E-06	1	2	25	70	9125	6.00E-05	3.29E-07	2.00E-02	2.00E-02	3.00E-03	1.64E-05
Iron	7750.000	100	1.0E-06	1	2	25	70	9125	1.11E-02	6.07E-05		5.00E-01		1.21E-04
Lead	2290.000	100	1.0E-06	1	2	25	70	9125	3.27E-03	1.79E-05				
Manganese	68.200	100	1.0E-06	1	2	25	70	9125	9.74E-05	5.34E-07		1.00E-01		5.34E-06
Mercury	0.310	100	1.0E-06	1	2	25	70	9125	4.43E-07	2.43E-09		3.00E-04		8.09E-06
Nickel	138.000	100	1.0E-06	1	2	25	70	9125	1.97E-04	1.08E-06	1.00E-01	2.00E-02	1.97E-03	5.40E-05
Selenium	2.400	100	1.0E-06	1	2	25	70	9125	3.43E-06	1.88E-08		5.00E-03		3.76E-06
Vanadium	46.000	100	1.0E-06	1	2	25	70	9125	6.57E-05	3.60E-07	8.00E-03	7.00E-03	8.21E-03	5.14E-05
Zinc	3200.000	100	1.0E-06	1	2	25	70	9125	4.57E-03	2.50E-05	4.00E-01	2.00E-01	1.14E-02	1.25E-04
												HAZARD INDEX	4.44E-01	1.06E-02

TABLE : D-23
SITE : TRONIC PLATING
ENV. MEDIUM : STORM DRAIN SEDIMENTS
EXPOSURE TYPE : DERMAL CONTACT/PRESENT & FUTURE/UTILITY WORKER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	Concentration (mg/kg)	kg/mg	Skin Surface Area Contacted (cm2/day)	Soil Skin Adherence Factor (mg/cm2)	Absorption 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	1130.000	1.0E-06	3120	0.6	0.005	2	25	70	9125	8.28E-07	5.00E-05	1.66E-02

TABLE : D-24
SITE : TRONIC PLATING
ENV. MEDIUM : STORM DRAIN WATER
EXPOSURE TYPE : DERMAL CONTACT/PRESENT AND FUTURE/UTILITY WORKER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	Concentration (mg/L)	Skin Surface Area Contacted (cm2)	Dermal Permeability Constants (cm/hr)	Exposure Time (hrs/day)	Exposure Frequency (days/yr)	L/cm3	Exposure Duration (years)	Body Weight (kg)	Averaging Time (days)	Chronic Body Dose (mg/kg/day)	Chronic Protective Body Dose (mg/kg/day)	Chronic Risk Ratio
Cadmium	8.270	3120.00	8.4E-04	4	2	0.001	25	70	9125	6.79E-06	5.00E-05	1.36E-01

APPENDIX E

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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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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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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1,1 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
Sol. (water): 400 mg/l at 20°C
BP: 37°C
MP: -122.1°C
VP: 500 mmHg 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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1,1,1-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 are used are consumptive (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 Other 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

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:	2.0×10^{0} mg/kg/day (mixed xylenes)
Chronic Inhalation RfD:	8.57×10^{-2} mg/kg/day (mixed xylenes)
Subchronic Oral RfD:	4.0×10^{0} mg/kg/day
Subchronic Inhalation RfD:	8.57×10^{-2} mg/kg/day
MCL:	10 mg/l
AWQC:	NA

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BASE-NEUTRAL/ACID EXTRACTABLE (SEMI-VOLATILES)

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:	2×10^{-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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DIBENZOFURAN

Use

Dibenzofuran has been used as an insecticide and as a laboratory chemical.

Chemical and Physical Properties

Chemical Formula: $(C_6H_4)_20$

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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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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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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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.[4] 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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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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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	Ambient Water Quality Criteria (d) Fish Consumption
								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^{0}	--	--	--	--	0.0001		
Benzo(a)pyrene	B2	6.1 x 10^{0}/5.79 x 10^{0}	--	--	--	--	0.0002		
Benzo(b)fluroanthene	B2	--/5.79 x 10^{0}	--	--	--	--	0.0002		
Benzo(k)fluroanthene	B2	--/5.79 x 10^{0}	--	--	--	--	0.0002		
Benzo(g,h,i)perylene	D	ND/ND	--	--	--	--	NA		
Chrysene	B2	--/5.79 x 10^{0}	--	--	--	--	0.0002		
Dibenz(a,h)anthracene	B2	--/4.5 x 10^{-1}	--	--	--	--	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^{0}	--	--	--	--	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		

-- = 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.

PESTICIDES

INORGANICS

ALUMINUM

Use

Aluminum is used in the shipbuilding, electrical, aircraft, automobile, light engineering, and jewelry industries. Powdered aluminum is used in paints and the pyrotechnic industry.

Chemical and Physical Properties

Chemical Formula: Al

SG: 2.708
BP: 2450°C
MP: 660°C
Sol. (water): insoluble, soluble in acids and alkalies

Fate and Transport

Aluminum does not exist in the environment in its elemental form. It is, however, a constituent of many minerals. When exposed to air it becomes coated with aluminum oxide, which prevents further corrosion (EPA, 1980). Aluminum is generally not regarded as a water pollution problem (EPA, 1977).

Pharmacokinetics

Little information on aluminum pharmacokinetics was available.

Aluminum has been found in all human organs. The lungs, however, show a higher concentration than all other organs. This probably results from inhalation of dust or fumes (EPA, 1977). The presence of aluminum in human organs also indicates absorption through ingestion (Sittig, 1991). Ingestion of aluminum affects its concentration in the liver, brain, testes and blood (Ondreicka, et al., 1966). Various diseases also influence aluminum concentration of body organs (Sorensen et al., 1974).

Human Toxicity

Noncarcinogenic

Systemic Effects

Fibrotic lung disease and severe and fatal lung damage have been observed in workers exposed to dust of aluminum metal (EPA, 1977). Aluminum is suspected of inducing neurotoxic effects, characterized by gradual loss of motor, speech, and cognitive functions. Another target organ for aluminum

toxicity is the bone. Low bone formation or osteomalacia has been linked to aluminum exposure. A form of anemia, which is not related to iron deficiency, has also been linked to aluminum exposure (EPA, 1992). Aluminum particles deposited in the eye may cause necrosis of the cornea (Sittig, 1991).

Teratogenic and Developmental Effects

Muller et al. (1990) administered 400 mg Al/kg/day to pregnant rats on days 1-7, 1-14 or 1-21 of gestation. No effects on maternal body weight or food intake were observed in dams on gestational days 1-7 or 1-14. In dams exposed on gestational days 1-21, a significant decrease in maternal body weight was observed. Aluminum tends to accumulate in the testes (EPA, 1980).

Mutagenic Effects

No information was available in the literature.

Carcinogenic Effects

Metallic aluminum was tested for carcinogenic activity, with no tumors resulting (Furst, 1971). EPA (1977) reported that carcinogenicity studies have failed to produce cancer in experimental animals. Other studies (Milham, 1979p; Anderson et al., 1982) indicate, however, a possible cancer risk from aluminum exposure.

No definite conclusion regarding the carcinogenicity of aluminum can be made from the available literature.

Ecotoxicity

Aluminum concentrations in water of over 1.5 ppm causes physiological and behavioral changes in rainbow trout (Freeman and Everhart, 1971). Aluminum seems to be toxic to plants at soil pH values below five (EPA, 1980).

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	$1 \times 10^{+0}$ mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	pH dependent

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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 $CdC1_2$ 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 $CdC1_2$ 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 $CdC1_2$) 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:	6.3×10^{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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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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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 $CaC0_3$ 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
Subchronic Inhalation 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
Subchronic Inhalation RfD:	1.14×10^{-4} 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
Subchronic Inhalation RfD:	8.57×10^{-5} 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)
Subchronic Inhalation RfD:	NA
MCL:	0.1 mg/l
AWQC:	Water and Fish Consumption - 13.4 µg/L
	Fish Consumption - 100 µg/L

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SELENIUM

Use

Selenium, exists in three forms; a red amorphous powder, a grey form, and red crystals. It is used primarily in the manufacture of selenium rectifiers. It is also utilized as a pigment for ruby glass, paints, and dyes; as a vulcanizing agent for rubber; a decolorizing agent for green glass; a chemical catalyst in the kjeldahl test; and an insecticide. It is used in the manufacture of electrodes, selenium photocells, selenium cells, and semiconductor fusions mixtures; in photographic toning baths; and for dehydrogenation of organic compounds. Selenium is utilized in veterinary medicine, antidandruff shampoos, radioactive scanning of the pancreas, and for photostatic and x-ray xerography. It may be alloyed with stainless steel, copper, and cast steel (Sittig, 1991).

Physical and Chemical Properties

AW:	78.96	MP:	200°C (red crystals)
SG:	4.28 (amorphous)		217°C (grey form)
	4.46 (red crystals)	VP:	1 mm Hg at 356°C
	4.81 (grey form)		

Fate and Transport

Selenium is released to the atmosphere in emissions from both natural and man-made sources, although man-made sources constitute the bulk of releases. Naturally, continental dust flux and volcanic dust and gas flux release selenium to the atmosphere (Lantzy and MacKenzie, 1979). U.S. EPA (1984) reports that approximately 90 percent of the overall release of selenium to the atmosphere from man-made sources is contributed by coal combustion and copper production. Other man-made sources of atmospheric selenium include glass manufacturing, selenium-recovery plants, burning of fuel oil, and refuse burning (NAS, 1976). NAS (1976) reports that, although a small fraction of selenium may exist in the gaseous state in the atmosphere, the predominant form of selenium is particulate. Chemical reactions in the troposphere may cause speciation of selenium, but removal of selenium from the atmosphere may occur primarily through wet and dry deposition rather than these tropospheric reactions (U.S. EPA, 1984; NAS, 1976). The residence time of selenium in the atmosphere appears to be related to particle size and is expected to be a few hours to several days (U.S. EPA, 1984).

In aquatic systems, the fate of selenium is related to the pH and oxidation-reduction potential of the water (U.S. EPA, 1984). Insoluble elemental selenium or metal selenide is formed in anaerobic and/or low pH waters. In aerobic and/or high pH (>6), water soluble selenite or selenate, which sorb or coprecipitate onto hydrous iron and manganese oxide, are formed. These selenium complexes control the mobility of the soluble selenium species. Volatile H_2Se may be formed in reducing aquatic environments. Selenium may be converted into volatile methylated products by sediment-dwelling microorganisms. Callahan et al. (1979) reports that biotransformation of selenium may result in its release to the atmosphere. The bioconcentration factor for selenium in freshwater and marine fish is approximately 400.

As in aquatic systems, the fate of selenium in soils is related to the pH and redox potential of the soil (U.S. EPA, 1984). Heavy metal selenides that exist in immobilized form are formed in acidic and poorly aerated soils. In well aerated and alkaline soils, selenites and selenates may be formed. The selenites may be immobilized via adsorption or complexation or both with iron and manganese hydroxides. NAS (1976) reports, however, that the selenates may leach from soils into ground water. In fact, Page (1981) detected selenium at a median concentration of 2 ppb in all of the ground water samples collected in New Jersey.

Pharmacokinetics

Glover (1970) examined urinary selenium levels of selenium-rectifier plant employees. Those employees exposed to higher levels of unspecified compounds in the air excreted higher levels of selenium in their urine than employees exposed to lower selenium levels. Although data do not permit quantification, absorption of selenium via inhalation is apparent. ATSDR (1989) reports that oral absorption of selenium orally can be affected by the physical state of the compound (solid or solution); the chemical form of selenium (organic or inorganic), and the dosing regimen. ATSDR (1989) reports, however, that the degree of selenium absorption in humans is independent of the exposure level. In humans, numerous studies reveal that absorption of sodium selenite or selenomethionine can exceed 80 percent for both small and large doses (ATSDR, 1989). Other studies indicate that humans absorb selenomethionine more efficiently than sodium selenite (ATSDR, 1989).

ATSDR (1989) reports that several studies indicate that different selenium compounds show different patterns of distribution in the body. Selenium from selenomethionine has been observed to concentrate in the pancreas of humans while selenium from sodium selenite concentrates in the liver and kidney (ASTDR, 1989).

Selenium is an integral part of glutathione peroxidase (GSH-Px), an enzyme found in most human tissues. GSH-Px is principally involved in the metabolism and removal of hydrogen peroxide and lipid hydroperoxides thereby protecting cellular membranes and lipid-containing organelles (Rotruck et al. 1973). Other proteins, including a selenoprotein in muscle and possibly nicotinic acid hydroxylase, incorporate or require selenium (Reddy and Massaro, 1983; Stadtman, 1983). Therefore, as ATSDR (1989) reports, the metabolism of selenium involves pathways for incorporation of selenium into selenium-dependent enzymes as well as pathways for excretion of selenium from the body. Intermediate metabolism of selenium involves oxidation and reduction reactions resulting, primarily in the respiratory and urinary excretion of the metabolites (ATSDR, 1989). ATSDR (1989) reports on numerous studies which state that a noticeable garlic odor of the breath results from selenium exposure in humans and that this is probably due to excretion of dimethyl selenide in expired air.

Excretion of selenium from the human body has been shown by numerous studies reported in ATSDR (1989) to occur via the urine, feces, expired air, and perspiration. Selenium veral studies indicate that the initial rate of excretion appears to be dose-dependent. Urinary and fecal excretion appear to be equivalent, each accounting for approximately 50 percent of the total output (LeVander and Morris, 1984; Stewart et al., 1978). Olson et al. (1963) and McConnell and Roth (1966) report that excretion of selenium in expired air becomes more significant at high selenium exposure levels.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Selenium toxicity has been clinically classified into three types: acute selenosis, subacute selenosis, and chronic selenosis (U.S. EPA, IRIS). Acute selenosis results from consuming relatively large amounts of selenium over a short period of time. Unsteady walking, cyanosis of the mucous membranes, and labored breathing usually occur, sometimes resulting in death. Other pathological effects include ongestion of the liver; endocarditis; myocarditis; degeneration of the smooth musculature of the gastrointestinal tract, gall bladder, and bladder; and erosion of the long bones (Francke and Moxon, 1936).

Impaired vision, ataxia, disorientation, and respiratory distress are the results of subacute selenosis, exposure to large doses of selenium over a longer period of time than acute selenosis. Rosenfeld and Beath (1964) state that it is most frequently observed in grazing livestock feeding on selenium-accumulating plants and has been referred to as "blind staggers."

Prolonged exposure of animals to more moderate levels of selenium results in chronic selenosis including skin lesions involving alopecia, hoof necrosis and loss, emaciation, and increased serum transaminases and alkaline phosphatase levels. In man, chronic selenosis is characterized by chronic dermatitis, fatigue, anorexia, gastroenteritis, hepatic degeneration, enlarged spleen, and increased concentrations of selenium in the hair and nails (Harr and Muth, 1972).

Yang et al. (1989 a,b) studied a population of approximately 400 individuals living in an area in China with unusually high environmental concentrations of selenium. Persistent clinical signs of selenosis were observed in only 5/349 adults. The mean blood selenium concentration in this group was 1.346 mg/l. Clinical signs observed included the characteristic "garlic odor" of excess selenium excretion in the breath and urine, thickened and brittle nails, hair and nail loss, lowered hemoglobin levels, mottled teeth, skin lesions, and CNS abnormalities (peripheral anesthesia, acroparesthesia, and pain in the

extremities). Dietary intake levels of 750-850 µg selenium/day resulted in increased blood coagulation time and a reduction in blood glutathione concentration. 1.261 mg selenium/day was the lowest selenium concentration to cause overt signs of selenosis. 0.853 mg selenium/day produced no clinical signs of selenosis.

Longnecker et al. (1991) evaluated a group of 142 randomly selected volunteers in South Dakota and Wyoming, some of who came from ranches with suspected high selenium intake based on previous cases of livestock selenosis. The subjects were followed for one year. The average selenium intake was 239 µg/day, approximately 2 to 3 times higher than the national average. No signs of selenium toxicity were observed at any intake level, including intakes as high as 724 µg/day.

Teratogenic and Other Developmental Effects

In his study on the high-selenium-area in China, Yang et al. 91989) observed malformations in chickens hatched from locally produced eggs. Teratogenic effects were not observed in human infants, however, even though selenium has been reported to be transmitted through the placenta to the fetus in animals. Yang et. al. (1983) also reported that chicken eggs from this area had very low hatchability and some of those that did hatch had deformed embryos.

Tarantal et al. (1991) found selenomethionine to have no teratogenic or developmental effects in macaques exposed to selenium concentrations as high as 0.3 mg/kg/day on gestational days 20 to 50. Schroeder and Mitchener (1971) exposed four generations of CD mice to 3 ppm selenium as selenate (390 µg/kg/day). There was a significant increase in young deaths in the F1 generation and an increase in the number of runts in generations F1 through F3. There was also a decrease in breeding events in the F3 generation. Rosenfeld and Beath (1954) administered selenium as potassium selenate to sires and pregnant rats through five breeding cycles at 1.5, 2.5, or 7.5 ppm selenium (75, 125, of 375 µg/kg/day). There was a 50 percent reduction in the number of young reared at the 2.5 ppm level. At 7.5 ppm, female fertility, the number of survivors, and the rate of growth in the young were all reduced. Nobunaga et al. (1979) found that 6 ppm selenium as selenite decreased the body weight of surviving IVCS mice fetuses when administered 30 days prior to mating and throughout gestation.

Mutagenic Effects

U.S. EPA (IRIS) reports that the data on the mutagenicity of selenium and its compounds are equivocal. Selenate and selenite were mutagenic in a reverse mutation assay with *S. typhimurium* strains TA98, TA100, and TA1537 in the absence of rat hepatic homogenates (Noda et al., 1979). In the same assay, Lofroth and Ames (1978) found selenate, but not selenite, to be mutagenic. Selenite (selenious acid and sodium selenite) produced DNA damage in *Bacillus subtilis* strains 17A and 45T. Selenate (selenic acid and sodium selenate), however, was negative in the rec assay (Nakamuro et al., 1976).

Whiting et al. (1980) report that sodium selenide, sodium selenate, and sodium selenate (in order of decreasing activity) caused an increase in unscheduled DNA synthesis in Chinese hamster ovary cells in the presence or absence of glutathione at concentrations of 1×10^{-4} M. 1×10^{-5} M sodium selenite increased chromosomal aberrations in rat lymphocytes (Newton and Lilly, 1986). Nakamuro et al. (1976) reports that sodium selenite, selenious acid, selenic acid, and selenium oxide had the same effect on human lymphocytes at 2.6×10^{-6} M. Newton and Lilly (1986) also report that 10-12 mg/kg sodium selenite (near lethal dose) administered intravenously produced an increase in chromosomal aberrations in the bone marrow of rats. An increase in SCES in human whole blood cultures was induced by elemental selenium, selenium dioxide, sodium selenide, and sodium selenite (in order of decreasing activity), but not sodium selenate (Ray and Attenburg, 1980).

Carcinogenic Effects

U.S. EPA (IRIS) reports that several investigators have studied the association between serum selenium and the risk of cancer through prospective, case-control, and nested-case-control studies. These investigators found that patients with cancer, particularly gastrointestinal cancer, prostatic cancer, or Hodgkin's lymphoma, had significantly lower blood selenium levels in blood than healthy patients. The risk of cancer for subjects at the lowest serum selenium level was twice that of subjects with higher levels.

Shamberger and Frost (1969) report in an ecological study that an inverse relationship exists between human cancer death rates and the selenium concentrations in foliage plants of several Canadian provinces. The cancer death rate in provinces with selenium-containing plants was 122.2 ±7.87 per 100,000 people and in provinces without these plants, 139.9 ±4.0 per 100,000 people. Similarly, Shamberger and Willis (1971) report a correlation between decreased human cancer death rates and an increase in the selenium in forage crops in California. The cancer death rate was 141.2 per 100,000 in high-selenium areas (selenium 0.11 ppm of forage crops), 190.1 per 100,00 in medium-selenium areas (0.05 - 0.10 ppm), and 233.0 per 100,000 in low-selenium areas (0.02 - 0.05 ppm). The ratio of observed to expected cancer death rates by anatomic site for men in 17 paired cities including high- and low-selenium

areas was investigated by Shamberger and Willis (1971) as well. The anatomic sites that would come into contact with dietary selenium, such as the pharynx, esophagus, stomach, bladder, and intestine showed a substantially lower rate ratio in the high-selenium cities than in the low-selenium areas. U.S. EPA (IRIS) reports that several other studies have found an inverse relationship between environmental selenium levels and the incidences of human cancer including colon and breast cancer.

Glover (1970) studied approximately 300 workers exposed to selenium in an electronics rectifier process over a 26-year period. No statistically significant increase in cancer mortality was observed.

Ecotoxicity

The U.S. EPA (1987) reports acute toxicity values for selenium (IV) (oxidation state = 4) in 23 freshwater fish and invertebrate species in 22 genera that range from 340 µg/l for the amphipod to 203,000 µg/l for the leech. Although 12 of the 23 species are fishes, the 2 most sensitive and 2 most resistant are invertebrates. Chronic toxicity values for 21 freshwater fish and invertebrate species range from >47 to 692 µg/l. A 90-day LC_{50} of 54 µg/l was obtained for the rainbow trout in a separate test (U.S. EPA, 1987). Nine species of freshwater algae have toxicity values ranging from 500 to 30,000 µg/l. Selenium (IV) uptake by fishes takes about 100 days to reach steady-state and bioconcentration factors from 2 to 452 have been reported (U.S. EPA, 1987).

In saltwater, acute toxicity values for 16 fish and invertebrate species range from 599 µg/l for haddock larvae to 17,350 µg/l for the fourspine stickleback. Fish and invertebrates have similar ranges of sensitivities to selenium (IV) and the acute toxicity values for the 7 most sensitive species differ only by a factor of 3.2. Chronically, the mysid and sheepshead minnow have toxicity values of 211.7 µg/l and 675.2 µg/l, respectively. In a test with a saltwater diatom, a concentration of 7,930 µg/l selenium (IV) caused a 50 percent reduction in chlorophyll *a*. Three species of algae, however, were stimulated by concentrations of 10 to 10,000 µg/l. Steady-state bioconcentration factors for the chela muscle of the adult shore crab and the whole adult euphausiid were 3.88 and 200, respectively (U.S. EPA, 1978).

Standards, Criteria and Guidelines

EPA Class D Carcinogen
(Selenium sulfide class = B2)

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:	5.0×10^{-3} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.05 mg/l
AWQC:	Water and Fish Consumption: 10 µg/l
	Fish Consumption: 6.8 mg/L (recalculated)

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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
Subchronic Inhalation RfD:	NA
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
Subchronic Inhalation RfD:	NA
MCL	0.002/0.001 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
SG: 5.96 at 20°C
Sol. (water): at 20°C insoluble
Sol. (organics): insoluble

BP: 3,380°C
MP: 1,890°C

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
Subchronic Inhalation RfD:	NA
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
Subchronic Inhalation RfD:	NA
MCL:	5 mg/l (secondary)
AWQC:	NA

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