Gailis

CH2M HILL

TRANSMITTAL

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Date: April 5, 1984

Our Project No. W66204.B0

ttn: Ann P. Gailis

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SECTION II

INTRODUCTION

Under the Comprehensive Environmental Response Compensation and Liability Act (CERCLA), the US EPA has been given broad powers to act in response to releases of hazardous substances into the environment. The US EPA may act if such releases present a real or potential threat to public health or welfare or to the environment. This mandate is carried out in threediscrete phases: 1) Emergency Removal Actions 2} Planned Removal Actions 3) Planned Remedial Actions.

One of the principal objectives of the above named actions is to assure that residual concentrations of hazardous chemicals released into the environment do not pose a significant threat to human health. One aid to achieving this goal is the preparation of an Endangerment Assessment.

In its broadest sense, the Endangerment Assessment must reach a proper balance between a chemical toxicity versus actual potential for human exposure. To achieve this end, such diverse conditions as demographic, geographic, physical, chemical and biological factors must be considered.

The following report represents an Endangerment Assessment for Vertac Chemical Corporation, Jacksonville, Arkansas. Vertac recently has drawn National attention for problems associated with incorrect chemical waste disposal practices. Vertac was a manufacturer of 2,4-dichlorophenoxy acetic (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). The site has been identified by Federal and State investigators for heavy dioxin contamination.

Due to the limited availability of actual data on the Vertac Site, the preparers of this report relied heavily upon a literature search of published reports on dioxin and its chemically associated compounds. Values expressed as exposure limits (see Section V - "Acceptable Contamination Levels") are taken from published literature. These limits are useful as guidelines only and should not be taken as abolute exposure limits.

SECTION III

BACKGROUND

Site History

The site known as Vertac Chemical Corporation, located on approximately 93 acres in Jacksonville, Arkansas, came into existance in the early 1940's. At that time, it was part of an ordinance plant needed to assist in World War II efforts. After the war and in 1948, the site was purchased by Reasor-Hill Company. Reasor-Hill converted the old ordinance plant into a pesticide manufacturing facility. The herbicide 2,4,5-trichlorophenoxy acetic acid was manufactured here during the fourteen years Reasor-Hill owned and/or operated the plant.

Hercules Powder Company (now Hercules, Inc.) purchased the plant in 1962. Agent Orange, a 50:50 mixture of 2, 4, 5-T and 2, 4-dichlorophenoxy acetic acid (2, 4-D) was manufactured at the site during this time. Agent Orange was produced for use as a defoliant during the Vietnam conflict. The site was operated by Hercules until 1972 when it was leased to Transvaal, Inc. Transvaal purchased the property in 1976. In 1978, Transvaal merged with its parent/holding company Vertac, Inc., which continued to manufacture industrial and agricultural chemicals. Vertac ceased to manufacture 2, 4, 5-T in April, 1979. The toxic chemicals 2,3,7,8-tetrachlorodibenzo-p-dioxin, commonly known as dioxin, is an unavoidable trace contaminent in 2,4,5-T and 2,4-D.

An explosion is known to have ocurred at the plant in 1974. Episodes of chloracne were reported to have occured in an undisclosed number of employees as a result of the explosion.

In 1978, Senator Mark Hatfield (D-OR) initiated a nationwide dioxin survey. Initial screening resulting from this survey at Vertac, showed dioxin in waste sludges and various production processes. As a direct result of the initial survey, the US EPA Region VI and the Arkansas Department of Pollution Control and Ecology (ADPC&E) conducted a sampling and analysis program to identify sources of dioxin contamination attributable to Vertac from on site sources and off site migration.

In March, 1980, a consent decree was filed with the US District Court in Little Rock, Arkansas. The agreement was to settle suits initiated by the Department of Justice on behalf of the US EPA against Vertac. The purpose of the suits were to correct waste disposal problems at the Jacksonville site. Under terms of the consent decree, Vertac has agreed to the following:

- Retain an independent consulting firm to study groundwater and surface conditions at the site to identify areas which may require remedial work.

- Propose and implement remedial actions, if any, to prevent the discharge of pollutants from the site into the environment.

- Develop a plan for the orderly management of wastes stored on the plant site by treatment or off-site disposal.

- Conduct a study of the fate and movement of pollutants in Rocky Branch Creek and Bayou Meto.

- Propose remedial measures to remove or stabilize pollutants in Lake Dupree, located in Jacksonville City Park.

- Establish a trust-fund in the principal sum of $60,000 restricted to assure long-term maintenance of remedial work at the plant site.

- Develop and implement standards for wastewater pretreatment prior to discharge to the Jacksonville sewage treatment plant.

Location of the Site

The Vertac Plant is located in Jacksonville, Arkansas and is approximately 7 1/2 miles from Little Rock. It is comprised of a 93 acre fenced tract. Its eastern border and main access point is Marshall Road. It is bordered on the south by a residential sub-division off Braden Street.

Levels of Contamination

Numerous multimedia samples (i.e., air, surface water, groundwater, drum and sediment) have been analyzed at Vertac Site since 1979. In April 1982, Vertac contracted an independent environmental firm, Developers International Services Corporation (DISC) of Memphis, Tennessee to study existing on-site conditions. Results of the DISC sampling programs are summarized in Figures III-A through D following. The DISC study results show widespread on-site contamination through surface water, groundwater and sediment samples. Surface water samples contained the suspected carcinogens chlorobenzene; 2,4-dichlorophenol; 2-chlorophenol; 2-(2,4,5-trichlorophenoxy) propionic acid; 2,4-dichlorophenoxyacetic acid (2,4-D); 1,2,4-trichlorobenzene, and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) in the part per billion range.

Groundwater samples showed the suspected carcinogens 1,2,4-trichlorobenzene, 2,4,5-T; 2(2,4,5-T)P; 2-Chlorophenol; 2,4-dichlorophenol; chlorobenzene; and 1,4-dichlorobenzene in the part per billion level.

Sediment samples showed the suspected carcinogens 1,2,4-trichlorobenzene; 1,4-dichlorobenzene; chlorobenzene; 2-chlorophenol; and 2,4 dichlorophenol in the part per billion range and 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) in the part per trillion range.

The DISC report concludes that no threat of contamination migration from the cooling water or sediment into the groundwater is likely even though they all contain some of the above contaminents. It is believed that the source of these contaminents is the old drum storage or burial areas.

DISC scientists found concentrations of 50-100 parts per trillion dioxin in surface soils at the site. Except for elevated dioxin concentrations near the Reasor-Hill landfill area, DISC researchers feel these represent background levels. Previous samples taken by EPA and ADPC&E show significant dioxin contamination in site soils. This is an area of concern since soil can blow off the site into adjacent residential areas.

One further area of concern is dioxin levels as high as 800 ppt in edible portions of fish in downstream waters. Although an official ban on catching downstream fish is in effect, eyewitnesses have reported fisherman in these areas.

CHEMICAL NAME	DATE SAMPLED	CONCENTRATION (ppb)	TYPE SAMPLE	LOCATION
2-CHLOROANISOLE	8/13/82	3050.0	SURFACE WATER	1
	8/13/82	510	SURFACE WATER	9
	8/5/82	26515	GROUND WATER	6
2,4-DICHLOROANISOLE	8/5/82	94.0	GROUND WATER	8
4-CHLOROANISOLE	7/23/82	170.0	GROUND WATER	10
	8/5/82	10000	GROUND WATER	5
	8/13/82	2750	SURFACE WATER	1
TOLUENE	NO SIGNIFICANT AMOUNTS DETECTED			

*LOCATION:
1. East Ditch at E. Plant Boundary, Entire 3" depth
2. Central Ditch, east of culvert, entire 4 " depth
3. East Ditch, average thickness 1.5'
4. Central Ditch, average thickness 0.2'
5. Well No. 9
6. Well No. 12
7. Well No. 18
8. Well No. 4
9. Rocky Branch below Cooling Pond Ave. thickness 0.3'
10. Well No. 6
11. Well No. 7
12. Cooling Pond SE Quad. Avg. thickness 1.0'
13. Cooling Pond NW Quad. Avg. thickness 0.8'
14. Cooling Pond NE Quad. Avg. thickness 1.0'
15. Cooling Pond-Center, Mid depth
16. Rocky Branch Below Equilization Basin
17. Rocky Branch, Below Cooling Pond, mid depth
18. Cooling Pond SW Quad. Avg. thickness 0.9'

*KEY:
GW - Ground Water
SW - Surface Water
Sed - Sediment

CHEMICAL	DATE SAMPLED	CONCENTRATION (ppb)	TYPE SAMPLE	LOCATION
2,4-D	8/13/82	39000	SURFACE WATER	1
	8/13/82; 9/17/82	1033; 783.0	SURFACE WATER	2
	8/13/82; 9/17/82	5.6; <8	SURFACE WATER	16
	8/13/82; 9/17/82	<8; <8	SURFACE WATER	17
	8/13/82; 9/17/82	<8; <8	SURFACE WATER	18
	8/5/82	761	GROUND WATER	8
	7/23/82	7.5	GROUND WATER	11
	7/23/82	9.0	GROUND WATER	5
	8/5/82	98500	GROUND WATER	6
	8/6/82	438	GROUND WATER	7
2,4,5-T	8/13/82	12400	SURFACE WATER	1
	8/13/82; 9/17/82	1033; 543	SURFACE WATER	2
	8/13/82; 9/17/82	<4; <4; <4	SURFACE WATER	16
	8/5/82	4195	GROUND WATER	8
	8/5/82	70970	GROUND WATER	6
	8/6/82	978.0	GROUND WATER	7
2(2,4,5-T)P	8/5/82	45	GROUND WATER	8
	8/6/82	208	GROUND WATER	7

CHEMICAL	DATE SAMPLED	CONCENTRATION (ppb)	TYPE SAMPLES	LOCATION
2(2,4,5-T)P	8/5/82	25520	GROUND WATER	6
	8/13/82	<8	SURFACE WATER	1
	8/13/82; 9/17/82	33.0; 34.13	SURFACE WATER	2
	8/13/82; 9/17/82	<8; 25.53; 27.20	SURFACE WATER	16
	8/13/82	9.6	SURFACE WATER	17
	8/13/82	16	SURFACE WATER	18
2,3,7,8-D	8/13/82	3836 ppt	SEDIMENT	19
	8/13/82	2826 ppt	SEDIMENT	14
	8/13/82	7859 ppt	SEDIMENT	4
2,3,7,8-D	8/6/82	6450 ppt	SEDIMENT	3
	8/13/82	4350 ppt	SEDIMENT	12
	8/13/82	9981 ppt	SEDIMENT	13
	8/6/82	839 ppt	SEDIMENT	9

*LOCATION:
1. East Ditch at E. Plant Boundary, Entire 3" depth
2. Central Ditch, east of culvert, entire 4" depth
3. East Ditch, average thickness 1.5'
4. Central Ditch, average thickness 0.2'
5. Well No. 9
6. Well No. 12
7. Well No. 18
8. Well No. 4
9. Rocky Branch below Cooling Pond Ave. thickness 0.3'
10. Well No. 6
11. Well No. 7
12. Cooling Pond SE Quad. Avg. thickness 1.0'
13. Cooling Pond NW Quad. Avg. thickness 0.8'
14. Cooling Pond NE Quad. Avg. thickness 1.0'
15. Cooling Pond-Center, Mid depth
16. Rocky Branch Below Equilization Basin
17. Rocky Branch, Below Cooling Pond, Mid depth
18. Cooling Pond SW Quad. Avg. thickness 0.9'

*KEY:
GW - Ground Water
SW - Surface Water
Sed - Sediment

SECTION IV

PHYSICAL/CHEMICAL ASSESSMENT

INTRODUCTION

During their July through September, 1982 program, Developers, International Services Corporation (DISC) collected soil, surface water, sediment and groundwater samples for analysis. Classes of compounds analyzed include Chlorophenols, Chlorobenzenes, Chloroanisoles, Toluene and Chlorophenoxy herbicides. Since these results are not inconsistent with previously reported analytical results they represent the classes of compounds discussed herein.

CHLOROPHENOXY HERBICIDES

2,3,7,8-Tetrachloro-dibenzo-p-dioxin (TCDD,Dioxin)

2,3,7,8-TCDD was first reported in the chemical literature in 1872, however, its acute toxicity did not become understood until the 1950's. In it's pure form at room temperature TCDD is a colorless crystalline solid. Because it is an unwanted by-product in the manufacture of various herbicides, pesticides and chlorophenols it is usually found in chemical wastes and sludges remaining from manufacturing processes.

Physical Properties

Chemical Formula: $C_{12}H_4Cl_4O_2$
Molecular weight: 321.96
Boiling point at 1 ATM, F: not available
Solubility in water, g/100 g water at 20 C: 200 ppm
Flash point: NA
Vapor pressure at 20 C mm/Hg: NA
Melting point, F: 581 F
Upper explosive limit in air % by volume: NA
Lower explosive limit in air % by volume: NA
Specific gravity: NA

Incompatibilities: None known

Stability:

Chemically TCDD is quite stable. Thermal destruction requires temperatures greater than 700 C. It binds strongly to soils and particulates. Various studies have shown that dioxins are quickly degraded by sunlight or artificial light. Once dioxins penetrate soils, however, studies show they can persist for long periods of time. Studies at Times Beach, Missouri suggest that dioxins can remain underground in soils for many years.

Analytical Procedures:

Scientists at the Brehm Laboratory of Wright State University have been performing dioxin analyses under the auspices of several Federal and State agencies since 1972. Brehm has developed and applied complex multimedia sampling protocols.

A new analytical technique has been developed in a joint Brehm Laboratory/Battelle Columbus Laboratory which was funded by a prime contract between Battelle and the US EPA. This new method is presented as Appendix A of this report. USEPA Approved Municipal and Industrial Waste Water Method #613 GC/MS; 0.002g/L.

2,4,5-Trichlorophenoxyacetic acid (2,4,5-T)

2,4,5-T is a herbicide which was developed for use during World War II. It was discovered in 1955 that the contaminant TCDD associated with 2,4,5-T, caused chloracne in workers exposed to the compound. Dispite this knowledge, use of the herbicide spread. It was used as a weed killer on rangeland, pastures, nursery and rice crops. In 1974 the US EPA banned use of 2,4,5-T on food crops. It was widely used in Vietnam from 1962 until 1969. It is a colorless to tan, odorless solid or used as a liquid mix for herbicide.

Physical Properties

Chemical Formula: $C_8H_5Cl_3O_3$
Molecular Weight: 256
Boiling point at 1 atm,F: Decomposes
Solubility in water, g/100 g water at 20 C: 0.03%
Flash point, closed cup, F(or open cup if 0 C): Incombustible
Vapor Pressure at 20 C mmHg: 0.00mm
Melting point F: 316 F
Upper Explosive Limit in Air, % by volume: Incombustible
Lower Explosive Limit in Air, % by Volume: Incombustible
Specific gravity: 1

Reactivity

Incompatabilities: strong oxidizers
Instability: Temperatures 158 C may cause sealed metal container to burst.

Analysis

NIOSH Manual of Analytical Methods, 2nd Editing, Volume 5, 1979, available from the Government printing office, Washington,D.C.

2,4-Dichlorophenoxyacetic Acid (2,4-D)

2,4-D is a herbicide which is commonly used in its salt or ester forms. Agent Orange is a 50:50 mixture of 2,4-D and 2,4,5-T. It is a colorless, odorless solid.

Physical Properties

Chemical Formula: $Cl_2 C_6 H_4 OCH_2 COOH$
PEL: 10 mg/m per 8 hrs.
Molecular Weight: 221
Boiling Point: Decomposes
Specific gravity: 1.1 (estimate)
Vapor Density: (air = 1 at boiling point of 2,4-D): 7.63
Melting point: 140 C
Vapor pressure at 20 C: Essentially zero
Solubility in water at 20 C: 0.07 ppm

Incompatabilities: Contact with strong oxidizers may cause fires and explosions.

Analytical Methods:

NIOSH Manual of Analytical Methods, 2nd Edition, Vol.3, 1977.

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2-(2,4,5-Trichlorophenoxy)propionic acid; Silvex

Physical Characteristics

Molecular Formula: $O_3Cl_3C_9H_7$
Form: crystals
Molecular weight: 270
Boiling point at 1 atm, F: NA
Solubility in water, g/100 g water at 20 C: slightly soluble
Flash point, closed cup, F: 140 F
Vapor Pressure at 20 C mmHg: NA
Melting Point, F: 327 F
Upper Explosive Limit in air, % by volume: combustable
Lower explosive limit in air, % by volume: combustable
Specific gravity: NA

Incompatibilities: Combustible substances

CHLOROPHENOLS

Chlorinated phenol is a class of 19 compounds, made up of a benzene ring to which one hydroxy (OH) group plus from one to five chlorine atoms are attached. Dioxins are found as undesirable contaminants formed in the manufacture of these compounds.

Chlorophenols are industrially important as raw materials for the manufacture of other products including pesticides. Any chlorophenol with a chlorine atom attached to the benzene m/mg at the number 2 carbon position may be a dioxin precursor. This precludes the occurance of dioxins in pesticide manufacture only.

2-Chlorophenol

Physical Characteristics

Chemical Formula: C_6H_5CLO
Reportable Quantity: 1 lb CWA 307(A)
Description: colorless liquid
Molecular weight: 128.6
Boiling point at 1 atm, F: 347 F
Solubility in water, g/100g water at 20 C: 2.8 g/100 ml
Flash point, closed cup F: 147 F
Vapor pressure at 20 mm/Hg: 1 mm at 53.78°F
Melting point, F: 48.2 F
Upper explosive limit in air, % by volume: combustible
Lower explosive limit in air, % by volume: combustile
Minimum explosive concentraction for a dust/vapor: auto-ignition 1022 F.
Specific gravity: 1.265

Incompatibilities:

Active metals
Amines
Oxidizers
Oxygen
Moisture
Heat
Peroxides

Analytical Method:

EPA Method for Analysis of Municipal and Industrial Waste Water, Method #604; GC; Detection limit; 0.3 ug/L.

2,4-Dichlorophenol

Physical Characteristics

Chemical Formula: $C_6H_4Cl_2O$
Reportable quantity: 1 lb. (WA 307(A)
Physical Description: white solid, pale yellow crystals
Molecular weight: 163
Boiling point at 1 atm, F: 410 F
Solubility in water, g/100g water at 20 C: 0.45 g
Flash point, closed cup: 237 F
Vapor pressure at 20 C mm Hg: 1 mmHg
Melting point, F: 113 F
Upper Explosive Limit in air, % by volume: combustible
Lower explosive limit in air, % by volume: combustible
Specific gravity: 1.383

Incompatabilities

Heat, 2,4-dichlorophenol decomposes at high temperatures, releasing toxic gases:

oxidizers
peroxides
acids
oxygen

Analytical Method:

EPA Method for Municipal and Industrial Wastewater Method #604; G.C.; Detection limit, 0.39 ug/L.

2,4,6-Trichlorophenol

Physical Characteristics

Chemical Formula: $C_6H_3Cl_3O$
Physical Description: yellow solid or colorless needles, strong phenolic odor.
Molecular weight: 197
Boiling point at 1 atm, F: 475°F
Solubility in water g/100 g water at 20°C: 0.09g
Flash point: non-flammable
Vapor pressure at 20°C mmHg: 1 mmHg at 170°F
Melting point: 156.2°F
Upper explosive limit, % by volume: combustible
Lower explosive limit, % by volume: combustible
Specific gravity: 1.490

Incompatabilities:

Heat: Decomposes at high temperatures, toxic or dangerous gases are released.

Strong oxidizers	Oxygen
Peroxides	Steam

Incompatibilities:

Heat: Decomposes at high temperatures, releasing toxic and/or dangerous gases.
Water
Steam

CHLOROBENZENES

Chlorobenzenes are a commercially important family of organic chemicals, especially as raw materials in the manufacture of other products. 2,4,5-trichlorophenol, which is the starting material for the manufacture of various industrial and agricultural chemicals, including the herbicide2,4,5-T,is made from 1,2,4,5-tetrachlorobenzene.

Chlorobenzene

Physical Characteristics

Physical Appearance: Chlorobenzene is a colorless liquid with a mild aromatic odor.
Formula: C_6H_5Cl
Molecular Weight: 112.5
Boiling Point (.760 mmHg): 132°C
Specific Gravity: 1.1
Vapor Density: 3.9
Melting Point: -44°C
Vapor Pressure at 20°C: 8.8 mmHg
Solubility in water g/100g water at 20°C: 0.05
Evaporation Rate: 1
Flash point: 28.9°C

Reactivity

Heat contributes to instability

Incompatibilities

Stong oxidizers

Analytical Method

NIOSH Manual of Analytical Methods, 2nd Edition, Vol.2, 1977.
EPA Methods for Municipal and Industrial Wastewater, Method #601, G.C. with Purge and Trap; Detection Limit, 0.25 ug/L.

1,4-Dichlorobenzene (p-dichlorobenzene)

Physical Characteristics

Formula: 1,4-C6H4Cl2
Appearance: Colorless solid with camphor-like odor

Molecular Weight: 147
Boiling point (760 mmHg): 174°C
Specific Gravity: 1.46
Vapor Density: 5.1
Melting Point: 53°C
Vapor Pressure at 20°C: 0.4 mmHg
Solubility in Water g/100 g water at 20°C: 0.008
Evaporation Rate: NA
Flash Point: 65.6°C

Reactivity

Very stable

Incompatibilities: none

Analytical Method

NIOSH Manual of Analytical Methods, 2nd Edition, Vol.3, 1972.
EPA Method for Municipal and Industrial Wastewater, Method #601, G.C. with purge and trap; Detection limits 0.25 ug/L.

1,2-Dichlorobenzene

Physical Characteristics

Formula: 1,2-C6H4Cl2
Physical Appearance: Colorless to pale yellow liquid with a pleasant aromatic odor.
Molecular weight: 147
Boiling point (760 mmHg): 180°C
Specific Gravity: 1.3
Vapor Density: 5.1
Melting Point: -17.6°C
Vapor Pressure at 20°C: 1.2 mmHg
Solubility in water, g/100 g water: 0.015
Evaporation Rate: 1
Flash Point: 66°C

Reactivity

Heat contributes to instability.

Incompatibilities: Contact with strong oxidizers, hot aluminum or aluminum alloys may cause fires and explosions.

Analytical Method: EPA Method for Municipal and Industrial Waste Water, Method #601; Purge and Trap; Detection Limits 0.15 ug/L.

1,2,4-Trichlorobenzene

Physical Characteristics

Chemical Formula: C6H3Cl3
Physical Appearance: Colorless liquid with aromatic odor
Molecular weight: 181.4
Boiling Point at 1 atm F: 415°F
Solubility in water g/100g water at 20°C: insoluble
Flash Point: 230°F
Vapor Pressure at 20°C mmHg: 1 mm at 101°F
Melting Point: 62.6 F
Upper Explosive Limit in air, % by volume: 6.6% at 302°F
Lower Explosive Limit in air, % by volume: 2.5% at 302°F
Specific Gravity: 1.46

Reactivity:

Decomposes at high temperatures, releasing toxic and/or dangerous gases.

Incompatibilities:

Strong oxidizers

Analytical Method:

EPA Methods for Municipal and Industrial Wastewater, Method #612, G.C.; Detection Limit, 0.05 ug/L.

1,3-Dichlorobenzene

Physical Characteristics

Formula: 1,3-C6H4Cl2
Molecular Weight: 147.0
Melting Point: 24.7°C
Boiling Point: 173°C
Vapor Pressure: (25°C), torr: 2.28
Solubility in water (temperature unknown), mg/l: 123

Analytical Method:

EPA Methods for Municipal and Industrial Wastes, Method #612, G.C.; Detectioon Limit 0.32 ug/L.

Hexachlorobenzene

Physical Characteristics

Formula: C6Cl6
Physical Appearance: solid needle-like crystals
Molecular weight: 284.76
Boiling Point at 1 atm °F: Sublimes at 613°F
Solubility in water, g/100g water 20°C: insoluble

Flash Point: 468°F
Upper Explosive Limit in air, % by volume: combustible
Lower Explosive Limit in air, % by volume: combustible
Specific Gravity: 2.04

Incompatabilities:

Dimethyl Formaldehyde

Reactivity:

Heat; Decomposes at high temperatures, releasing toxic and/or dangerous gases.

Analytical Method:

EPA Methods of Municipal and Industrial Wastewater, Method #612, G.C.; Detection Limit 0.05 ug/L.

Toluene

Toluene is an industrially important compound used in a variety of process operations. Some of its uses include: use as a solvent in pharmaceutical, chemical, rubber and plastics industries; as a thinner for paints and coatings; as a starting material and intermediate in organic and chemical synthesis; in manufacture of insecticides.

Physical Characteristics

Chemical Formula: C6H5CH3
Appearance: Colorless liquid with an aromatic odor.
Molecular Weight: 92.1
Boiling Point: 111°C
Specific Gravity: 0.86
Vapor Density: 3.14
Melting Point: -95°C
Vapor Pressure at 20°C: 22 mmHg
Solubility in water, g/100 g water at 20°C: 0.05
Evaporation Rate: 2.24
Flash Point: 4°C

Reactivity

Heat: Containers may burst at elevated temperatures.

Incompatibilities:

Strong oxidizers

Analytical Methods

NIOSH Manual of Analytical Methods, 2nd Ed., Vol.3,1977.
EPA Methods for Municipal and Industrial Wastewaters; Method #602, G.C.; Purge and Trap; Detection limit, 0.2 ug/L.

Chloroanisoles

After contacting numerous sources (i.e., Chemical Manufacturers Association, Hazardline, etc.) it has become evident that Chloroanisles are not a commercially available product. It is our belief, therefore, that they are most likely formed as a competing reaction or as a contaminent during the manufacture of 2,4-D and 2,4,5-T. 2-Chloroanisole is probably formed by the acylation of 2-chlorophenol during a phase separation with toluene or other toluene consuming step. In a similar manner, 2,4-dichloroanisole is probably formed by the acylation of 2,4-dichlorophenol. If this theory is correct, we would expect to see high levels of chloroanisoles in toluene still bottoms and lower levels as contaminents in finished product.

Physical data (i.e., melting point, vapor pressure, specific gravity, etc.) have not been worked out for these compounds. It is therefore impossible, at this point, to predict how they will react in the environment. It is possible to say however, that they are persistent in the environment since they are detected by chemical analysis four years after Vertac ceased to manufacture 2,4,5-T. Chloroanisoles have been detected in the part per billion levels in ground water and surface water samples.

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SECTION V

ENVIRONMENTAL ASSESSMENT

INTRODUCTION

The fate and transport characteristics of the compounds addressed in this report (chlorophenoxy herbicides, chlorobenzenes, chlorophenols) are discussed in this section. The concentration of a pollutant in the environment depends on the amount and form of the chemical released to the environment; the pathways for migration; the chemical and physical properties of the compound; the elapsed time from the release of the chemical to the sample collection and analysis and the behavior of the chemical in the environment. The fate and transport of the compound in the environment depends on the physical, chemical and/or biological processes which affect it.

Compound specific characteristics that may affect the concentration of a pollutant in the environment are: volatilization--the loss of organic compounds from the water to the air; and sorption--the ability of chemicals to adsorb or absorb to suspended and bottom sediments. Volatilization is an important pathway for compounds with high vapor pressures or low solubilities and the sorption to soil or sediments is generally by more hydrophobic compounds. Chemical processes include: photolysis--photochemical transformation of a compound through either direct or indirect reactions produced from excited states or chemical radicals; oxidation--free radical and singlet oxygen reactions; and hydrolysis--the introduction of a hydroxyl group (-OH) into a chemical structure. The biological processses bioconcentration/bioaccumulation and biotransformation and biodegradation involve the uptake and concentration of a compound by living species, and the enzyme-catylzed transformation and utilization of a compound by microorganisms respectively.

Other factors relevent to an environmental assessment include the migration route--air, surface water, ground water, sediments or surface runoff; the potential to migrate--geology, hydrology, climatic conditions, disposal methods and waste characteristics; the population at risk and the current regulations or guidelines.

CHLOROPHENOXY HERBICIDES

2,3,7,8-Tetrachloro-dibenzo-p-dioxin (dioxin)

Dioxin, produced as an unavoidable by-product and contaminant of the herbicide 2,4,5-T, has been discharged or disposed of at the Vertac site since 1948. It was originally discharged into Rocky Branch Creek via the production wastewater and later landfilled onsite as liquid and solid wastes. While data is still insufficient to conclude the most probable fate of dioxin in the environment, it appears that sorption and bioaccumulation are the primary processes affecting its transport.

The potential for dioxin to leave the site is supported by sample results showing dioxin as far as 50 miles downstream of its potential source. While recent sample results do not reveal detectable limits of dioxin in the surface or ground waters (its solubility is 0.2 ppb in water), it has shown up in sediment samples (0.839 - 9.981 ppb), fish (0.800 ppb) and surface soil samples. It has been generally found that dioxins are more tightly bound to soils having relatively higher organic content. Dioxins applied to the surface of such soils generally remain in the upper 6 to 12 inches. They migrate more deeply into sandier soils, to depths of 3 feet or more. In areas of heavy rainfall, not only is vertical migration enhanced but lateral displacement also occurs by soil erosion with runoff and/or flooding. Dioxins may appear in normal water leachate of soils that have received several dioxin applications. Transport of dioxin via surface waters appears to be primarily by erosion of contaminated soils and sediments. Dioxin contamination may also be transported offsite via airborne dust particles. Few studies have been done to determine whether dioxins accumulate in plants. In the limited numbers of studies conducted, results seem to indicate very small amounts, if any, are accumulated.

Dioxin is currently listed as 1) a toxic pollutant under the Clean Water Act Section 307 (A) 40CFR 129; 2) a toxic hazardous waste under RCRA 40CFR 261.33 (E); 3) a toxic substance under the Toxic Substances Control Act; and 4) as a poisonous solid, N.O.S. or poison B, solid, N.O.S. by the Department of Transportation. Current EPA levels acceptable are 0.050 ppb. EPA is proposing a short-term control plan for sites with dioxin levels above 1 ppb.

2,4-Dichlorophenoxyacetic acid (2,4-D)

2,4-D was produced at the site for the manufacture of Agent Orange. In February of 1982, there was an inventory of 9,472 drums of 2,4-D still bottoms. Recovery of 2,4-D wastes by Vertac began in July of 1982. Similarily to dioxin, 2,4-D was discharged and disposed of onsite in production wastewater and in landfills.

The still bottoms were originally stored onsite in leaking containers with no storage facilities provided.

2,4-D was found on site in both surface water (trace - 39 ppm) and ground water (trace - 98.5 ppm). It is not clear from the sample results provided whether 2,4-D was not found or not anablyzed for in the sediments. The presence of 2,4-D in part per million concentrations in the aqueous samples raises some questions as it is not very soluble (solubility is 0.07 ppm at 20 C). The most important factor with respect to the mobility of this compound in the environment is the organic matter content of the soil. 2,4-D is readily adsorbed by organic matter. In acidic systems it is adsorbed by clay particles. Therefore, the primary means of transport would tend to be via sediments and soil runoff in the aquatic systems and airbourne contaminated particulates.

The reported ppm concentrations of 2,4-D in the surface and ground water may represent a supersaturated solution. As the samples were collected in July, a higher solubility can be expected if the water temperatures were elevated. However, since the compound tends to be adsorbed to organic matter, the question of total suspended and total dissolved solids in the sample is raised. The highest concentrations of 2,4-D in surface waters were located in the production wastewater discharge ditch. The depth of water in the ditch was only 3 to 4 inches. 2,4-D was not found above the detection limit of 8 ppb int the cooling pond. In the groundwater samples 2,4-D concentrations were highest in the wells near the North Waste Burial Area and the Reasor-Hill Area. Both wells are located between the disposal areas and Rocky Branch Creek. There appear to be two probable migration routes of 2,4-D: 1) via onsite drainage ditches to adjacent surface waters and 2) by leaching from the disposal areas into the groundwater. Once the 2,4-D reaches surface water, its fate is uncertain. Because it does not show up in samples from cooling pond water at mid-depth and it tends to be adsorbed to organic matter, sediment samples should be analyzed for 2,4-D. This is supported by the specific gravity of 2,4-D (i.e is greater than 1). This indicates the compound would tend to sink to the lower strata. 2,4-D is not expected to bioaccumulate in the environment or enter the food chain. Other processes which affect the fate of 2,4-D in the environment are biotransformation/biodegration and photochemical decompositon. The presence of dioxin is 2,4-D as an impurity or breakdown substance increases the concern of environmental concentrations of 2,4-D.

2,4-dichlorophenoxyacetic acid is currently listed on 1) the Toxic Substances Control Act Inventory; 2) Clean Water Act Section 307 (A); 3) RCRA 40CFR 261.24 as characteristic of EP toxicity; 4) 40CFR 122.21 testing requirements for NPDES permit applications; 5) the Department of Transportation Hazardous MaterialsTable 49CFR 172.101 as Other Regulated Material ORM-A. 2,4-D is currently regulated under the Clean Water Act Section 304 (A); Water Quality Criteria for chlorophenoxy herbicides, at

100 ug/l (ppb) for domestic water supply (health); and under 40CFR 141.12 the National Interim Primary Drinking Water Regulations, Maximum Contamination Level (MCL) at 0.1 mg/l (100 ug/l).

2,4,5-Trichlorophenoxyacetic acid (2,4,5-T)

2,4,5-T was one of the major products produced at the Vertac site from 1948 until April 1979. As with 2,4-D and dioxin, 2,4,5-T was released into the environment by the discharge of production wastewater into the central ditch and Rocky Branch Creek and the onsite storage and disposal of production still bottoms. The presence of these compounds onsite and in the surrounding environments fours years after production ceased is indicative of the persistancy of the chemicals and/or quantities released.

2,4,5-T was found in both surface water (trace - 1.24 ppm) and groundwater (trace - 70.97 ppm) samples taken by DISC in July and August 1982. It is not clear whether 2,4,5-T was not found or not analyzed for in the sediment samples taken. 2,4,5-T is not very soluble (solubility is 0.03 ppm at 20 C). 2,4,5-T behaves similarly to 2,4-D with respect to its fate and transport in the environment. It is readily adsorbed by organic matter and under acidic conditions by clay particles. Therefore, the most important means of transport in the environment is via sediments and soil runoff and airborne contaminated particles. The organic matter content of the soil is the basis for the degree to which 2,4,5-T is adsorbed.

As discussed with respect to 2,4-D, the highest concentrations of 2,4,5-T were found in the onsite production wasterwater drainage ditches and the monitoring wells which lie between the burial areas (both the North Waste [(#12) and Reasor-Hill (#18)] and the cooling ponds and Rocky Branch Creek. the probable routes of migration are overland via the surface drainage ditches and by leachate migration into groundwater and surface water. Once the 2,4,5-T reached the surface water, its most likely fate will be adsorption to organic matter and sedimentation. Additional samples of the cooling pond and Rocky Branch Creek sediments should be analyzed for 2,4,5-T to determine if this is a potential route of migration. The advisory Committee on 2,4,5-T concluded that the herbicide is not bioaccumulated in the environment and is rapidly excreted by animals. It is therefore their belief that 2,4-D does not enter the food chain. Other processes which affect the fate of2,4,5-T in the environment are biotransformation/biodegradation and photochemical decomposition.

2,4,5-trichlorophenoxyacetic acid is currently listed on 1) the Toxic Substances Control Act Inventory; 2) Clean Water Act Section 307 (A); 3) RCRA 40CFR 261.24 as characteristic of EP toxicity; 4) 40CFR 122.21 testing requirements for NPDES permit applications; and 5) the Department of Transportation Hazardous Materials Table 49CFR 172.101 as Other Regulated Material ORM-A. 2,4,5-T is currently regulated under the Clean Water Act Section

304 (A) Water Quality Criteria for chlorophenozy herbicides at 10 ug/l (ppb) for domestic water supply (health) and under 40CFR 141.12 the National Interim Primary Drinking Water Regulations Maximum Contamination Level (MCL) at 0.01 mg/l (10 ug/l).

2 - (2,4,5-Trichorophenoxy) Propionic Acid [2 - (2,4,5-T) P]

Silvex

It cannot be determined at this time whether Silvex was intentionally manufactured at the Vertac Site, or was a result of 2,4,5-T reacting with heat during production. Silvex, along with the previously mentioned phenoxy herbicides, was released into the environment by discharge of wastewater and the onsite storage and disposal of still bottoms.

Silvex was found in both surface water (trace - 8 ppb) and groundwater (trace 45.0 ppb) samples taken by DISC in 1982. Since the properties of Silvex are similar to 2,4-D and 2,4,5-T the probable routes of migration are alike - overland via surface drainage ditches, and by leachate migration into groundwater and surface water. Furthermore, Silvex can be volatilized and enter the ambient air, adsorbed by organic matter and be degraded by micro-organisms.

2 - (2,4,5-Trichlorophenoxy) Propionic Acid - Silvex - is currently listed under the 1) Clean Water Act Section 311 (B) (4) and 2) RCRA Section 3001 as a hazardous substance. Silvex is currently regulated under the Clean Water Act Section 311 (B) (4) at 10 ug/l.

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CHLOROBENZENES

Chlorobenzene

Chlorobenzene was found on-site in both surface water (trace - 4030 ppb) and groundwater (trace to 5.4 ppb). This compound was not detected in sediment. Chlorobenzene, as other derivtives, is insoluble in water. It appears that primary transportation of these compounds could be from sediment runoff or suspended particlates in water. Chlorobenzene has an intermediate protential for biodegredation. This compound is expected to volatilize rapidly in surface waters.

It should be noted that the highest concentration of chlorobenzene was detected in a sample collected on August 13, 1982, from the center of the cooling pond at mid-depth. Subsequent sampling from September 17, 1982, of the north and south ends of the cooling pond at mid-depth revealed no chlorobenzene. The three other surface water samples showed a similar decline of chlorobenzene to the nondetectable level from August 13, 1982 to September 17, 1982. Concentrations of chlorobenzene in groundwater does not appear at represent a

threat to the environment.

The recommended Ambient Water Quality Criteria (AWQC) based on toxicity data to protect human health from the potential toxic effects of chlorobenzene, was determined to be 488 ppb. The recommended AWQC is 20 ppb chlorobenzene based on organoleptic data for controlling undesireable odors and tastes.

Chlorobenzene is currently listed on 1) the Toxic Substances Control Act Inventory; 2) Clean Water Act Section 307 (A); 3) RCRA 40CFR261.31 as an EPA hazardous waste; 4) 40CFR122.21 testing requirement for NPDES.

Dichlorobenzene

Dichlorobenzenes were detected in on-site groundwater (trace to 843.8 ppb) and sediment (trace to 2.5 ppb) at Vertac. The 1,2, 1,3, and 1,4 isomers of dichlorobenzene act similarly (i.e., are insoluble in water and appear to migrate in a similar fashion to chlorobenzene), and will be discussed together as dichlorobenzene. Dichlorobenzene wil bioaccumulate. The sorption process is substantial for dichlorobenzene. In addition, they volatilize rapidly in surface water, which may explain their absence in surface water samples. Dichlorobenzene is susceptible to attack by hydroxyl radicals in the atmosphere.

The low ppb concentration of dichlorobenzene in sediment does not represent a threat to the environment. Well number 6, sampled on July 23, 1982, was the only sampled groundwater location that showed dichlorobenzene. 1,4-dichlorobenzene was present at 843.8 ppb, while the 1,3 and 1,2 dichlorobenzene isomers were observed at 15.2 and 6.5 ppb respectively. It appears that contamination of this well by dichlorobenzene comes from the clay capped burial area.

Dichlorobenzenes in general show signs of acute toxicity to fw aquatic life at 1120 ppb and freshwater aquatic at 763 ppb. The recommended AWQC of 400 ppb has been established to protect human health from the toxic properties of dichlorobenzene.

1,4 Dichlorobenzene is currently listed on 1) the Toxic Substance Control Act Inventory; 2) the Clean Water Act Section 307 (A); 3) CERCLA Section 306 (A).

Trichlobenzene

The 1,2,4-isomer of trichlorobenzene was detected in groundwater (trace to 30.4 ppb), surface water (trace to 2.0 ppb) and sediment (trace to 2.0 ppb) samples at the Vertac Facility. 1,2,4-trichlorobenzene has a high potential for bioaccumulation in lipid tissues of organisms. Sorption plays a major role in the fate of this compound and 1,2,4-trichlorobenzene is expected to volatilize rapidly. This compound is reported to be

suscpectible to attack by hydrroxyl radicals in the atmosphere. Migration and transportation of trichlorobenzene appears to be similar to chlorbenzene.

Concentrations of trichlorobenzene found in sediments of the central ditch does not appear to represent a significant environmental threat. Similarly, 2 ppb of trichlorobenzene in surface water of the East ditch would not pose a threat. Well number 6 and 12 contain 39.6 ppb and 29.0 ppb of trichlorobenzene. Similarly with dichlorobenzene, trichlorobenzene appears to have entered the groundwater via leaching through the burial area. Rapid volatilization explains the absence of these compounds in surface waters.

Trichlorobenzene is currently listed on 1) the Toxic Substance Control Act Inventory; 2) the Clean Water Act Section 307 (A); 3) 40CFR122.21 testing requirements for NPDES; 4) RCRA 40CFR261.32, EPA hazardous waste.

Hexachlorobenzene

Hexachlorobenzene was found on-site in sediment in concentrations ranging from trace to 12.8 ppb. This compound is persistant and may bioaccumulate or enter the food chain. Hexachlorobenzene is insoluble in water and exhibits transportation characteristics similar to other chlorobenzenes. Sorption also appears to play a major role in the fate of this compound. Only cental ditch sediments contain detectable concentrations of hexachlorobenzene. It does not appear that this compound is migrating off-site via groundwater, surface water or sediment transportation routes. The 12.8 ppb concentration of hexachlorobenzene in sediment does not appear to represent a signicicant threat to the environment. Hexachlorobenzene is a suspected carcinogen and therefore has a recommended AWQC of zero. This level was assigned based on the non-threshold theory of carcinogenicity. Data indicates that freshwater aquatic species may exhibit acute toxicity to total chlorinated benzenes at concentrations as low as 250 ppb.

CHLOROPHENOLS

Five chlorinated phenolic compounds were detected on-site in sediment, surface and groundwater at Vertac. Only three sediment samples contained concentrations of chlorophenol ranging from 3.3 ppb to 20.1 ppb. These concentrations although they may indicate off-site migration of the above compounds (i.e., 20.1 ppb was detected in the sediment sample of Rocky Branch, below the cooling ponds), do not represent a significant threat to the environment in sediments.

Of concern is the concentraion of these chemicals in the part per million in range surface water and groundwater samples. Well numbers 9,12 and 18 are significantly contaminated (up to 166 ppm) with chlorophenol. The isomers of concern in groundwater

are 2-chlorophenol, 4-chloro-3-methyl phenol, 2,4-dichlorophenol, and 2,4 6-trichloropenol. Pentachlorophenol (PCP) was detected at 6 ppb over its detection limit in one groundwater sample.

Only surface water samples from the east and central ditches contain any of the chlorinated phenols (-20 pm at the east ditch and less than 1 ppm in the cental ditch).

It appears that these compounnds have entered or are entering groundwater via leaching from waste piles.

2-Chlorohenol is moderately toxic, somewhat persistant and slightly soluble in water. Acute toxicity of this compound to freshwater aquatic life occurs at concentrations as low as 4380 ug/l.

Data indicates that toxicity of chlorinated phenols to freshwater aquatic life generally increases with chlorination, the acute toxicity to freshwater species from 4-chloro-3-methyl-phenol occurs at 30 ug/l. Chronic toxicity occurs at 970 ug/l for 2,4,6-trichlorophenol.

Using organoliptic data for controlling undesirable taste and ordors, the recommend Ambient Water Quality Criteria for 2 chlorophenol is 0.1 ug/l. Neither volatilization, nor sorption, appear to affect the fate of 2 chlorophenol. This substance probably does not bioaccumulate either.

2-Chlorophenol is currently listed on 1) the TSCA Inventory; 2) CERCLA Section 306 (A); 3) 40CFR122.21 testing requirements for NPDES; 4) CWA Section 307 (A); 5) RCRA 40CRF261.32.

There is insufficient data to derive a level that would protect humans form the toxic effects of 4-chlorophenol. A recommended level of 0.1 ug/l prevents undesirable odors and tastes.

2,4-dichlorophenol is non-persistant and remains toxic only over short time periods or at limited distances from the pollutant source. This compound is a suspected carcinogen. 2-chlorophenol is persistant and tends to remain in the water column. This substance is nonbioaccumulative. Acute toxicity of 2, chlorophenol to freshwater aquatic life occurs at concentrations of 4380 ug/l. There is insufficient data to derive a level which would protect humans against the potential toxicity of this compound. Data from studies show ambient water quality criteria to protect against undesirable tastes and ordors is estimated at 0.1 ug/l (EPA, 1980). Mircrobial degradation of these 2 compounds has been reported.

Based on toxicity data for the protection of pulbic health, the recommended ambient water quality criteria for 2,4,5, trichlorophenol was determined to be 2.6 mg/l. However, 1.0 ug/l of this compound in water may produce foul taste and ordor.

2,4,6-trichlorophenol is persistant and non-bioaccumulative This compound is freely soluble in water. For the maximum protection of human health from the potential carcinogenic effects of exposure to this compound, the AWQC should be zero based on or non-threshold assumption of carcinogenicity. The levels of 2,4,6-trichorophenol that may result in 10-6 increased cancer risk over a lifetime is 1.2 ug/l (EPA, 1980). This compound sorbs to organic material.

2,4,6-trichlorophenol is currently listed on 1) the Toxic Substance Control Act Inventory; 2) Clear Water Act Section 307 (A); 3) CERCLA Section 306 (A); 4) RCRA f40CFR261.32.

Since no toxicity data is available for 4 chloro-3 methyl phenol, an ambient water level of 300 ug/l is suggested to avoid undesirable tastes and odors. This persistant compound shows no evidence of bioaccumulation.

4-chloro-3-methylphenol is currently listed on 1) the Toxic Substance Control Act Inventory; 2) CERCLA Section 306 (A); 3) CWA Section 307 (A).

Pentachlorophenol (PCP) is very persistent and may bioaccumulate and enter the food chain as well as biodegrade. Data indicates that chronic toxicity to freshwater aquatic life may occur at PCP levels as low as 3.2 ug/l. PCP is almost insoluble in water. Dusts of this compound may cause irritation. PCP also penetrates the skin. PCP is absorbed into leaf litteeer and other organic matter in the soil and sediments of freshwater sheds.

PCP is currently listed on 1) the Toxic Substance Control Act Inventory; 2) CWA Section 307 (A); 3) CERCLA Section 306 (A); 4) 40CFR122.21 testing requirements for NPDIS; 5) RCRA 40CFR261.31.

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SECTION VI

TOXICITY EVALUATION

INTRODUCTION

Available toxicological information has been reviewed for chemicals under evaluation at the Vertac Site. Pertinent experimental mammalian animal studies which may be supportive data for potential toxic effects and biological responses in humans are presented. These studies include acute and chronic toxicity, reproductive effects including teratogenicity, and carcinoginicity. Human health effects are discussed in terms of route of exposure, type of exposure, eg. acute or chronic exposure, and type of harmful or toxic effect, i.e. acute, chronic, local and systemis effects. Where the information is available, documentation on observed human health effects are presented, including clinical case histories or epidemiological studies. This review also includes some in vitro studies which pertain to potential toxic effects in humans and animals.

The major routes of entry of toxic agents into the body are through inhalation, ingestion and dermal absorption. The extent of exposure via these routes of entry depends on the particle size, volatility and solubility of the toxic agent. In the industrial environment, hazardous exposures are most often by inhalation or dermal absorption (topical). Inhalation- Toxic substances can be inhaled in the form of dusts, mists, aerosols, vapors, gases, fumes or smoke, where the substance are either absorbed or deposited in the lungs. The respirable fraction of airborne dusts is that fraction of particles in a size range which permits them to penetrate the lungs when inhaled. Particles having an aerodynamic diameter of 5 to 30 um are deposited mostly in the nasopharyngeal air passages; particles ranging from 1 to 5 um are deposited in the tracheobronchial regions; particles less than 1 um generally penetrate to the alveolar region and are deposited by sedimentation. Cutaneous Absorption-Some substances have a propensity for being absorbed through the skin, and in sufficient quantities can produce toxic effects. The cutaneous route of exposure includes absorption through the skin, mucous membranes and eyes, either by exposure to airborne concentrations or by direct contact with the liquid or solid substances. Ingestion-
Toxic substances can be orally ingested and absorbed by the gastrointestinal tract when they are present in the food chain or in drinking water. Occupational exposures by ingestion often occur when contaminated surfaces are touched or toxic materials are directly handled, and good hygiene practices are not followed, i.e., hands are not washed, or where smoking and eating in the contaminated area is permitted.

CHLOROPHENOXY HERBICIDES

2,4-dichlorophenoxy acetic acid and 2,4,5-trichlorophenoxyacetic acid are used in several hundred commercial formulations, and

sometimes combined in mixtures, e.g. Dacamine 2D/2T. Also in this group of compounds is 2-(2,4,5-trichlorophenoxy)proprionic acid, commonly known as Silvex.

Human Health Effects -These compounds and their salts and esters are moderately irritating to the eyes, skin and respiratory and gastrointestinal tract. Some of these compounds, such as 2,4-D, can enter the body through the skin. They also are absorbed from the lungs and through the gastrointestinal tract. Generally, they are considered to be of low to moderate toxicity. They do not remain stored in fat to a great degree, and are excreted within hours, or at the most, within days. There have been a limited number of reports in medical literature of toxic effects in humans from exposure to 2,4-D. Three cases of peripheral neuritis were reported for workers occupationally exposed to 2,4-D. Local depigmentation in some individuals has been attributed to prolonged and repeated dermal contact with chlorophenoxy compounds [1,2].

Single doses of 5 mg/kg of body weight of 2,4-D and 2,4-T were administered to human subjects without adverse effects. No adverse effects were seen in one person who consumed 500 mg of 2,4-D per day for 3 weeks [2].

Acute Effects - The signs and symptoms of acute poisoning from ingestion of large amounts of chlorophenoxy compounds are irritation of the mouth, throat and gastrointestinal tract, spontaneous emesis, chest pains (due to esophagitis), abdominal pain,and diarrhea. Injury of the gastrointestinal tract usually does not progress to ulceration and perforation. Once compounds are absorbed they can cause fibrillary muscle twitching,skeletal muscle tenderness, and myatonia (stiffness of extremities). Symptoms associated with ingestion of very large amounts of chlorophenoxy acids are metabolic acidosis, fever, tachycardia, hyperventilation, vasodilation and sweating. Some cases have been characterized by coma and convulsions [2, p.29].

Effects in Experimental Animals - The animal toxicity of 2,4-D and 2,4.5-T is similar. The low cumulative effect of these compounds has been demonstrated in feeding studies where animals tolerated repeated exposures to doses slightly smaller than the single toxic dose [3].

Based on animal studies done by Rowe and Hymas, it was concluded that 2,4-D had a low chronic toxicity. For 2,4-D, oral LD50 values for several animal species ranged from 100 to 1,000 mg/kg. For 2,4,5-T, oral LD50 values ranged from 300 to 1,000 mg/kg for several species. Dogs were found to be more sensitive than other animal species, with an LD50 of 100 mg/kg for 2,4,5-T isopropylestec [4]. In animal experiments, large doses of 2,4-D caused vomiting, diarrhea, anorexia, weight loss, ulcers of the mouth and pharnyx, and injury to the kidney and liver, and central nervous system. Muscular effects have been seen in some species, specifially myatonia, or stiffness of the extremeties, which was apparently due to CNS damage. Also, in heavily dosed

animals, demyelination was observed in the dorsel columns of the cord, and EEG changes indicated functional disturbances in the brain.

2,4 Dichlorophenoxyacetic Acid (2,4-D)

In March 1979 the Environmental Protection Agency declared an emergency suspension of 2,4,5 trichlorophenoxyacetic acid (2,4,5-T), which contains tetrachloro-dioxin (TCDD) as a contaminant. This action generated public concern about the possibility of dioxin contamination in 2,4-D and the potential adverse health effects of TCDD contaminated 2,4-D. The concern was focused on the potential for cancer and miscarriages from exposure to TCDD in 2,4-D. Further concern about 2,4-D developed as a result of the Agent Orange contraversy. This defolient used during the Vietnam War, was composed of 2,4,5-T and 2,4-D, and was never registered by EPA for civilian use in the United States. In response to public concern, the EPA initiated a review of available toxicological data on the potential health effects of 2,4-D. Conclusions regarding potential human health effects were published in the April 22, 1980 Fact Sheet. EPA has based these conclusions on the acid form of 2,4-D, although there are many 2,4-D salt and ester derivatives. Based on their review, EPA concluded that 2,4-D was of low to moderate toxicity, and did not pose an imminent health hazard when used properly. Based on available studies, 2,4-D is not known to be carcinogenic in animals or humans. Results of mutagenicity tests have been mixed; the majority of results have been negative, while there have been three positive results reported. Reproductive studies conducted on mice, rats and hamsters showed slight fetotoxic effects, at lower dose levels, including edema (swelling of tissues.) Very high dose levels caused skeletal malformations and cleft palates.

In the fall of 1980 the EPA initiated a monitoring program to test for dioxin contaminants in commercial 2,4-D products. This program was the result of a ban by the Agriculture Canada of some 2,4-D products which were found to contain 2,7 dichlorodibenzo-p-dioxin and 1,3,7 trichlorodibenzo-p-dioxin. The most toxic of the dioxins 2,3,7,8 TCDD was not found in the samples taken in Canada. EPA Reports (2,4-D Fact Sheets) were released on January 23, 1981 and July 21, 1981, which concluded that the majority of samples taken during the monitoring program were free of any form of dioxin, while a small percentage of the samples contained very low levels of the toxic dioxin isomers which are of a much lower degree of toxicity than 2,3,7,8 TCDD. [1,2]

2,4,5-Trichlorophenoxyacetic Acid

The mammalian toxicity of 2,4,5-T is low. It is slightly irritating to the skin, and overexposure to this compound may cause abdominal pain, nausea, vomiting, diarrhea and blood in the stool. Dioxin isomers are unwanted contaminants in 2,4,5-T, especially the extremely toxic isomer, 2,3,7,8-TCDD. Chloracne seen in industrial workers who worked in a 2,4,5-T

manufacturing facility, was attributed to the TCDD contaminants (2,3,7,8-TCDD or 2,3,6,7-TCDD).

Several incidents of human exposure to 2,3,7,8-TCDD have involved the 2,4,5-T herbicide compound. In March 1979, the EPA declared an emergency suspension of 2,4,5-T and Silvex [2-(2,4,5-trichlorophenoxy)proprionic acid], because of a reported increase in miscarriages in humans in Alsea, Oregon an area where the herbicides were sprayed. These effects were believed to be a result of exposure to the dioxin contaminant in 2,4,5-T, and possible contamination of Silvex, because of its similarity to 2,4,5-T. The defoliant, Agent Orange, used during the Vietnam War, is a 50:50 mixture of 2,4-D and 2,4,5-T, and is alleged to have caused serious medical problems in many Vietnam war veterans.
Long term exposure to dioxin contaminants in 2,4,5-T may cause chloracne and liver damage. Hepatic toxicity resulting from exposure to 2,3,7,8-TCDD has been demonstrated in animal studies, and has been observed in human workers after industrial exposure. Teratogenic, embryotoxic and fetotoxic effects have been produced in animals exposed to 2,4,5-T containing 2,3,7,8-TCDD, and have been attributed to the teratogenic and fetotoxic potential of 2,3,7,8-TCDD.

Some positive results have been reported on mutogenicity tests done on 2,4,5-T[7]. 2,4,5-T is not known to be carcinogenic in humans or animals.

Dioxins

Unwanted contaminants in 2,4,5-Trichlorophenoxyacetic acid have been 2,3,7,8-tetrachlorodibenzo-p-dioxin and other dioxin isomers, especially 2,3,6,7-tetrachlorodibenzo-p-dioxin. 2,3,7,8-TCDD is a potent animal teratogen and causes severe chloracne in man. 2,3,6,7-TCDD is a potent acnegenic agent and is hepatoxic in animals. On a molecular basis 2,3,7,8-TCDD is perhaps the most poisonous synthetic chemical. Not only is this TCDD isomer extremely poisonous but it also has extremely high potential for producing adverse effects under conditions of chronic exposure. Human exposure to 2,3,7,8-TCDD has induced chloracne, polyneuropathy, mystagmus, and liver dysfunction as manifested by hepatomegay and enzyme elevations. In animals, this compound has shown to be teratogenic, embryotoxic, carcinogenic, and cocarcinogenic. It has been established that under certain conditions 2,3,7,8-TCDD can enter the human body from a 2,4,5-T treated food chain and can accumulate in the fatty tissues and secretions, including milk. Estimates done by accepted risk assessment procedures indicate that daily human exposure to 0.01 ug of 2,3,7,8-TCDD is the dosage expected to result in "incipient carcinogenicity". Additionally, daily human exposure to 4 mg of 2,3,7,8 TCDD would be expected to result in a shortened lifespan, and daily exposure to 290 mg would likely result in acute toxicity [5, p.147].

ORGANOCHLORINE COMPOUNDS

Human Health Effects -Major routes of entry are inhalation of vapors and percutaneous absorption of the liquid. Most of these compounds are readily absorbed through the skin and from the gastrointestinal tract. When dosage is adequate, most organochlorine compounds interfere with axionic transmission of nerve impulses resulting in disruption of the nervous system functions and brain. These disruptions include behaviorial changes, sensory and equilibrium disturbances, involuntary muscle activity, and depression of vital centers including respiration.

Acute Effects - Poisoning from organochlorine compounds will result in the following symptoms: apprehension, excitability, dizziness, headache, disorientation, weakness, paresthesiae, muscle twitching, tremor, tonic and clonic convulsions, and unconsciousness. Nausea and vomiting occur soon after ingestion. First symptoms from dermal absorption are apprehension, twitching, tremors, confusion and convulsions. Other symptoms are respiratory depression, pallor which occurs in moderate to severe poisonings, and cyanosis as a result of respiratory interference from convulsions [2, p. 14-15].

CHLORINATED BENZENES

1,2-dichlorobenzene is used as a fumigant, insecticide, solvent and chemical intermediate. 1,4-dichlorobenzene is used as an insecticide, disinfectant, moth preventative and chemical intermediate. Hexachlorobenzene finds use as a seed protectant. Other chlorinated benzenes are used as insecticides and solvents.

Chlorinated benzenes are irritating to the eyes, skin and upper respiratory tract. Acute exposures to these compounds may cause drowsiness, incoordination and unconsciousness. Animal studies have shown damage to the liver, kidney and lungs as a result of chronic exposures [1, p. 258].

Hexachlorobenzene

Hexachlorobenzene is an organochlorine pesticide used primarily as a fungicide. Mass poisonings from HCB occurred in Turkey where several thousand citizens ate wheat that had been treated with the chemical. Prolonged ingestion of treated wheat produced porphyria cutanea tarda. This disease is characterized by extreme skin manifestations including bullous dermatitis, deep scarring, permanent loss of hair, skin atrophy, and hyperpigmentation. Other symptoms were excretion of red urine, muscle wasting and liver enlargement [3, p. 394].

Limited information is available on the chronic toxicity associated with long term exposure to hexachlorobenzene. It is known that hexachlorobenzene is carcinogenic in some animal species, and is a suspected human carcinogen [7,8,9]

Chlorobenzene

Effects in Experimental Animals-Chlorobenzene has produced narcotic effects in test animals at 1200 ppm. Levels from 220 to 660 ppm were tolerated [4,p.49;11] Cats showed severe narcosis from exposure to 8000 ppm after 1/2 hour, and died 2 hours after the exposure. Chronic exposures of several animal species to 1000 ppm for 7 hours/day, 5 days/week over a period of 44 days, resulted in observable histopathological changes to the liver, kidney and lungs. In this experiment, guinea pigs exposed to 475 ppm showed slight histopathological changes in the liver [6, Chlorobenzene]. The hematapoietic effect on the blood which can result from exposure to benzene has not been associated with exposure to chlorobenzene.

Human Health Effects- Irritation to the eyes and mucous membranes begin to occur at 200 ppm, and there is a pronounced odor. Prolonged or repeated skin contact may cause skin burns. Liver, kidney and lung damage may occur after prolonged exposures to chlorobenzene.

Dichlorobenzene

Effects in Experimental Animals - 1,2-Dichlorobenzene has been found to cause liver and kidney damage in animals. In a study by Cameron and Thomas cited in the ACGIH Documentation for Threshold Limit Values, liver damage was found in animals which were exposed to concentrations ranging from 50 to 800ppm for a few hours. (4) Rats exposed to 977 ppm for 7 {4, p.76} hours died. They survived when exposed to two hours at these levels. After 3 hours of exposure to 539 ppm, animals survived, but necrosis of the liver and kidney changes were found at necropsy [6, O-Dichlorobenzene].

Human Health Effects-O-Dichlorobenzene is irritating to the eyes and upper respiratory tract. Liquid in contact with the skin or eyes can cause burns. Eye irritation from exposure to airborne levels becomes noticeable at 25 to 30 ppm, and painful to some at 60 to 100 ppm for exposures lasting more than a few minutes. This compound has been known to cause sensitization dermatitis [6]. Chronic exposure to high concentrations may result in damage to the liver and kidneys. O-dichlorobenzene is not known to be carcinogenic in humans or animals.

P-Dichlorobenzene

Effects in Experimental Animals-Liver necrosis has been found in animal studies with rabbits and rats. Chronic exposure of animals to 798 ppm resulted in eye irritation, marked tremors, weakness, loss of weight, and some deaths; nonspecific eye changes were noted in rabbits as well as liver necrosis, and kidney and lung damage [6, p-Dichlorobenzene]. Slight liver necrosis was found in some rats injected with 0.005 g of PDB [4, p. 77;12].

Human Health Effects-p-dichlorobenzene causes irritation to the eyes, nose and throat. Exposure may result in headache, swelling of the eyes and running nose. Prolonged overexposure may cause weight loss, loss of appetite, nausea, vomiting, anorexia, and liver damage with jaundice. Clinical cases have been cited in literature where persons who experienced prolonged exposure developed these symptoms, but the level of exposure was not indicated [4,6].

1,2,4 Trichlorobenzene

Effects in Experimental Animals- As with the mono and dichlorobenzenes, animal studies with 1,2,4-trichlorobenzene have produced liver and kidney damage. Adverse effects were seen in acute and subacute inhalation studies where animals were exposed to 1,2,3/1,2,4 trichlorobenzene (8%/92% weight/weight). It was determined from the study that the target organs from nonlethal exposures of cats, dogs, rats, rabbits and guinea pigs included the liver, kidney, ganglion cells at all brain levels, and mucous membranes [4, p. 400]. Slight adverse effects were seen in rat inhalation studies where animals were exposed to levels ranging from 0 to 100 ppm. In one experiment, 20 male rats, 4 rabbits and 2 male dogs were exposed for 7 hours/day, 5 days/week for 6 weeks to 30 ppm or 100 ppm of 1,2,4-trichlorobenzene, (98.4% purity, 1.4% 1,2,3-trichlorobenzene. Adverse effects were not seen at 30 ppm except for elevations in urinary uroporphyrin and coproporphyrin in rats only at 15 and 30 exposure days. In another study performed with 99.07% pure 1,2,4 trichlorobenzene, rats, rabbits and monkeys were exposed at 0, 25, 50 and 100 ppm for 7 hours/day, 5 days/week for 26 weeks. Microscopic liver and kidney changes were observed only in the rat groups. Rats appeared to be more susceptible to 1,2,4-TCB than other species in which systemic damages were not found [4, p. 401].

Human Health Effects- 1,2,4 Trichlorobenzene is irritating to the eyes and respiratory tract and can cause dermal irritation. Irritation from industrial exposures has occurred at levels as low as 5 ppm (4). Contact with the liquid can cause burns to the eyes and skin. Prolonged overexposures may result in damage to [4, p. 401] liver, kidneys and lungs. Very limited data is available on the human health effects or potential effects from 1,2,4 TCB and 1,2,3 TCB.

CHLOROPHENOLS

The degradation of herbicides 2,4-dichlorophenoxyacetic acid and 2,4,5-trichlorophenoxyacetic acid to chlorophenol isomers is on important source of human exposure to these compounds in the environment. Other sources of exposure are (1) from chlorination of phenols present in natural water and in effluents from waste treatment plants, (2) direct addition of chemicals from industrial sources, (3) wet and dry atmospheric fallout.

2-Chlorophenol is used as a starting material for higher chlorophenol isomers. Synthesis of herbicides 2,4-D and 2,4,5-T involve 2,4-dichlorophenol and 2,4,5-trichlorophenol as intermediates. 2,4,5-trichlorophenol and 2,4,6-trichlorophenol and their salts are used a germicides in the preservation of wood, leather and other materials. Pentachlorophenol has been use for several decades as a wood preservative and fungicide. It is ubiquitous in the environment; low levels have been detected in sewer water, municipal water supplies, human food stuffs, and in blood, urine and fat of nonoccupationally exposed persons [10].

Generally, the toxicity of chlorophenols to higher organisms is greater as the degree of chlorination of the isomer increases. However, there are exceptions; based on LD 50 values, 2,4-dichlorophenol and the trichlorophenols (2,4,5 and 2,4,6) are less toxic than 2-chlorophenol, and 2,4 dichlorophenol is less readily absorbed through the skin. Pentachlorophenol is the most toxic of the chlorophenol isomers.

Information on effect of long term exposures and chronic toxicity in humans of chlorophenol compounds has not been documented. The extremely poisonous compound 2,3,7,8-tetrachlorodibenzo-p-dioxin is a known impurity in technical grade formulations of chlorophenols, and may be responsible for reported industrial cases of chloracne.

Of the compounds under discussion, 2-chlorophenol, 2,4-dichlorophenol, 4 chloro-3methyl phenol and pentachlorophenol are not known to be carcinogenic in humans or in laboratory animals. Results of a carcinogen bioassay, completed under the National Institute for Cancer testing program, have identified 2,4,6-trichlorophenol as a positve animal carcinogen in mice and rats [7]. Some positive results from mutagenicity tests have been reported for two isomers, 2-chlorophenol and 2,4,6-trichlorophenol [7].

2-Chlorophenol and 2,4-Dichlorophenol

Human Health Effects- Mono and dichlorophenols will burn the skin and eyes. These compounds can be absorbed through the skin - 2-chlorophenol is readily absorbed through the skin, whereas 2,4-dichlorophenol is less readily absorbed. Symptoms of acute poisoning from 2-4-dichlorophenol include tremors, convulsions,

shortness of breath and inhibition of the respiratory system.

Data on the absorption, distribution, metabolism and elimination in humans of these compounds is not available. Low level or chronic effects from human exposure to 2-chlorophenol has not been documented. One study suggested adverse effects in humans from exposure to 2,4-dichlorophenol. Chemical workers involved in the manufacture of 2,4-dichlorophenol and 2,4,5-trichlorophenol developed chloracne and porphyria. Workers were exposed to several other chemicals, including possible exposure to tetrachlorodibenzo-p-dioxin [10, p. 119;13], so that it is uncertain how much 2,4-dichlorophenol contributed to these toxic effects.

Effects in Experimental Animals - Based on available animal toxicity data for oral LD 50 values, lower chlorophenols can be considered of low to moderate toxicity. LD 50 values for 2,4-dichlorophenol via the oral intraperitoneal, and subcutaneous routes of administration ranged from 430 to 1730 mg/kg in one study [10, p. 67]. For 2-chlorophenol, the median lethal doses range from 100 to 800 mg/kg body weight [10, p. 67].

Animal studies have indicated that 2-chlorophenol is excreted primarily in the urine in both free and conjugated forms. The ability of 2-chlorophenol and 2,4-dichlorophenol to inhibit oxidative phosphorylation in vitro tests has been suggested as a mechanism of toxic action [10, p. 117;14]. Lethal dosages of 2-chlorophenol administered to rats via subcutaneous, intraperitoneal and oral routes, produced restlessness, increased respiration, motor weakness, tremors, convulsions, dyspnea, coma and death. Pathological changes caused by lethal concentrations were kidney and liver damage, and hemorrhaging in the intestine.

2-4-dichlorophenol is moderately toxic to animals. Male mice receiving a daily dose of 230 mg/kg for 6 months showed no adverse changes in behavior, growth rate, or blood glutamic oxalacetate and glutamic or pyruvate transaminase levels; minor histopathological changes were found in the liver.

2,4,6-Trichlorophenol

Trichlorophenols gererally are considered to be mildly toxic, with 2,4,5- and 2,4,6-trichlorophenol appearing to be least toxic of the higher chlorophenols (oral LD 50 values as high as 3 g/kg of body weight in rats have been reported).

Human Health Effects- Contact may cause moderate skin irritation, eye irritation and possible corneal injury. Dusts may be very irritating to the nose and throat.

Documented cases of human toxicity due to trichlorophenol or tetrachlorophenol exposure have not been reported. Cases of acneform dermatitis from continuous daily exposure to trichlorophenol and tetrachlorophenol formulations have been reported. It has been suggested that the impurity 2,3,7,8-

tetrachlorodibenzo-p-dioxin, may have been involved in causing chloracne, present in technical grade 2,4,5-trichlorophenol may have been involved in causing chloracne among industrial workers in reported cases [10, p. 203]. In many reported cases, employees were exposed to a variety of compounds, and symptoms often cannot be attributed to a specific chlorophenol compound.

2,4,6-Trichlorophenol is not readily absorbed through the skin. This conclusion has been based on animal studies which indicated that 2,4,5 and 2,4,6-trichlorophenol did not penetrate rabbit or guinea pig skin. It has been concluded also from animal studies that these compounds are absorbed readily from the gastrointestinal tract. Information on rates of absorption from these compounds in humans and animals is not available. 2,4,5 and 2,4,6-trichlorophenol and some tetrachlorophenols are excreted primarily in the urine. In animal studies, compounds have been excreted in the free forms and as conjugates of sulfuric and glucuronic acids [10, p. 199]. Information on the transport, distribution, metabolism, and elimination of higher clorophenols in humans and animals is not available or not conclusive.

Effects in Experimental Animals- Trichlorophenols and tetrachlorophenols have the ability to uncouple oxidative phosphorylation, which may contribute to their toxic action, or may be the primary mechanism in experimental animals [10, p. 200]. 2,4,6-trichlorophenol inhibits the enzymes lactate dehydrogenase and hexokinase in in vitro systems [10, p. 201].

Acute Toxicity - In a study done by Farquharson, Gage, and Northover (1958), in which rats were administered lethol doses of twelve chlorophenol isomers, it was found that 2,4,6-trichlorophenol has a convulsive action in experimental animals. Lethal doses administered to experimental rats produced intermittant convulsions , followed by loss of righting reflex, dyspnea, coma and death. For other compounds including 2,4,5-trichlorophenol and 2,3,4,6-tetrachlorophenol, symptoms of poisoning did not include this convulsive action [10, p. 201;15].

Injections of 2,3,4,6-tetrachlorophenol produced a marked rise in temperature, whereas 2,4,6-trichlorophenol caused only a slight elevation in temperature. Other effects of poisoning which were noted in the animals were chromodacryorrhea, lacrimation, saliration, and diarrhea [10, p. 202;15].

Chronic Toxicity - In a chronic feeding study where rats were maintained on diets with daily intakes of 10,30,100,300 or 1000 mg/kg of 2,4,5-trichlorophenol, no adverse effects were seen in doses up to 100 mg/kg. Minor microscopic damage in the kidneys or liver were seen in rats receiving 300mg/kg or 1000 mg/g. When rabbits were fed 20 oral doses by intubation in a 29 day period, very slight kidney changes were seen at 100 mg/kg, and very slight kidney and liver changes were seen at doses of 500 mg/kg [10, p. 202;16].

Carcinogenicity - 2,4,5- and 2,4,6-trichlorophenol are not known to be carcinogenic in animals or humans. 2,4,5-trichlorophenol has produced benign and malignant trumors in mice when used with an initiator, dimethylbenzanthracene. The compound does not appear to be tumoragenic in the absence of the initiator. 2,4,6-trichlorophenol has not been tumoragenic in tests.
Teratogenicity - Information on reproductive effects of 2,4,6-trichlorophenol is not available.

Pentachlorophenol

Pentachlorophenol is a compound which has been used for many years primarily as a wood preservative. The acute toxicity of this compound is very high. Commercially produced pentachlorophenol may be contaminated with dioxin compounds which are more toxic than pentachlorophenol. [6, Pentachlorophenol].

Effects in Experimental Animals- The acute toxicity of pentachlorophenol is high (oral LD 50 for rats, 50 mg/kg; oral LD 50 for rabbits 70 mg/kg; oral LD 50 for hamsters, 168 mg/kg).

Chronic effects from continuous exposure to low doses have not been clearly demonstrated in experimental animals. Subchronic and chronic feeding studies have been done in several mammalian species. In a 90 day feeding study with two groups of male rats fed pure pentachlorophenol (containing low levels of chlorodibenzo-p-dioxin contaminants) and technical grade pentachlorophenol (containing relatively high levels of these contaminants) at doses equivalent to approximately 50 mg/kg body weight per day, pathologic changes in the liver were seen when examined microscopically [10, p. 389; 21]. A dose related decrease in calcium deposits in the kidneys of rats given dietary doses of pentachlorophenol at 25, 50 and 200 mg/kg body weight for 12 weeks. [10, p. 391; 22].

The compound has been shown to cause adverse reproductive effects in rats (See "Teratogenicity").

Human Health Effects- Pentachlorophenol is a highly toxic compound which is readily absorbed through the skin. Acute systemic toxicity in humans can occur following absorption through the respiratory tract, gastrointestinal tract and skin. This compound is rapidly absorbed through the gastrointestinal tract following ingestion. In cases of severe or fatal poisonings, symptoms include loss of appetite, respiratory difficulties, anasthesia, hyperpyrexia, sweating, dyspnea and rapidly progressive coma. Many cases of human intoxication have been reported, most of which involved direct absorption of pentachlorophenol or its sodium salt through the skin. Cases of pentachlorophenol poisoning which resulted primarily from inhalation of vapors or dusts have been reported as well. Acute poisoning from pentachlorophenol centers in the circulatory system and is accompanied by heart failure. [4, p. 198]. The physiologic injury which results from poisoning is mainly

vascular. Pentachlorophenol dust and mist cause irritation of the eyes and upper respiratory tract; absorption results in an increase in metabolic rate and hyperpyrexia; prolonged skin exposure causes an acneform dermatitis. Human exposure to dust or mist concentrations greater than 1 mg/M_3 causes pain in the nose and throat, and violent sneezing and cough; $0.3Mg/M_3$may cause some nose irritation. Persons who work routinely with pentachlorphenol may have some tolerance to these respiratory effects, and may tolerate airborne concentrations up to 2.4 mg/m_3. Systemic intoxication is cumulative and has been fatal. Intoxication is characterized by weakness anorexia, weight loss, and profuse sweating; there also may be headache, dizziness, nausea, vomiting, dyspnea, and chest pain. In fatal cases, the body temperatue is frequently extremely high and death has occurred as early as 3 hours after the onset of symptoms. Other effects which may result from repeated exposure to pentachlorophenol are acneform dermatitis, bronchitis, and liver damage. [6, Pentachlorophenol].

Minimum lethal concentrations for pentachlorophenol in air have not been defined. The treshold limit value, which is based on an 8 hour time weighted average exposure is 0.5 mg/m_3 [4]. The risk of intoxication via inhalation is greater during hot weather; although the vapor pressure of PCP is low (.00011 to 0.12 mmHg at 20 C to 100 C) toxic levels of vapor can build up in hot, enclosed areas.

The exact dosage which produces illness in humans is not known. An oral lethal dose in humans of 29 mg/kg has been reported [10, p. 239]. Symptoms of poisoning occur at concentrations of 40 to 80 mg/liter in the blood. In fatal cases, blood levels have ranged from 46 to 156 mg/liter, and urine levels from 28 to 520 mg/liter [10, p. 382].

For acute intoxications, the urine pentachlorophenol concentration is frequently higher than the blood level. In humans and animals, pentachlorophenol is excreted primarily through the urine. One study suggested a ratio of 1.5 to 2.5 of pentachlorophenol in blood to pentachloropehnol in urine in humans [10, p. 369;17]. Initial urinary elimination following exposure to pentachlorophenol may be rapid, but return to background levels may take a month or longer. Approximately 50% of the body load is excreted in the urine in 24 hours, and 70% to 80% is excreted in four days [10, p. 237;18]. Renal competancy, that is, the capacity of the renal system to handle the pentachlorphenol load, appears to be a factor in the extent of individual susceptibility to pentachlorophenol poisoning [10, p. 239].

Long term chronic effects from exposure to low levels of pentachlorophenol have not been seen in humans. Low background levels of PCP have been found in the blood and urine of occupationally and non-occupationally exposed persons, but chronic effects from these levels have not been reported. A reversible effect on the kidney has been seen, where PCP exposure caused a decreased creatinine clearance and phosphorous reabsorption in the kidney. These effects were seen in workers chronically exposed to PCP in the wood treatment industry. Wood treaters were tested before, during, and after vacation, and significant differences were seen in blood and urine phosphorous levels and in creatinine clearance [10, p. 400;19]. In one study it was found that workers continuously exposed to PCP had elevated levels of gamma-mobility C-reactive protein in the serum. Elevated C-reactive protein levels are associated with inflammatory disorders and tissue damage, and it was inferred that PCP exposure may produce inflammation or tissue or damage [10, p. 401;20].

Carcinogenicity- No evidence exists that pentachlorophenol is carcinogenic in humans and animals.

Teratogenicity - This compound does not appear to be teratogenic in rats. However, embrytoxic and fetotoxic effects have been observed in experiments with rats. Developmental effects were

observed when high doses of pentachlorophenol were administered to maternal rats, which could have been due to direct toxic effect on the maternal rat, as placental transfer of the compound is minimal [10, p. 409;23].

Mutagenicity - Some positive results have been reported from mutagenicity testing of pentachlorophenol [7].

TOLUENE

Human Health Effects -Routes of exposure - Toluene can effect the body if it is inhaled, if it comes in contact with the eyes or skin, or if it is swallowed. It may enter the body through the skin. Short term exposure - Toluene may cause irritation of the eyes, respiratory tract, and skin. It may also cause drowsiness. Peculiar skin sensation may be produced such as a "pins and needles" feeling or numbness. Very high concentrations may cause unconsciousness and death. The liquid splashed in the eye may cause irritation and temporary damage. Long term exposure - Repeated or prolonged exposure to liquid toluene may cause drying and cracking of the skin. Toluene vapors cause narcosis. Controlled exposure of human subjects to 200 ppm for 8 hours produced mild fatigue, weakness, confusion, lacrimation, and paresthesia; at 600 ppb for 8 hours there was also euphoria, headache, dizziness, dilated pupils and nausea; at 800 ppm for 8 hours, symtpoms were more pronounced, and after effects included nervous-ness, muscular fatigue, and insomnia persisting for several days [6, Toluene].

SECTION VI

TABLE I

Permissable Exposure Limits for Occupational Exposure to Airborne Contaminants

Vertac Site, Jacksonville, Arkansas

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Permissable Exposure Limits for Occupational Exposures to Airborne Contaminants

Chemical Groups & Compounds	OSHA[1] Permissable Limits		ACGIH[2] Threshold Limit Values				NIOSH[3] Recommended Allowable Levels	
	PPM[4]	TWA MG/M^3 (5)	PPM	TWA[6] MG/M^3	PPM	STEL[7] MG/M^3	PPM	MG/M^3
2,4-Dichlorophenoxyacetic acid (2,4-D)	-	10	-	10	-	20		
2,4,5-Trichlorophenoxyacetic acid (2,4,5-T)	-	10	-	10	-	20		
2-(2,4,5-Trichlorophenoxy) Propionic acid	-	-	-	-	-	-		
2,3,7,8-Tetrachlorodibenzo —p-dioxin	-	-	-	-	-	-		
2-Chlorophenol	-	-	-	-	-	-		
4-Chloro-3 methyl phenol	-	-	-	-	-	-		
2,4-Dichlorophenol	-	-	-	-	-	-		
2,4,6-Trichlorophenol	-	-	-	-	-	-		
Pentachlorophenol	-	0.5 (skin[8])	-	0.5 (skin)	-	-		

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Permissable Exposure Limits for Occupational Exposures to Airborne Contaminants (cont'd.)

Chemical Groups & Compounds	OSHA[1] Permissable Limits		ACGIH[2] Threshold Limit Values				NIOSH[3] Recommended Allowable Levels	
	TWA		TWA		STEL[7]			
	PPM[4]	MG/M^3	PPM	MG/M^3	PPM	MG/M^3	PPM	MG/M^3
1,3-Dichlorobenzene	-	-	-	-	-	-	-	-
1,4-Dichlorobenzene	75	450	75	450	110	675	-	-
1,2-Dichlorobenzene	C[9] 50	C 300	C 50	C 300	-	-	-	-
1,2,4-Trichlorobenzene	-	-	5	40	-	-	-	-
1,2,3-Trichlorobenzene	-	-	-	-	-	-	-	-
Hexachlorobenzene	-	-	-	-	-	-	-	-
Chlorobenzene	75	350	75	350	-	-	-	-
2-Chloroanisole	-	-	-	-	-	-	-	-
2,4-Dichloroanisole	-	-	-	-	-	-	-	-
4-Chloroanisole	-	-	-	-	-	-	-	-
Toluene	200 300 (Acceptable Ceiling Concentration) 500 (Acceptable Maximum peak above the acceptable ceiling concentrations)	-	100 (skin) - -	375 - -	150 - -	560 - -	100 C 200 (10 minutes)	

TABLE I

NOTES

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1. OSHA - Occupational Safety and Health Administration, General Industry Standards, 29CFR 1910.1000 (a) Table Z-1, and (b) Table Z-2.

2. ACGIH - American Conference of Governmental Industrial Hygienists, Threshold Limit Values for Chemical Substances and Physical Agents in the Work Environment with Intended Changes for 1983-84.

3. NIOSH - National Institute for Occupational Safety and Health, Recommended Standard for Toluene.

4. PPM - Parts of vapor of gas per million parts of contaminated air by volume at 25° C and 760mm Hg pressure.

5. MG/M3 - Approximate milligrams of particulate per cubic foot of air.

6. TWA - Time Wighted Average: permissable exposure limits are 8-hour time weighted average limits. An employee's cumulative exposure for an entire 8-hour work shift of a 40-hour work week shall not exceed the 8-hour time weighted average limit. Excursions are permitted above the limit as long as they are compensated by excursions below the limit, and as long as the weighted average of the exposures for the entire 8-hour period does not exceed the limit. The exception is where ceiling values are designated (see note No. 9).

7. STEL - Short Term Exposure Limit: The concentration to which workers can be exposed continuously for a short period of time without suffering from (1) irritation, (2) chronic or irreversible tissue damage, or (3) narcosis of sufficient degree to increase the likelihood of accidental injury, impair self-rescue or materially reduce work efficiency, and provided that the daily TLV-TWA is not exceeded (ACGIH, Threshold Limit Values, 1983-84).

8. SKIN - this notation refers to the potential contribution to the overall exposure by the cutaneous route which includes eyes and mucous membranes, either by airborne exposure or by direct contact with the substance.

9. C- Ceiling Limits: The limit which concentrations should not be permitted to exceed at any time during an 8-hour work shift. Ceiling limits are applied to substances which are fast acting, that is they produce an immediate toxic response, e.g. extreme irritation.

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SECTION VI

TABLE II

Warning Properties of Chemicals Under Evaluation

Vertac Site, Jacksonville, Arkansas

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ODOR THRESHOLD (parts per million)

COMPOUND	LEVEL	SOURCE
2,4-Dichlorophenoxyacetic acid	Negligable vapor pressure	
2,4,5-Trichlorophenoxyacetic acid	Negligable vapor pressure	
Chlorobenzene	60	AIHA
2-Dichlorobenzene	2-4 50 50	AIHA May Patty
4-Dichlorobenzene	15-30	Patty
1,2,4-Trichlorobenzene	3	ACGIH
2,4-Dichlorophenol	not available	
2,4,6-Trichlorophenol	not available	
Pentachlorophenol	not available	
Toluene	10-15 ppm (olfactory fatigue occurs rapidly)	ANSI Patty

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IRRITATION LEVEL (parts per million, unless otherwise indicated)

COMPOUND	EYE	NOSE	THROAT	ODOR	SOURCE
2,4-Dichlorophenoxy-acetic acid	Negligable vapor pressure, warning properties not pertinent			Odorless	NIOSH Occupational Health Guidelines Chris
2,4,5-Trichloro-phenoxyacetic acid	Not knownto be an eye irritant			Odorless	NIOSH Occupational Health Guidelines Chris
Chlorobenzene	eye irritant			Sweet, almond odor	
2-Dichlorobenzene	20-30			Pleasant odor	AIHA
4-Dichlorobenzene	80-160			Distinct Aromatic (mothballs)	Patty
1,2,4-Trichloro-benzene	3-5		3-5		ACGIH
2,4-Dichlorophenol				Medicinal odor	Chris
2,4,6-Trichlorophenol				Strong phenolic odor	SAX
Pentachlorophenol	>1mg/m^3	0.3mg/m^3			ACGIH
Toluene	300-400 100-500	100-500	100-500		Grant

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SECTION VI

TABLE III

Summary of Toxicity Data

SECTION VI

SUMMARY OF TOXICITY DATA

COMPOUND	TOXIC DOSE TDLo	TCLo	LDLo	LD_{50}	LCLo	LC_{50}	ROUTES OF EXPOSURE	SPECIES EXPOSED	DURATION OF EXPOSURE	TOXIC EFFECTS	REFERENCE
2,4-Dichlorophenoxyacetic acid			80 mg/kg				ORAL	HUMAN		NAUSEA OR VOMITING, SOMNOLENCE, COMA	ARPAAQ, 94,207,72
				370 mg/kg			ORAL	RAT			FMCHA2-D, 88,80
				1500mg/kg			SKIN	RAT			WRPCA 29,119,70
			666mg/kg				INTRAPERITONIAL	RAT			TIHTAB 29,85,47
				368mg/kg			ORAL	MOUSE		GASTRITIS, SOMNOLENCE, FATTY LIVER DEGENERATION	AJVRAJ 15,622,54
			125mg/kg				INTRAPERITONIAL	MOUSE			TXAPA 923,288,72
				100mg/kg			ORAL	DOG		STIFFNESS, COMA	AEHLAU 7,202,63
			800mg/kg				ORAL	RABBIT			AMFMAR 12,26,51
				1400mg/kg			SKIN	RABBIT		ATAXIA, PRIMARY IRRITATION	AFDOAO, 16,3,52
			400mg/kg				INTRAPERITONIAL	RABBIT			JIHTAB 29,85,47
			400mg/kg				INTRAVENOUS	RABBIT			JIHTAB 29,85,47
				469 mg/kg			ORAL	GUINEA PIG		GASTRITIS, SOMNOLENCE, FATTY LIVER DEGENERATION	AJVRAH 15,622,54
			666 mg/kg				INTRAPERITONIAL	GUINEA PIG			JIHTAB 29,85,47
				500 mg/kg			ORAL	HAMSTER			TXAPA9 48,A192,79
				541 mg/kg			ORAL	CHICKEN		GASTRITIS, SOMNOLENCE, FATTY LIVER DEGENERATION	AJVRAH 15,622,54
				375 mg/kg			ORAL	MAMMAL			SCIEAS 166,465,69

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SECTION VI

SUMMARY OF TOXICITY DATA

COMPOUND	TOXIC DOSE TDLo	TCLo	LDLo	LD_{50}	LCLo	LC_{50}	ROUTES OF EXPOSURE	SPECIES EXPOSED	DURATION OF EXPOSURE	TOXIC EFFECTS	REFERENCE
2,4,5-Trichloro-phenoxyacetic acid				300mg/kg			ORAL	RAT			BREVAH 10,97,65
				500 mg/kg			UNREPORTED	RAT			30ZDA9 -,17,71
				389 mg/kg			ORAL	MOUSE		GASTRITIS, SOMNO-LENCE, (GENERAL DEPRESSED ACTIVITY), FATTY LIVER, DE-GENERATION	AJVRAH 15,622,54
				100 mg/kg			ORAL	DOG			PAREAQ 14,225,62
				381 mg/kg			ORAL	GUINEA PIG			PCOC -1178,66
				425 mg/kg			ORAL	HAMSTER		NOT REPORTED	MUREAN 65,83,79
				310 mg/kg			ORAL	CHICKEN		GASTRITIS, SOMNO - LENCE (GENERAL DEPRESSED ACTIVITY), FATTY LIVER DE-GENERATION	AJVRAH 15,622,54
				500 mg/kg			ORAL	MAMMAL			SCIEAS 165,465,69
2-(2,4,5-Trichloro phenoxy) Proprionic acid				650 mg/kg			ORAL	RAT			BREVAH 10,97,65
				650 mg/kg			ORAL	MAMMAL			SCIEAS 165,465,69
				650 mg/kg			UNREPORTED	MAMMAL			30ZDA9 -173,71
2,3,7,8-Tetra-chloroditenzo-p-dioxin				22500 ng/kg			ORAL	RAT		UNREPORTED	EVSRBT 2,708,73
				114 ug/kg			ORAL	MOUSE			TXAPA9 29,229,74
			80 ug/kg				SKIN	MOUSE			RCOCB8 21,101,78
			200 ug/kg				UNREPORTED	MOUSE			ANYAA9 320,204,79

SECTION VI

SUMMARY OF TOXICITY DATA

COMPOUND	TOXIC DOSE TDLo	TCLo	LDLo	LD_{50}	LCLo	LC_{50}	ROUTES OF EXPOSURE	SPECIES EXPOSED	DURATION OF EXPOSURE	TOXIC EFFECTS	REFERENCE
			3 mg/kg				ORAL	DOG		WEIGHT LOSS OR DECREASED GAIN, SOMNOLENCE (GENERAL DEPRESSED ACTIVITY) ALCERATION OR BLEEDING FROM SMALL INTESTINE	ADCSAT 120,55,73
			70 ug/kg				ORAL	MONKEY			TXAPA9 43,175,78
			[illegible] ug/kg				ORAL	RABBIT			NATUAS 232,395,71
				275 ug/kg			SKIN	RABBIT			EVHPA2 5,97,73
				500 ng/kg			ORAL	GUINEA PIG		NOT REPORTED	EVSRBT 2,708,73
				5051 ug/kg			ORAL	HAMSTER		WEIGHT LOSS, OR DECREASED GAIN, EFFECTS TO HAIR NOT SPECIFIED	TXAPA9 59,405,81
			25 ug/kg				ORAL	CHICKEN		DYSPNEA, WEIGHT LOSS, OR DECREASED GAIN	FCTXAV 11,585,73
2 Chloro-phenol				670 mg/kg			ORAL	RAT			FEPRA7 2,76,43
				230 mg/kg			INTRAPERITONEAL	RAT			BJPCAL 13,20,58
				950 mg/kg			SUBCUTANEOUS	RAT			FEPRA7 2,76,43
				670 mg/kg			ORAL	MOUSE			TIVSAI 33,258,69
			950 mg/kg				SUBCUTANEOUS	RABBIT			HBAMAK 4,1361,35
			120 mg/kg				INTRAVENOUS	RABBIT			HBIXAC 5,112,59
			800 mg/kg				SUBCUTANEOUS	GUINEA PIG			HBAMAK 4,1361,35

SUMMARY OF TOXICITY DATA

COMPOUND	TOXIC DOSE TDLo	TCLo	LDLo	LD_{50}	LCLo	LC_{50}	ROUTES OF EXPOSURE	SPECIES EXPOSED	DURATION OF EXPOSURE	TOXIC EFFECTS	REFERENCE
				2830 mg/kg			ORAL	RABBIT			14CYAT 2,1394,63
			4100 mg/kg				INTRAPERITONEAL	GUINEA PIG			RMSRAG 16,449,1896
1,2 dichloro-benzene				500 mg/kg			ORAL	RAT			WRPCA2 7,135,68
					621 ppm		INHALATION	RAT	7 hours		AMIHAB 17,180,58
				340 mg/kg			INTRAPERITONEAL	RAT			MEPAAX 20,519,69
			400 mg/kg				INTRAVENOUS	MOUSE			JPBAA7 44,281,37
				500 mg/kg			ORAL	RABBIT			85ARAE 3,32,76
			250 mg/kg				INTRAVENOUS	RABBIT			JPBAA7 44,281,37
			2000 mg/kg				ORAL	GUINEA PIG			14CYAT 2,1336,63
					300 ppm		INHALATION	GUINEA PIG	24 hours		
1,4 dichloro-benzene	300 mg/kg						ORAL	HUMAN		HYPERMOTILITY, DIAHRREA, EFFECTS TO EYE AND RESPIRATORY SYSTEM	PCOC -851,66
			221 mg/kg				UNREPORTED	MAN			85DCA1 2,73,70
				500 mg/kg			ORAL	RAT			WRPCA2 9,119,70
				2562 mg/kg			INTRAPERITONEAL	RAT			JAPMA8 38,124,49
				2950 mg/kg			ORAL	MOUSE			GUCHAZ 6,183,73
				5145 mg/kg			SUBCUTANEOUS	MOUSE			TOIZAG 20(516),772, 73

SECTION VI

SUMMARY OF TOXICITY DATA

COMPOUND	TOXIC DOSE TDLo	TCLo	LDLo	LD_{50}	LC_{Lo}	LC_{50}	ROUTES OF EXPOSURE	SPECIES EXPOSED	DURATION OF EXPOSURE	TOXIC EFFECTS	REFERENCE
			400 mg/kg				SUBCUTANEOUS	FROG			HBTXAC 5,112,59
				440 mg/kg			ORAL	MAMMAL			TIVSAI 33,358,69
4 chloro-phenol				261 mg/kg			ORAL	RAT			28ZPAK -,78,72
				281 mg/kg			INTRAPERITONEAL	RAT			BJPCAL 13,20,58
				1030 mg/kg			SUBCUTANEOUS	RAT			FEPRA7 2,76,43
4-chloro-3-methyl-phenol			500 mg/kg				ORAL	RAT			JPETAB 90,260,47
				400 mg/kg			SUBCUTANEOUS	RAT			QJPPAL 12,212,39
			30 mg/kg				INTRAPERITONEAL	MOUSE			QJPPAL 12,212,39
			200 mg/kg				SUBCUTANEOUS	MOUSE			QJPPAL 12,212,39
2,4-dichloro-phenol				580 mg/kg			ORAL	RAT			FEPRA7 2,76,43
				430 mg/kg			INTRAPERITONEAL	RAT			BJPCAL 13,20,58
				1730 mg/kg			SUBCUTANEOUS	RAT			FEPRA7 2,76,43
				1600 mg/kg			ORAL	MOUSE			TO1ZAG 19,356,72
2,4,6-trichloro-phenol				820 mg/kg			ORAL	RAT			PCOC- 1176,66
				276 mg/kg			INTRAPERITONEAL	RAT			BJPCAL 13,20,58

SECTION VI

SUMMARY OF TOXICITY DATA

COMPOUND	TOXIC DOSE TDL$_o$	TCL$_o$	LDLo	LD$_{50}$	LCL$_o$	LC$_{50}$	ROUTES OF EXPOSURE	SPECIES EXPOSED	DURATION OF EXPOSURE	TOXIC EFFECTS	REFERENCE
pentachloro-phenol			29 mg/kg				ORAL	HUMAN			272XA3 -256,63
	196 mg/kg						ORAL	MAN		CHANGE IN MOTOR ACTIVITY, SWEATING, INCREASE IN BODY TEMP.	3F17R 20025
				50 mg/kg			ORAL	RAT			FMCHA2 D233,80
				105 mg/kg			SKIN	RAT			BJIMAG 26,59,59
				56 mg/kg			INTRAPERITONEAL	RAT			BJPCAL 13,20,58
				100 mg/kg			SUBCUTANEOUS	RAT			FEPRA7 2,76,43
			135 mg/kg				SUBCUTANEOUS	DOG			HBTXAC 5,123,59
			70 mg/kg				ORAL	RABBIT			JPETAB 76,104,42
			40 mg/kg				SKIN	RABBIT			JPETAB 76,104,42
			135 mg/kg				INTRAPERITONEAL	RABBIT			HBTXAC 5,123,59
			70 mg/kg				SUBCUTANEOUS	RABBIT			JPETAB 76,104,42
				168 mg/kg			ORAL	HAMSTER			TXAPA9 48,A192,79
chlorobenzene				2910 mg/kg			ORAL	RAT			14CYAT 2,1334,63
			7400 mg/kg				INTRAPERITONEAL	RAT			RMSRAG 16,449,1896
			7000 mg/kg				SUBCUTANEOUS	RAT			RMSRAG 16,449,1896
					15 gm/m3		INHALATION	MOUSE			GISAAA 20(8),19,55

SUMMARY OF TOXICITY DATA

COMPOUND	TOXIC DOSE TDL o	TCL o	LDL o	LD_{50}	LCL o	LC_{50}	ROUTES OF EXPOSURE	SPECIES EXPOSED	DURATION OF EXPOSURE	TOXIC EFFECTS	REFERENCE
			2800 mg/kg				ORAL	GUINEA PIG			14CYAT2,1338,63
,2,4-trichloro-benzene				756 mg/kg			ORAL	RAT			AOHYA3 12,209,69
				300 mg/kg			ORAL	MOUSE		BEHAVIORAL CHANGES: ATAXIA;CONVULSIONS: OR EFFECT ON SEIZURE THRESHOLD: ALTERED SLEEP TIME (INCL. CHANGE IN RIGHTING REFLEX)	NAIZAM 20,569,78
			500 mg/kg				INTRAPERITONEAL	MOUSE			CBCCT 4,107,52
nexachloro-benzene			220 mg/kg				UNREPORTED	MAN			85DCA1 2,73,70
				10,000 mg/kg			ORAL	RAT			85DPAn -,71/76
toluene		200 ppm					INHALATION	HUMAN		RECORDINGS FROM SPECIFIC AREAS OF CNS: ANTIPSYCHOTIC; CHANGES IN BLOOD - DECREASE IN RED CELL COUNT, APLASTIC ANEMIA (CHANGES MAY HAVE BEEN DUE TO BENZENE CONTAMINANT IN TOLUENE)	JAMAAP 123,1106,43
		[illegible] ppm					INHALATION	MAN		HALLUCINATIONS, DISTORTED PERCEPTIONS: CHANGE IN MOTOR ACTIVITY: CHANGE IN PHYSIOLOGICAL TESTS	WEHSAL 9,131,72
				[illegible]000 mg/kg			ORAL	RAT		NOT REPORTED	AMIHAB 19,463,59
					4000 ppm		INHALATION	RAT	4 hours		AIHAAP 30,470,69
			800 mg/kg				INTRAPERITONEAL	RAT			7XAPA9 1.156.59
				[illegible]000 mg/kg			UNREPORTED	RAT		NOT REPORTED	GISAAA 45[12],54,80
						[illegible]20 ppm	INHALATION	MOUSE	[illegible] HRS		JIHTAB 25,300,43

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SUMMARY OF TOXICITY DATA

COMPOUND	TOXIC DOSE TDLo	TCLo	LDLo	LD_{50}	LCLo	LC_{50}	ROUTES OF EXPOSURE	SPECIES EXPOSED	DURATION OF EXPOSURE	TOXIC EFFECTS	REFERENCE
				1120 ug/kg			INTRAPERITONEAL	MOUSE			AGGHAR 18,109,60
				2000 mg/kg			UNREPORTED	MOUSE		NOT REPORTED	GISAA 45(12)64,80
				14 gm/kg			SKIN	RABBIT			UCDS 7/23/70
			920 mg/kg				SUBCUTANEOUS	FROG			AEPPAE 130,250,28

SOURCE: "REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES;" VOLUME I, II, AND III, 1981-82 EDITION. NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH, U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES, DHHS (NIOSH) PULBICATION NO. 83-107, JUNE 1983

REFERENCES: SEE " REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES," PP 1045-1087, FOR REFERENCE CODES.

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APPENDIX A

ANALYTICAL METHOD

The analytical procedure ultimately developed and described herein for determination of TCDD's in various industrial process waste samples utilizes two separate GC-MS systems. A gas chromatograph coupled to a low-resolution quadrupole mass spectrometer (GC-QMS) is used for preliminary identification of TCDD's in the extracts of the waste samples. A second apparatus coupling a gas chromatograph and a high-resolution mass spectrometer (GC-MS-30) is used to confirm the results obtained with the GC-QMS technique. The analysis method entails two steps, sample preparation and instrumental analysis, as described below. It should be emphasized that, even with the elaborate separation techniques employed here, the 2,3,7,8-TCDD isomer is still not resolved from the other TCDD isomers if these are present in sample extracts. As a result, the quantitative data obtained here for TCDD's must be considered an upper limit rather than an absolute level for any individual TCDD isomer.

SAMPLE PREPARATION

The following procedures were developed as an approach to preparation of industrial waste samples and have been successfully applied in this study.

1. Place a 2.0 g aliquot of the sample in each of the two extraction vessels. To each aliquot, add an appropriate quantity of ^{37} Cl4-2,3,7,8-TCDD dissolved in "distilled-in-glass" benzene as an internal standard. Spike one of the two aliquots with an additional known quantity of authentic native 2,3,7,8-TCDD at a concentration equal to the nominal amount expected in the sample.

2. Add 30 ml "distilled-in-glass" petroleum ether to each sample and mix thoroughly.

3. Extract each organic solution with 50 ml of double-distilled water and discard the aqueous layer.

4. Extract each solution with 50 ml of 20 percent potassium hydroxide and discard the aqueous basic layer.

5. Extract each solution with 50 ml of double-distilled water and discard the aqueous portion.

6. Extract each solution with 50 ml of concentrated sulfuric acid and discard the aqueous acidic layer.

7. Repeat step 6 until the acid layer is nearly colorless.

8. Extract each organic solution with 50 ml of double-

distilled water and discard the aqueous layer.

9. Dry each organic solution over anhydrous sodium sulfate.

10. Quantitatively transfer each organic solution to another vessel, and concentrate to a volume of approximately 1 ml by passing a stream of purified nitorgen over the surface of the liquid while applying gentle heat (50 C) to the vessel.

11. Construct a chromatography column for each sample by packing a disposable glass pipette (I.D.=0.8 cm) with glass wool and 2.8 g of Woelm basic alumina (previously activated by maintaining it at 600 C for a minimum of 24 hours, then cooled in a dessicator for 0.5 hour prior to use).

12. Quantitatively transfer each concentrated organic solution to the top of a column.

13. Elute each column with 10 ml of 3 percent "distilled-in-glass" methylene chloride in "distilled-in-glass" hexane, and discard the entire column effluent.

14. Elute each column with 20 ml of 20 percent methylene chloride in hexane and collect the eluate in four 5-ml fractions.

15. Elute each column with 10 ml of 50 percent methylene chloride in hexane and retain the entire column eluate for analysis.

16. Elute each column with 3 ml of 50 percent methylene chloride in hexane and retain the eluate for analysis.

17. Concentrate all six fractions in benzene to an appropriate volume (usually 0.1 to 1.0 ml) and proceed with analysis.

INSTRUMENTAL ANALYSIS

The application of GC-MS instrumentation methods for analysis of TCDD's requires knowledgeable and experienced personnel, dedication of the equipment, and significant capital and operating costs. The requirement for detecting low ppt levels of TCDD's in these analyses necessitates such a sensitive and selective analytical method. Because this is currently the only known method which meets these criteria, the relatively high expense is unavoidable.

The following is a brief description of the instrumentation required for the analytical procedures developed herein.

GC-QMS System

The GC-QMS system consists of a Varian Model 2740 Gas Chromatograph coupled directly (no helium separator is required) to an Extra-nuclear Quadrupole Mass Spectrometer. The GC was adapted to include a sophisticated system of remotely actuated high-temperature switching valves (Valco Co.) and Granville-Phillips molecular leak valves, so that the column effluent could be readily regulated (Tiernan et al. 1975a; Erk, Taylor, and Tiernan 1978).

With this arrangement, the total column effluent can be directed into the mass spectrometer ion source, or the effluent flow can be split, one portion going to the ion source and the other to a gas chromatographic detector, as desired. The use of a differential high-speed pumping system on the source vacuum envelope permits introduction of as much as 65 ml/min of effluent from the gas chromatograph into the mass spectrometer ion source. Admitting the total chromatograph effluent into the mass spectrometer source enhances the sensitivity of the analysis.

For the purpose of instrument control and data acquisition, the GC-QMS system is coupled to an Autolab System IV Computing Integrator. Additional capacity for off-line data reduction is available with a Hewlett-Packard 2116C Minicomputer, which is programmed to accept data (punched paper tape) from the system when necessary.

GC-MS-30 System

The GC-MS-30 system used in these studies consists of a Varian 3740 Gas Chromatograph coupled through an AEI silicone membrane separator to an AEI MS-30 Double-Focusing, Double-Beam Mass Spectrometer. The mass spectrometer is equipped with a unique electrostatic analyzer scan circuit developed by Wright State University, which permits the monitoring of as many as four mass peaks, essentially simultaneously, by rapidly and sequentially stepping and switching between the masses of interest, while maintaining picogram sensitivity for TCDD's. The data are recorded by use of a Nicolet 1074 Signal Averaging Computer.

Sample Analysis

Analysis consists of three steps as described below:

1. Analyze each eluate fraction (collected in the elution chromatography separation of the sample) on the low-resolution GC-QMS, using the following parameters:

   Varian 2740 Gas Chromatograph

   Column: 2 m x 3 mm I.D. glass packed with 3% OV-7 on Gas Chrom Q

Carrier gas: Helium at 65 ml/min (the total chromatographic column effluent is admitted to the mass spectrometer ion source)

Temperatures: Injector: 255 C
Column: 275 C
Transfer line: 295 C

Quadrupole mass spectrometer

Ionizing voltage:	23.5 eV	
Multiplier:	3200 V	
Resolution:	1:350	
Source envelope pressure:	1.4×10^{-4}	torr
Analyzer envelope pressure:	8.0×10^{-6}	torr
Masses monitored:	m/e 320,322	
Source temperature:	250 C	
Analyzer temperature:	120 C	

2. Confirm any samples showing positive levels of TCDD's on the low-resolution GC-QMS by analysis of the corresponding eluate fractions using high-resolution GC-MS-30 and the following operating parameters:

Varian 3740 Gas Chromatograph

Column: 1.8 x 2 mm I.D. coiled glass column packed with 3% Dexsil 300 on Supelcoport (100/120 mesh)

Carrier gas: Helium at a flow rate of 30 ml/min

Temperatures: Injector: 250 C
Column: 240 C
Transfer line: 285 C

AEI MS-30 mass spectrometer

Resolution:	1:12,500
Ionizing voltage:	70 eV
Masses monitored:	m/e 319.8966, 321.8936, 325.8805, and 327.8846
Temperatures:	Membrane separator: 215 C Transfer line: 270 C Source: 250 C

3. Determine the overall recovery of the analytical procedure by measuring the amount of internal standard ($^{37}Cl_4$-2,3,7,8-TCDD) recovered.