UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 2
290 BROADWAY
NEW YORK, NY 10007-1866

JUL 27 1999

Return Receipt Requested

Mr. William Watson
Hicksville Library
169 Jerusalem Avenue
Hicksville, New York 11801

Re: Additional Documents for Inclusion in the Anchor Chemical Superfund Site File

Dear Mr. Watson:

In 1995, the U.S. Environmental Protection Agency (EPA) sent to the Hicksville Public Library a number of documents relating to the investigation and clean up of the Anchor Chemicals Superfund Site, located at 500 West John Street in the Town of Oyster Bay. The Library, at the EPA's request, established a file for the Site so that the public could review the documents.

Since 1995, all federal environmental response actions and investigations have been completed at the Site. Therefore, EPA has decided to remove the Site from the Superfund National Priorities List (NPL). Enclosed for inclusion in the Library's Anchor Chemicals Site file are six additional documents:

- Record of Decision for the Anchor Chemical Superfund Site - September 29, 1995
- Removal Action Final Report - May 1996
- Superfund Support Inspection Report - April 4, 1996
- Superfund Support Inspection Report - July 21-23,1997
- Superfund Preliminary Site Closeout Report - September 30,1996
- Letter from Michael O'Toole of the New York State Department of Environmental Conservation to Richard L. Caspe of the US Environmental Protection Agency - dated July 7, 1999 ; Re: Proposed Deletion of Anchor Chemical from NPL

EPA will notify the public of its intent to delete the Site from the NPL within the next month. The notice will be published on the same day in the Hicksville Illustrated News and the Federal Register and will provide for a thirty day public comment period. I will send to you a copy of the notice once it is published. During the comment period, you may receive some requests to review the Library's file on the Site.

ANSON
ENVIRONMENTAL LTD.

33 Gerard Street • Suite 100
Huntington, New York 11743
516•351•3555
Fax 516•351•3615

VOLUME 4

REMEDIAL INVESTIGATION REPORT

Appendix F - M

Anchor Chemical Site
Hicksville, New York

March 1995

Prepared for:

K.B. Co.
375 North Broadway
Jericho, New York 11753

Prepared by:

Anson Environmental Ltd.
33 Gerard Street
Suite 100
Huntington, New York 11743

302171

APPENDIX F

Tank Closure Report

TANK CLOSURE REPORT

Anchor Chemical Site
500 W. John Street
Hicksville, New York

August 23, 1991

Prepared for:

Spiegel Associates
375 North Broadway
Jericho, New York 11753

Prepared by:

ROUX ASSOCIATES, INC.
775 Park Avenue
Huntington, New York 11743

ROUX

The underground tank inspection (Task 2) at the Anchor Chemical Site was performed in accordance with the Anchor Chemical Remedial Investigation Work Plan. This task consisted of inspecting and sampling (if applicable) 12 underground tanks of unknown status located beneath the concrete floor of the 500 West John Street building.

The underground tank inspection and closure was conducted from June 8 through June 14, 1991 by Enro-Serve, a subsidiary of Stout Environmental. Enro-Serve was contracted to Spiegel Associates to perform the underground tank inspection in accordance with the April 10, 1991 RI Work Plan and Project Operations Plan for the Site. Roux Associates, Inc., observed the underground tank inspection as consultant to Spiegel Associates, while Alliance Technology observed the operation as consultant to the United States Environmental Protection Agency (USEPA).

Prior to initiation of the work, Spiegel Associates hired a construction contractor to install plastic dust barriers within the building to contain any dust or debris created by the work. The building, which is currently occupied by a furniture company, was evacuated for the duration of the underground tank inspection as specified in the Site Health and Safety Plan.

On June 8, 1991 Enro-Serve cut through the building's concrete floor (approximately 4-inches thick) to expose the manways of eight of the underground tanks. All concrete debris was removed from the building and carted away the same day. The following week (June 10-14, 1991), Enro-Serve exposed the remaining four tanks to be inspected and opened the 12 tanks. The Enro-Serve worker who open the tanks wore a full-face respirator on an air line connected to bottled air, and took explosimeter and percent oxygen measurements within each tank immediately after opening the manways.

In the north room (the Combustible Mixing Room [CMR]), Enro-Serve exposed and opened the manways of Tanks 1 through 4. Tanks 1, 2, and 3 were found completely filled with concrete, while Tank 4 was found half filled with concrete (Figure 1). Tank 1 was slightly overfilled with concrete and the manway cover was bulging. All soil that had been removed from above the tanks was piled along the north wall inside the CMR. The soil appeared clean, and all Hnu readings (taken by Alliance) were reportedly zero.

In the south room (the Flammable Mixing Room [FMR]), the concrete slab was removed in about six locations, and the seven tanks to be investigated (Tanks 7, 9, 10, 12, 13, 14, and 17) had been located. Each tank had a manway with the exception of Tank 17. Enro-Serve opened the manways of these tanks and used a Sawsall saw to cut open Tank 17. Four of these tanks (Tanks 9, 12, 13, and 14) were found filled with concrete, and Tanks 7, 10, and 17 were empty and, based on our visual inspection and Hnu readings, appeared clean (Figure 1).

In the warehouse, Tank 16 was located and a manway exposed (Figure 1). When the manway was opened, Enro-Serve's initial explosimeter reading was within the explosive range, but this quickly dropped to background levels. The tank contained about 550 gallons of water. OVM and Hnu readings were zero above and inside the tank prior to sampling the water. Enro-Serve bailed water from the tank using a "sludge judge" (similar to a bailer) and collected samples. The samples collected by Enro-Serve were turned over to Roux Associates, Inc. to label, complete chain-of-custody documentation, pack on ice, and ship to CEIMIC Corporation's laboratory for TCLP analysis. Alliance collected a split sample to be analyzed by the USEPA's laboratory.

After sampling the water, Enro-Serve pumped the water from Tank 16 into eleven 55-gallon drums. All 55-gallon drums were sealed and placed just outside the building on pallets. Each drum lid was labeled as "Hazardous Waste Liquid". The 55-gallon drums were later moved to the northwest corner of the Site and properly stored on pallets on pavement and within a bermed area constructed of sand.

The four empty tanks (Tanks 7, 10, 16, and 17) were cleaned on July 12, 1991. Enro-Serve lowered a man into each tank to clean it. The worker within the confined space cleaning the inside of the tanks was in full Level B attire, in a full-face respirator on an airline (supplied bottled air), and attached to safety ropes. One Enro-Serve worker was outside the tank holding the safety ropes, and another was lowering equipment to the man in the tank. If liquid remained in the bottom of the tank, it was removed with a diaphragm pump. Each of the four tanks was scraped, and the scrapings removed either with a pump or manually (bucket). Oil-Dri absorbent was lowered into the tanks that had moisture in them, and spread around with a shovel. All the used Oil-Dri was removed from the tanks and drummed.

After scrapping Tank 10, the total vapor readings measured on the OVM and Hnu were approximately 234 ppm and 55 ppm, respectively. CO_2 gas (fire extinguisher), added to the tank to decrease the vapor concentration, brought the OVM reading down to 33 ppm. Enro-Serve then tried to remove all remaining vapors by pumping air into Tank 10 while all workers left the building. The OVM reading then increased to 201 ppm. This prompted Enro-Serve to power-wash Tank 10 with cold water to remove vapors trapped in the tank walls. Rinse water was vacuumed from the tank, Oil-Dri was used to absorb the remaining moisture, and the used Oil-Dri removed from the tank and drummed.

According to the federal underground storage tank (UST) regulations, a tank should be clean and "vapor-free" prior to abandonment. To accomplish this, Enro-Serve used dry ice and CO_2 gas (fire extinguisher) to evacuate the vapors which remained in Tanks 4, 7, 10, and 17. The final OVM and Hnu readings for each empty (and partially empty) tank are given in Table 1.

With the USEPA's approval, Enro-Serve and a concrete contractor filled Tanks 7, 10, 16, and 17, and the remainder of Tank 4 with concrete. The results of the tank inspections are summarized in Table 2.

After the tanks had been filled with concrete, Malcolm Barkan of MIB Consulting (affiliated with Enro-Serve) marked out the locations of six soil borings to be drilled within the building, and prepared a map of these locations.

The sample collected from Tank 16, along with additional sample volume collected from the drums stored at the Site, was shipped to CEIMIC Corp. (laboratory) by Roux Associates. The sample was analyzed for the toxicity characteristic leaching procedure (TCLP) parameters, plus ignitability, reactivity, and corrosivity according to USEPA Methods. Two metals were detected in the sample as follows: arsenic (0.3 ppm), and barium (0.02 ppm). All other analytes were below the applicable detection limit. CEIMIC Corporation's analytical report is given in Appendix A.

Respectfully submitted,

ROUX ASSOCIATES, INC.

Joanne Yeary
PR

Joanne Yeary
Senior Hydrogeologist

Paul Roux
President

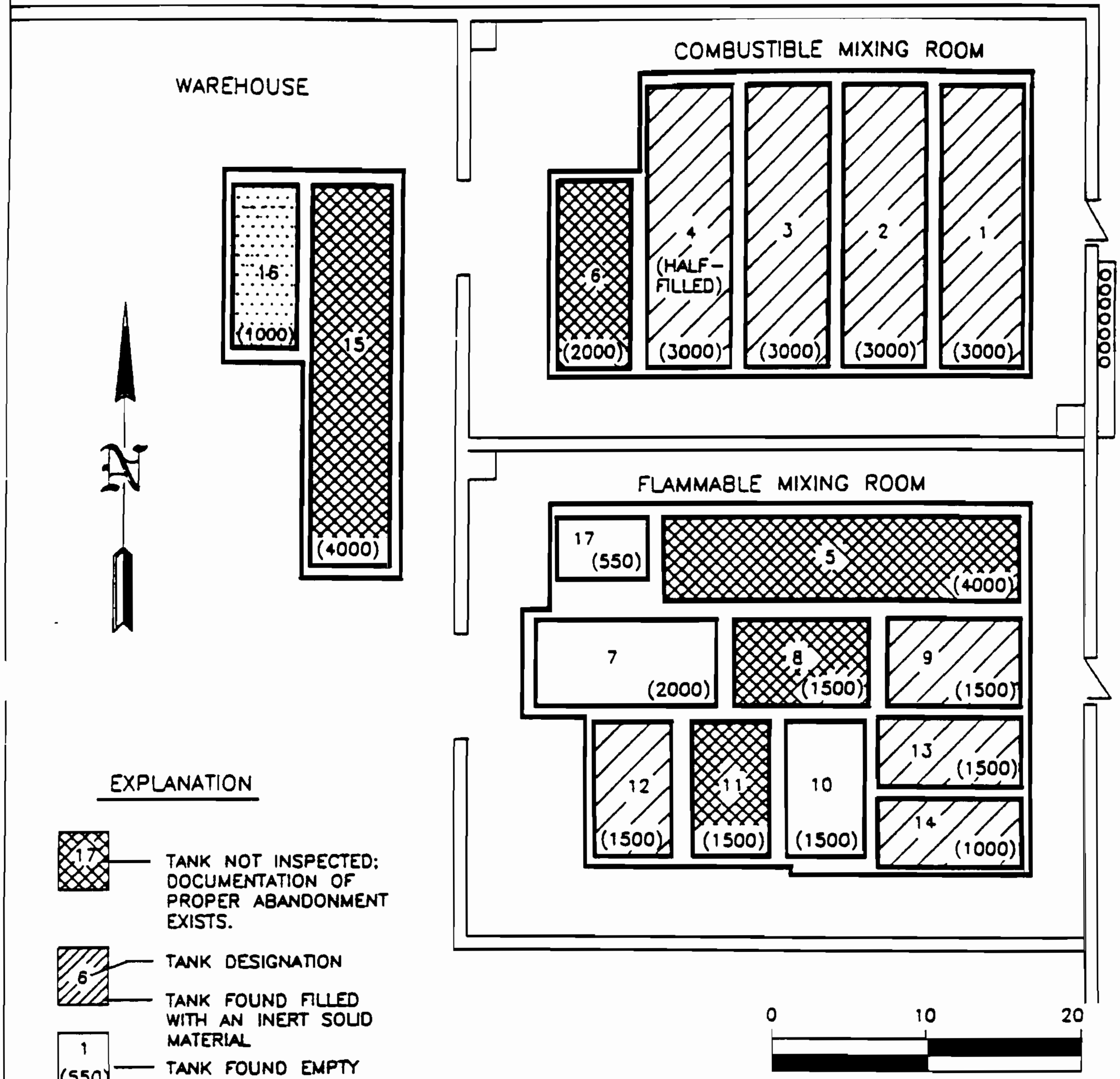
Table 1. Final Photoionization Meter Readings Taken Within the Underground Tanks on June 12, 1991, Anchor Chemical Site, Hicksville, New York.

Tank Designation	OVM Reading (ppm)	Hnu Reading (ppm)
Tank 4	0 to 1.3	6 to 7
Tank 7	0	19
Tank 10	9	5
Tank 16	0	0
Tank 17	4	3.8

Table 2. Summary of Underground Tank Inspections Performed from June 8 Through June 12, 1991, Anchor Chemical Site, Hicksville, New York.

Tank Designation	Tank Contents	Work Performed by Enro-Serve
Tank 1	Concrete	None
Tank 2	Concrete	None
Tank 3	Concrete	None
Tank 4	1/2 Concrete 1/2 Empty	Vapors removed, tank filled with concrete
Tank 7	Empty	Tank scrapped, dried, vapors removed, tank filled with concrete
Tank 9	Concrete	None
Tank 10	Empty	Tank scrapped, power-washed, dried, vapors removed and tank filled with concrete
Tank 12	Concrete	None
Tank 13	Concrete	None
Tank 14	Concrete	None
Tank 16	550 gallons of water	Water sampled and removed, tank scrapped, dried and filled with concrete
Tank 17	Empty	Tank scrapped, dried, vapors removed and tank filled with concrete

FIGURES



EXPLANATION

▩ 17 — TANK NOT INSPECTED; DOCUMENTATION OF PROPER ABANDONMENT EXISTS.

▨ 6 — TANK DESIGNATION

— TANK FOUND FILLED WITH AN INERT SOLID MATERIAL

☐ 1 (550) — TANK FOUND EMPTY

— TANK CAPACITY, IN GALLONS

⛆ 16 — TANK FOUND TO CONTAIN WATER; WATER SAMPLED

NOTE: STATUS OF TANKS DURING TASK 2 OF REMEDIAL INVESTIGATION, JUNE 8–14, 1991

Title:

STATUS OF UNDERGROUND STORAGE TANKS AT THE ANCHOR CHEMICAL SITE

HICKSVILLE, NEW YORK

Prepared for:

SPIEGEL ASSOCIATES

ROUX
ROUX ASSOCIATES INC
Consulting Ground-Water Geologists & Engineers

Compiled by: J.Y.	Date: 7/91	FIGURE
Prepared by: S.W.	Scale: SHOWN	1
Project Mgr: J.Y.	Revision:	
File No: SP603A02		

APPENDICES

APPENDIX A

CEIMIC
CORPORATION

"Analytical Chemistry for Environmental Management"

July 17, 1991

Mr. Fred Werfel
Spiegel Associates
375 North Broadway
Jericho, NY 11753

Dear Mr. Werfel:

Enclosed is the data report of results for the analyses of samples which were received at CEIMIC Corporation on June 12, 1991.

Due to difficulty with the herbicide analyses, the samples had to be reextracted out of holding time.

Please call if you have any questions.

Sincerely,

Kin S. Chiu
Organic Laboratory
Manager

KSC/11

enc.

10 Dean Knauss Drive, Narragansett, R.I. 02882 • (401) 782-8900 • FAX (401) 782-8905

VOLATILE ORGANIC ANALYSES

SURROGATE RECOVERY SUMMARY

Volatile Organics Analysis

Client: Roux Associates

Date Samples Received: 6/12/91

Project No.: 910312

Matrix: Leachate

Surrogate Compound	TCLP Extraction Blank	Samples -01	QC Limits
1,2-Dichloroethane-d4	97 %	96 %	76 - 114
Toluene-d8	99	102	88 - 110
Bromofluorobenzene	97	104	86 - 115

Reported by: KC

302187

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTICS LEACHING PROCEDURE (TCLP)

VOLATILE ORGANICS TARGET ANALYTES

Client: Roux Associates

Client Sample ID: TCLP Extraction Blank Date Sampled: NA

Laboratory ID: VTCLP0618-B1 Date TCLP performed: 6/18/91

Concentration in: ug/L (ppb) Date Leachate Analyzed: 6/24/91

	Actual		Adjusted*	
Target Analyte	Sample Result	Method Reporting Limit	Sample Result	Method Reporting Limit
Benzene	ND	5	--	--
Carbon tetrachloride	ND	5	--	--
Chlorobenzene	ND	5	--	--
Chloroform	ND	5	--	--
1,2-Dichloroethane	ND	5	--	--
1,1-Dichloroethylene	ND	5	--	--
Methylethylketone	ND	10	--	--
Tetrachloroethylene	ND	5	--	--
Trichloroethylene	ND	5	--	--
Vinyl chloride	ND	10	--	--
Pyridine	ND	1,000	--	--

* Actual sample result adjusted for matrix bias. Refer to matrix spike analysis summary form.

NA = Not applicable
ND = Not detected

Reported by: AJK

Approved by: KC

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTICS LEACHING PROCEDURE (TCLP)

VOLATILE ORGANICS TARGET ANALYTES

Client: Roux Associates

Client Sample ID: Tank 16

Laboratory ID: 910312-01

Concentration in: ug/L (ppb)

Date Sampled: 6/11/91

Date TCLP performed: 6/18/91

Date Leachate Analyzed: 6/20/9

	Actual		Adjusted*	
Target Analyte	Sample Result	Method Reporting Limit	Sample Result	Method Reporting Limit
Benzene	ND	5	ND	7
Carbon tetrachloride	ND	5	ND	6
Chlorobenzene	ND	5	ND	7
Chloroform	ND	5	ND	6
1,2-Dichloroethane	ND	5	ND	6
1,1-Dichloroethylene	ND	5	ND	5
Methylethylketone	ND	10	ND	12
Tetrachloroethylene	ND	5	ND	6
Trichloroethylene	ND	5	ND	6
Vinyl chloride	ND	10	ND	11
Pyridine	ND	1,000	ND	2,300

* Actual sample result adjusted for matrix bias. Refer to matrix spike analysis summary form.

Library Search:

Compound	Retention Time	Estimated Concentration
Unknown	3.92 min.	8
Acetone	4.47	310
Methylene Choride	5.23	200

ND = Not detected

Reported by: AJL

Approved by: KC

CEIMIC
CORPORATION

"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTICS LEACHING PROCEDURE (TCLP)

VOLATILE ORGANICS TARGET ANALYTES

MATRIX SPIKE ANALYSIS SUMMARY

EPA METHOD 8240

Client: Roux Associates

Client Sample ID: Tank 16

Laboratory ID: 910312-01

Date Analyzed: 6/26/91

Concentration in: ug/L (ppb)

Target Analyte	Sample Result	Spike Added	Spiked Sample Result	Percent Recovery
Benzene	ND	50	37	75%
Carbon tetrachloride	ND	50	44	88
Chlorobenzene	ND	50	37	74
Chloroform	ND	50	43	86
1,2-Dichloroethane	ND	50	40	79
1,1-Dichloroethylene	ND	50	49	98
Methylethylketone	ND	50	41	82
Tetrachloroethylene	ND	50	40	80
Trichloroethylene	ND	50	44	88
Vinyl chloride	ND	50	47	94
Pyridine	ND	2000	870	44

This matrix spike analysis summary applies to the following samples:
Tank 16

ND = Not detected

Reported by: AJK

Approved by: KC

TCLP SEMIVOLATILE ORGANIC ANALYSES

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

SURROGATE RECOVERY SUMMARY

Semivolatile Organics

Client: Roux Associates

Project No.: 910312

Date Samples Received: 6/20/91

Matrix: TCLP Leachate

		Samples		
Surrogate Compound	TCLP Extraction Blank	-01	-01MS	QC Limits
2-Fluorophenol	69 %	71 %	70 %	21-100
Phenol-d5	54	54	51	10 -94
2,4,6-Tribromophenol	72	65	72	10-123
Nitrobenzene-d5	82	87	87	35-114
2-Fluorobiphenyl	79	90	84	43-116
Terphenyl-d14	105	109	93	33-141

Reported by: GMD.

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTICS LEACHING PROCEDURE (TCLP)
SEMIVOLATILE ORGANIC ANALYSIS
EPA METHOD 8270

Client: Roux Associates
Date Sampled: NA
Client Sample ID: TCLP Extraction Blank
Date TCLP Performed: 6/17/91
Laboratory ID: STCLP0617-B1
Date Leachate Prepared: 6/18/9
Concentration in: ug/L (ppb)
Date Extract Analyzed: 6/20/91

	Actual		Adjusted*	
Target Analyte	Sample Result	Method Reporting Limit	Sample Result	Method Reporting Limit
2,4-Dinitrotoluene	ND	33	--	--
Hexachlorobenzene	ND	33	--	--
Hexachloro-1,3-butadiene	ND	33	--	--
Hexachloroethane	ND	33	--	--
Nitrobenzene	ND	33	--	--
1,4-Dichlorobenzene	ND	33	--	--
Methylphenols (total)	ND	33	--	--
Pentachlorophenol	ND	160	--	--
2,4,5-Trichlorophenol	ND	160	--	--
2,4,6-Trichlorophenol	ND	33	--	--

ND = Not detected

* Actual sample result adjusted for matrix bias. Refer to matrix spike analysis summary form.

Reported by: ____________ Approved by: KC

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTICS LEACHING PROCEDURE (TCLP)

SEMIVOLATILE ORGANIC ANALYSIS

EPA METHOD 8270

Client: Roux Associates	Date Sampled: 6/11/91
Client Sample ID: Tank 16	Date TCLP Performed: 6/17/91
Laboratory ID: 910312-01	Date Leachate Prepared: 6/18/9
Concentration in: ug/L (ppb)	Date Extract Analyzed: 6/20/91

	Actual		Adjusted*	
Target Analyte	Sample Result	Method Reporting Limit	Sample Result	Method Reporting Limit
2,4-Dinitrotoluene	ND	33	ND	52
Hexachlorobenzene	ND	33	ND	46
Hexachloro-1,3-butadiene	ND	33	ND	46
Hexachloroethane	ND	33	ND	59
Nitrobenzene	ND	33	ND	38
1,4-Dichlorobenzene	ND	33	ND	52
Methylphenols (total)	ND	33	ND	52
Pentachlorophenol	ND	160	ND	260
2,4,5-Trichlorophenol	ND	160	ND	270
2,4,6-Trichlorophenol	ND	33	ND	53

ND = Not detected

* Actual sample result adjusted for matrix bias. Refer to matrix spike analysis summary form.

Reported by: ______________ Approved by: KC

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTICS LEACHING PROCEDURE (TCLP)

SEMIVOLATILE ORGANIC ANALYSIS

MATRIX SPIKE ANALYSIS SUMMARY

EPA METHOD 8270

Client: Roux Associates

Client Sample ID: Tank 16MS

Laboratory ID: 910312-01MS

Date Analyzed: 6/20/91

Concentration in: ug/L (ppb)

Target Analyte	Sample Result	Spike Added	Spiked Sample Result	Percent Recovery
2,4-Dinitrotoluene	ND	125	79	63 %
Hexachlorobenzene	ND	125	89	71
Hexachloro-1,3-butadiene	ND	125	90	72
Hexachloroethane	ND	125	70	56
Nitrobenzene	ND	125	109	87
1,4-Dichlorobenzene	ND	125	80	64
Methylphenols (total)	ND	500	319	64
Pentachlorophenol	ND	250	155	62
2,4,5-Trichlorophenol	ND	250	151	60
2,4,6-Trichlorophenol	ND	250	155	62

This matrix spike analysis summary applies to the following samples:

Tank 16

Reported by: ______________

Approved by: KC

TCLP PESTICIDES ANALYSES

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTICS LEACHING PROCEDURE (TCLP)

SURROGATE RECOVERY SUMMARY

Organochlorine Pesticides Analysis

Client: Roux Associates

Date Samples Received: 6/12/91

Project No.: 910312

Client ID	Laboratory ID	Dibutylchlorendate Recovery
Tank 16	910312-01	91 %
QA/QC		
TCLP Extraction Blank	PTCLP-0617-B1	99
Tank 16MS	910312-01MS	86

Reported by:

CEIMIC
CORPORATION

"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTIC LEACHING PROCEDURE (TCLP)

ORGANOCHLORINE PESTICIDES

EPA Method 8080

Client: Roux Associates	Date Sampled: 6/21/91
Client Sample ID: TCLP Extraction Blank	Date TCLP Performed: 6/21/91
Laboratory ID: TCLP617-B1	Date Leachate Prepared: 6/18/9
Concentration in: ug/L (ppb)	Date Extract Analyzed: 6/21/91

	Actual		Adjusted*	
Target Analyte	Sample Result	Method Reporting Limit	Sample Result	Method Reporting Limit
gamma-BHC (Lindane)	ND	0.16	ND	0.21
Heptachlor	ND	0.16	ND	0.17
Heptachlor Epoxide	ND	0.16	ND	0.17
Endrin	ND	0.33	ND	0.33
Methoxychlor	ND	0.16	ND	0.19
Toxaphene	ND	3.3	ND	---
Chlordane	ND	0.16	ND	---

ND = Not detected

* Actual sample result adjusted for matrix bias. Refer to matrix spike analysis summary form.

Reported by: ______________ Approved by: ______________

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTICS LEACHING PROCEDURE (TCLP)

ORGANICHLORINE PESTICIDES

MATRIX SPIKE ANALYSIS SUMMARY

EPA METHOD 8080

Client: Roux Associates

Client Sample ID: Tank 16MS

Laboratory ID: 910312-01

Date Analyzed: 6/21/91

Concentration in: ug/L (ppb)

Target Analyte	Sample Result	Spike Added	Spiked Sample Result	Percent Recovery
gamma-BHC (Lindane)	ND	0.2	0.5	77 %
Heptachlor	ND	0.2	0.6	93
Heptachlor Epoxide	ND	0.2	0.6	93
Endrin	ND	0.5	2.2	133
Methoxychlor	ND	1.0	2.7	82
Toxaphene	ND	NA	---	---
Chlordane	ND	NA	---	---

This matrix spike analysis summary applies to the following samples:

Tank 16

Reported by: ______________

Approved by: ______________

CEIMIC
CORPORATION

"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTIC LEACHING PROCEDURE (TCLP)

ORGANOCHLORINE PESTICIDES

EPA Method 8080

Client: Roux Associates	Date Sampled: 6/21/91
Client Sample ID: Tank 16	Date TCLP Performed: 6/21/91
Laboratory ID: 910312-01	Date Leachate Prepared: 6/18/9
Concentration in: ug/L (ppb)	Date Extract Analyzed: 6/21/91

	Actual		Adjusted*	
Target Analyte	Sample Result	Method Reporting Limit	Sample Result	Method Reporting Limit
gamma-BHC (Lindane)	ND	0.16	ND	0.21
Heptachlor	ND	0.16	ND	0.17
Heptachlor Epoxide	ND	0.16	ND	0.17
Endrin	ND	0.33	ND	0.33
Methoxychlor	ND	0.16	ND	0.19
Toxaphene	ND	3.3	ND	---
Chlordane	ND	0.16	ND	---

ND = Not detected

* Actual sample result adjusted for matrix bias. Refer to matrix spike analysis summary form.

Reported by: ______________

Approved by: ______________

TCLP HERBICIDES ANALYSES

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

SURROGATE RECOVERY SUMMARY

Organochlorine Herbicides Analysis

Client: Roux Associates

Date Samples Received: 6/12/91

Project No.: 910312

Client ID	Laboratory ID	DCPAA* Recovery
Tank 16	910312-01	84%
QA/QC		
TCLP Extraction Blank	HTCLP0628-B2	89%
Matrix Spike	910312-01MS	86%
Laboratory Control Spike	H910702-LCS1	86%

DCPAA = Dichlorophenylacetic acid

Reported by: HL

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTIC LEACHING PROCEDURE (TCLP)

ORGANOCHLORINE HERBICIDES

EPA Method 8150

Client: Roux Associates	Date Sampled: NA
Client Sample ID: TCLP Extraction Blank	Date TCLP Performed: 6/28/91
Laboratory ID: HTCLP0628-B1	Date Leachate Prepared: 7/02/9
Concentration in: ug/L (ppb)	Date Extract Analyzed: 7/09/91

	Actual		Adjusted*	
Target Analyte	Sample Result	Method Reporting Limit	Sample Result	Method Reporting Limit
2,4-D	ND	100	--	--
2,4,5-TP (Silvex)	ND	33	--	--

ND = Not detected

* Actual sample result adjusted for matrix bias. Refer to matrix spike analysis summary form.

Reported by: HL

Approved by: KC

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTIC LEACHING PROCEDURE (TCLP)

ORGANOCHLORINE HERBICIDES

EPA Method 8150

Client: Roux Associates

Client Sample ID: Tank 16

Laboratory ID: 910312-01

Concentration in: ug/L (ppb)

Date Sampled: 6/11/91

Date TCLP Performed: 6/28/91

Date Leachate Prepared: 7/02/9

Date Extract Analyzed: 7/09/91

	Actual		Adjusted*	
Target Analyte	Sample Result	Method Reporting Limit	Sample Result	Method Reporting Limit
2,4-D	ND	100	ND	140
2,4,5-TP (Silvex)	ND	33	ND	50

ND = Not detected

* Actual sample result adjusted for matrix bias. Refer to matrix spike analysis summary form.

Reported by: HL

Approved by: KC

302204

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTIC LEACHING PROCEDURE (TCLP)

ORGANOCHLORINE HERBICIDES

MATRIX SPIKE ANALYSIS SUMMARY

EPA Method 8150

Client: Roux Associates

Client Sample ID: Tank 16

Laboratory ID: 910312-01

Date Analyzed: 7/09/91

Concentration in: ug/L (ppb)

Target Analyte	Sample Result	Spike Added	Spiked Sample Result	Percent Recovery
2,4-D	ND	5.0	3.4	69%
2,4,5-TP (Silvex)	ND	1.0	0.7	68

ND = Not detected
This matrix spike analysis summary applies to the following samples:

Reported by: HL

Approved by: KC

302205

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

ORGANOCHLORINE HERBICIDES

LABORATORY CONTROL SPIKE

EPA Method 8150

Client: Roux Associates

Client Sample ID: Laboratory Control Spike

Laboratory ID: H910702-LCS1

Date Sample Received: NA

Date Sample Prepared: 7/02/91

Date Sample Analyzed: 7/09/91

Matrix: Water

Target Analyte	% Recovery
2,4-D	70 %
Silvex	64

NA = Not applicable

Reported by: HL

Approved by: KC

302206

TCLP METALS ANALYSES

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTICS LEACHING PROCEDURE (TCLP)

METALS

EPA METHOD 1311

Client: Roux Associates

Client Sample ID: Tank 16

Date Sampled: 6/11/91

Laboratory ID: 910312-01

Date TCLP performed: 6/17/91

Concentration in: mg/L (ppm)

Date Leachate Analyzed: 6/19/91

Target Analyte	Actual Sample Result	Actual Method Reporting Limit	Adjusted* Sample Result	Adjusted* Method Reporting Limit
Arsenic	0.3	0.2	0.3	0.2
Barium	0.02	0.01	0.02	0.01
Cadmium	ND	0.01	ND	0.01
Chromium	ND	0.01	ND	0.01
Lead	ND	0.1	ND	0.1
Mercury	ND	0.0008	ND	0.008
Selenium	ND	0.3	ND	0.3
Silver	ND	0.02	ND	0.02

* Actual sample result adjusted for matrix bias. Refer to matrix spike analysis summary form.

Reported by: ____________

Approved by: ____________

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

TOXICITY CHARACTERISTICS LEACHING PROCEDURE (TCLP)

METALS

MATRIX SPIKE ANALYSIS SUMMARY

EPA METHOD 1311

Client: Roux Associates

Client Sample ID: 6/11/91 Laboratory ID: 910312-01S

Date Analyzed: 6/19/91 Concentration in: mg/L (ppm)

Target Analyte	Sample Result	Spike Added	Spiked Sample Result	Percent Recovery
Arsenic	0.263	0.500	0.771	102 %
Barium	0.023	0.500	0.546	105
Cadmium	ND	0.500	0.647	129
Chromium	ND	0.500	0.529	106
Lead	ND	0.500	0.552	110
Mercury	ND	0.00100	0.00107	107
Selenium	ND	0.500	0.586	117
Silver	ND	0.500	0.475	95

This matrix spike analysis summary applies to the following samples:

Tank 16

Reported by:________ Approved by:________

CEIMIC
CORPORATION

"Analytical Chemistry for Environmental Management"

QUALITY CONTROL

METHOD BLANK

Client: Roux Associates

Client Sample ID: Method Blank

Date Sample Received: 910312

Laboratory ID: 0618PBW

Date Analysis Completed: 6/19/91

Concentration in: mg/L (ppm)

Target Analyte	Sample Concentration	Method Reporting Limits
Toxicity Characteristics Leaching Procedure (TCLP)		
Arsenic	ND	0.2
Barium	ND	0.01
Cadmium	0.01	0.01
Chromium	ND	0.01
Lead	ND	0.1
Mercury	ND	0.0008
Selenium	ND	0.3
Silver	ND	0.02

ND = Not detected

Reported by: ____________

Approved by: ____________

CEIMIC
CORPORATION

"Analytical Chemistry for Environmental Management"

QUALITY CONTROL

METHOD BLANK

Client: Roux Associates

Client Sample ID: Filtration Blank

Date Sample Received: 910312

Laboratory ID: 0617FB

Date Analysis Completed: 6/19/91

Concentration in: mg/L (ppm)

Target Analyte	Sample Concentration	Method Reporting Limits
Toxicity Characteristics Leaching Procedure (TCLP)		
Arsenic	ND	0.2
Barium	0.03	0.01
Cadmium	ND	0.01
Chromium	ND	0.01
Lead	ND	0.1
Mercury	ND	0.0008
Selenium	ND	0.3
Silver	ND	0.02

ND = Not detected

Reported by: ______________________

Approved by: ______________________

302211

CEIMIC
CORPORATION

"Analytical Chemistry for Environmental Management"

QUALITY CONTROL

LABORATORY CONTROL SAMPLE

Client: Roux Associates

Client Sample ID: Laboratory Control Sample

Project No.: 910312 Laboratory ID: 0618LCSW

Date Analysis Completed: 6/19/91 Matrix: Aqueous

Target Analyte	% Recovery	Control Limits
Toxicity Characteristic Leaching Procedure (TCLP)		
Arsenic	140 %	75-125 %
Barium	104	75-125
Cadmium	127	75-125
Chromium	106	75-125
Lead	104	75-125
Mercury	106	75-125
Selenium	112	75-125
Silver	93	75-125

Reported by:

Approved by: Phyllis Shiller

INORGANIC ANALYTES

CEIMIC
CORPORATION

"Analytical Chemistry for Environmental Management"

INORGANIC ANALYTES

Client: Roux Associates

Client ID: Tank 16

Date Sample Received: 6/12/91

Laboratory ID: 910312-01

Date Analysis Completed: 6/19/91

Target Analyte	Result	Units	Method Reporting Limit
Flashpoint	No flash	°F	200 °F
pH	7.02	S.U.	--
Reactive Cyanide	ND	mg/L (ppm)	0.5
Reactive Sulfide	ND	mg/L (ppm)	2

ND = Not detected

Reported by:

Approved by:

CEIMIC
CORPORATION
"Analytical Chemistry for Environmental Management"

QUALITY CONTROL

METHOD BLANK

Client: Roux Associates

Client ID: Method Blank

Project No.: 910312

Laboratory ID: PBW

Date Analysis Completed: 6/19/91

Target Analyte	Result	Units	Method Reporting Limit
Reactive Cyanide	ND	mg/L (ppm)	0.5
Reactive Sulfide	ND	mg/L (ppm)	2

ND = Not detected

Reported by:

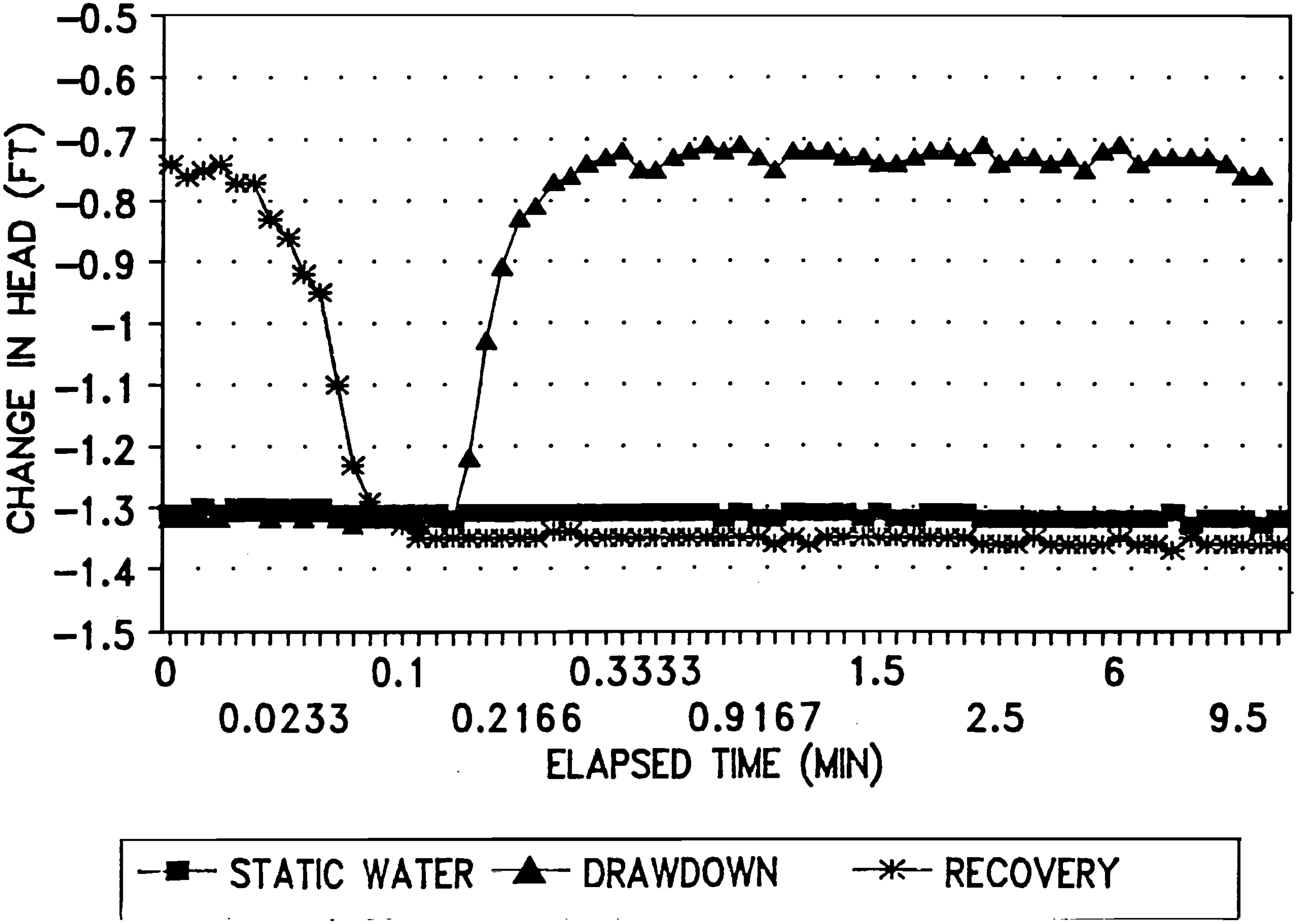
Approved by:

APPENDIX G

Pump Test Graphs

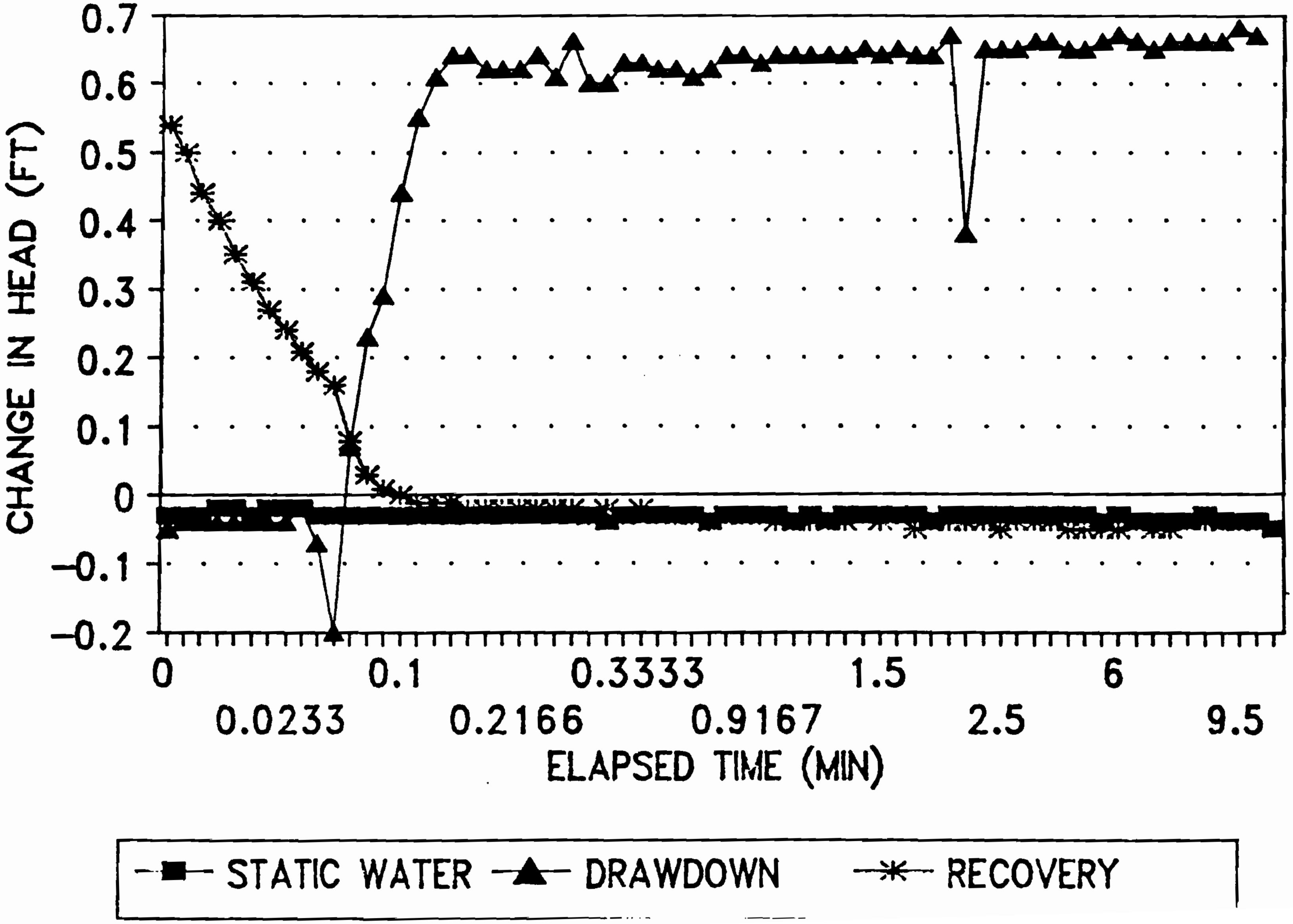
ANCHOR CHEMICAL

PUMP TEST MW-1D (04/21/92)



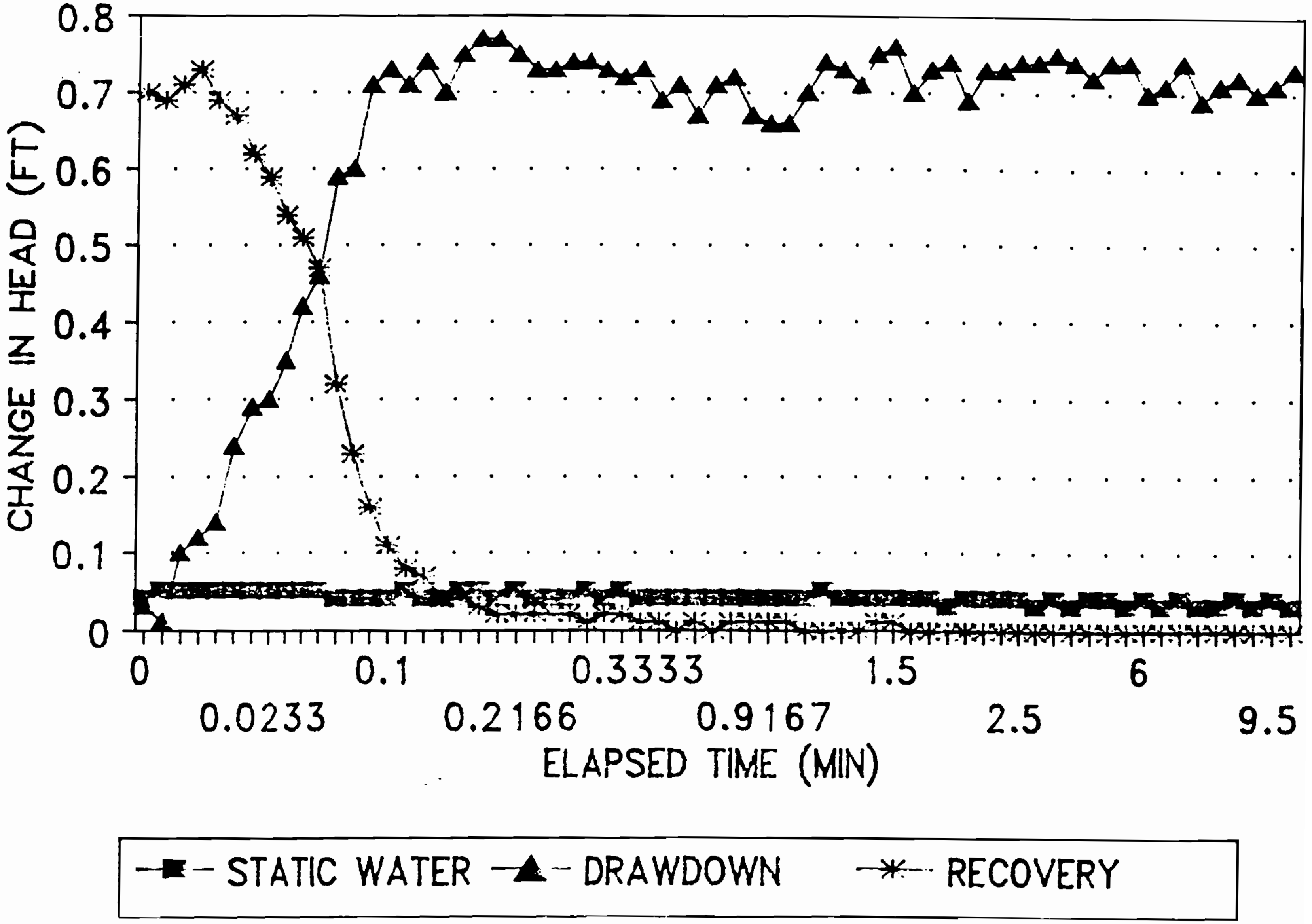
ANCHOR CHEMICAL

PUMP TEST MW-4 (04/22/92)



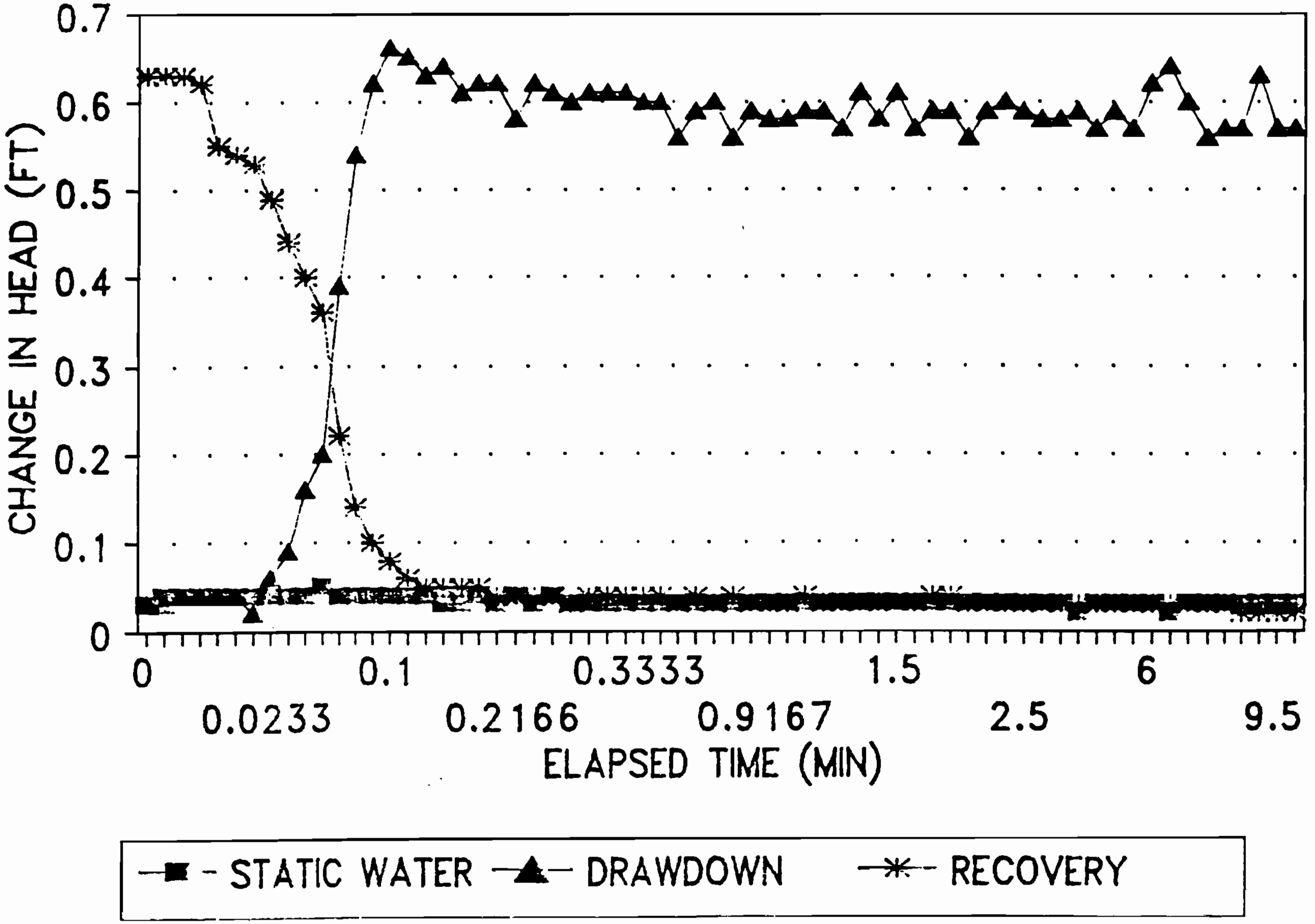
ANCHOR CHEMICAL

PUMP TEST MW-6S (04/21/92)



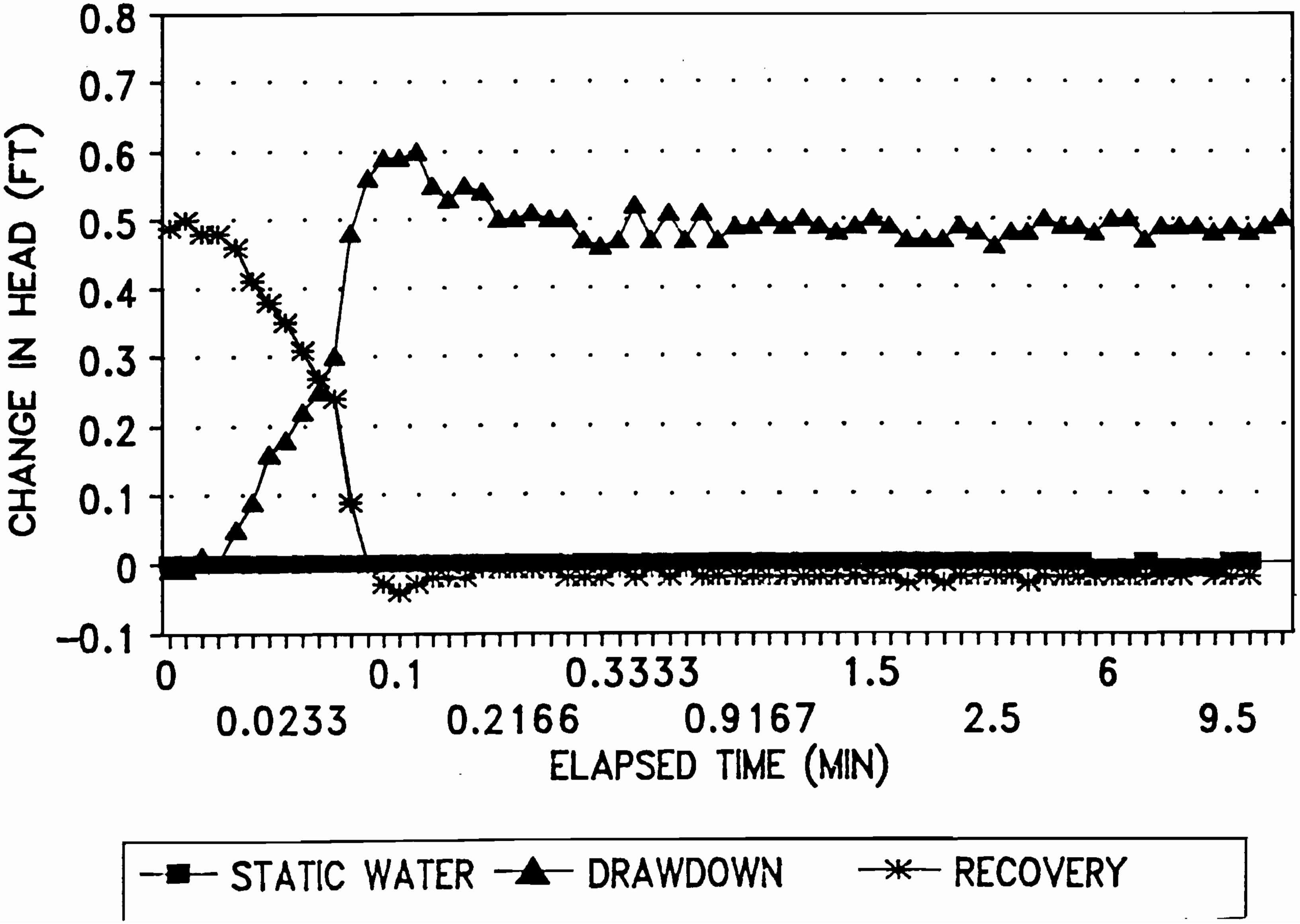
ANCHOR CHEMICAL

PUMP TEST MW-7S (04/22/92)



ANCHOR CHEMICAL

PUMP TEST MW-7D (04/22/92)



APPENDIX H

Pump Test Raw Data

```
          SE1000B
   Environmental Logger
       04/28   06:07

   Unit# 00979   Test# 0

INPUT 1:   Level  (F)  TOC

Reference            0.00
Scale factor        15.05
Offset             - 0.03

Step# 1    04/21   13:10

Elapsed Time      Value                  Elapsed Time     Value
------------   ----------                ------------   ----------
   0.0000         -1.32                     0.9167        -0.71
   0.0033          1.32                     1.0000        -0.73
   0.0066         -1.32                     1.0833        -0.75
   0.0099         -1.32                     1.1667        -0.72
   0.0133          1.32                     1.2500        -0.72
   0.0166         -1.31                     1.3333        -0.72
   0.0200          1.31                     1.4166        -0.73
   0.0233          1.32                     1.5000        -0.73
   0.0266         -1.31                     1.5833        -0.74
   0.0300         -1.32                     1.6667        -0.74
   0.0333         -1.31                     1.7500        -0.73
   0.0500         -1.32                     1.8333        -0.72
   0.0666         -1.33                     1.9167        -0.72
   0.0833          1.32                     2.0000        -0.73
   0.1000         -1.32                     2.5000        -0.71
   0.1166         -1.32                     3.0000        -0.74
   0.1333         -1.33                     3.5000        -0.73
   0.1500         -1.32                     4.0000        -0.73
   0.1666         -1.32                     4.5000        -0.74
   0.1833         -1.22                     5.0000        -0.73
   0.2000         -1.03                     5.5000        -0.75
   0.2166         -0.91                     6.0000        -0.72
   0.2333         -0.83                     6.5000        -0.71
   0.2500         -0.81                     7.0000        -0.74
   0.2666         -0.77                     7.5000        -0.73
   0.2833         -0.76                     8.0000        -0.73
   0.3000         -0.74                     8.5000        -0.73
   0.3166         -0.73                     9.0000        -0.73
   0.3333         -0.72                     9.5000        -0.74
   0.4167         -0.75                    10.0000        -0.76
   0.5000         -0.75
   0.5833         -0.73
   0.6667         -0.72
   0.7500         -0.71
   0.8333         -0.72
```

```
        SE1000B
  Environmental Logger
      04/28   06:11

  Unit# 00979   Test# 0

INPUT 1:  Level (F) TOC

Reference             0.00
Scale factor         15.05
Offset              - 0.03

Step# 2   04/21   13:21

Elapsed Time      Value
------------    ---------
   0.0000          -0.72
   0.0033          -0.74
   0.0066          -0.76
   0.0099          -0.75
   0.0133           0.74
   0.0166          -0.77
   0.0200          -0.77
   0.0233          -0.83
   0.0266          -0.86
   0.0300          -0.92
   0.0333           0.95
   0.0500          -1.10
   0.0666          -1.23
   0.0833          -1.29
   0.1000          -1.32
   0.1166          -1.33
   0.1333          -1.35
   0.1500          -1.35
   0.1666          -1.35
   0.1833          -1.35
   0.2000          -1.35
   0.2166          -1.35
   0.2333          -1.35
   0.2500          -1.35
   0.2666          -1.34
   0.2833          -1.34
   0.3000          -1.35
   0.3166          -1.35
   0.3333          -1.35
   0.4167          -1.35
   0.5000          -1.35
   0.5833          -1.35
   0.6667          -1.35
   0.7500          -1.35
   0.8333          -1.35
```

```
Elapsed Time      Value
------------    ---------
   0.9167          -1.35
   1.0000          -1.35
   1.0833          -1.36
   1.1667          -1.35
   1.2500          -1.36
   1.3333          -1.35
   1.4166          -1.35
   1.5000          -1.35
   1.5833          -1.35
   1.6667          -1.35
   1.7500          -1.35
   1.8333          -1.35
   1.9167          -1.35
   2.0000          -1.35
   2.5000          -1.36
   3.0000          -1.36
   3.5000          -1.36
   4.0000          -1.35
   4.5000          -1.36
   5.0000          -1.36
   5.5000          -1.36
   6.0000          -1.36
   6.5000          -1.35
   7.0000          -1.36
   7.5000          -1.36
   8.0000          -1.37
   8.5000          -1.35
   9.0000          -1.36
   9.5000          -1.36
  10.0000          -1.36
  12.0000          -1.36
```

```
        SE1000B
  Environmental Logger
     04/28  06:12

  Unit# 00979  Test# 1

INPUT 1:  Level (F) TOC

Reference          0.00
Scale factor      15.05
Offset           - 0.03

Step# 0   04/21  14:59

Elapsed Time      Value                Elapsed Time    Value
------------    ---------              ------------   --------
   0.0000          0.04                   0.9167         0.04
   0.0033          0.05                   1.0000         0.04
   0.0066          0.05                   1.0833         0.04
   0.0099          0.05                   1.1667         0.05
   0.0133          0.05                   1.2500         0.04
   0.0166          0.05                   1.3333         0.04
   0.0200          0.05                   1.4166         0.04
   0.0233          0.05                   1.5000         0.04
   0.0266          0.05                   1.5833         0.04
   0.0300          0.05                   1.6667         0.04
   0.0333          0.05                   1.7500         0.03
   0.0500          0.04                   1.8333         0.04
   0.0666          0.04                   1.9167         0.04
   0.0833          0.04                   2.0000         0.04
   0.1000          0.04                   2.5000         0.04
   0.1166          0.05                   3.0000         0.03
   0.1333          0.04                   3.5000         0.04
   0.1500          0.04                   4.0000         0.03
   0.1666          0.05                   4.5000         0.04
   0.1833          0.05                   5.0000         0.04
   0.2000          0.04                   5.5000         0.03
   0.2166          0.05                   6.0000         0.04
   0.2333          0.04                   6.5000         0.03
   0.2500          0.04                   7.0000         0.04
   0.2666          0.04                   7.5000         0.03
   0.2833          0.05                   8.0000         0.03
   0.3000          0.04                   8.5000         0.04
   0.3166          0.05                   9.0000         0.03
   0.3333          0.04                   9.5000         0.04
   0.4167          0.04                  10.0000         0.03
   0.5000          0.04
   0.5833          0.04
   0.6667          0.04
   0.7500          0.04
   0.8333          0.04
```

```
        SE1000B
   Environmental Logger
      04/28  06:14

   Unit# 00979  Test# 1

 INPUT 1:  Level (F) TOC

 Reference          0.00
 Scale factor      15.05
 Offset            -0.03

 Step# 1   04/21   15:10

 Elapsed Time     Value            Elapsed Time      Value
 ------------  ---------           ------------  ---------
    0.0000          0.03              0.9167          0.66
    0.0033          0.01              1.0000          0.66
    0.0066          0.10              1.0833          0.70
    0.0099          0.12              1.1667          0.74
    0.0133          0.14              1.2500          0.73
    0.0166          0.24              1.3333          0.71
    0.0200          0.29              1.4166          0.75
    0.0233          0.30              1.5000          0.76
    0.0266          0.35              1.5833          0.70
    0.0300          0.42              1.6667          0.73
    0.0333          0.46              1.7500          0.74
    0.0500          0.59              1.8333          0.69
    0.0666          0.60              1.9167          0.73
    0.0833          0.71              2.0000          0.73
    0.1000          0.73              2.5000          0.74
    0.1166          0.71              3.0000          0.74
    0.1333          0.74              3.5000          0.75
    0.1500          0.70              4.0000          0.74
    0.1666          0.75              4.5000          0.72
    0.1833          0.77              5.0000          0.74
    0.2000          0.77              5.5000          0.74
    0.2166          0.75              6.0000          0.70
    0.2333          0.73              6.5000          0.71
    0.2500          0.73              7.0000          0.74
    0.2666          0.74              7.5000          0.69
    0.2833          0.74              8.0000          0.71
    0.3000          0.73              8.5000          0.72
    0.3166          0.72              9.0000          0.70
    0.3333          0.73              9.5000          0.71
    0.4167          0.69             10.0000          0.73
    0.5000          0.71
    0.5833          0.67
    0.6667          0.71
    0.7500          0.72
    0.8333          0.67
```

```
          SE1000B
   Environmental Logger
      04/28   06:15

   Unit# 00979   Test# 1

 INPUT 1:   Level  (F)  TOC

 Reference            0.00
 Scale factor        15.05
 Offset             - 0.03

 Step# 2    04/21    15:21

 Elapsed Time      Value            Elapsed Time     Value
 ------------    ---------          ------------   ---------

    0.0000          0.70               0.9167          0.01
    0.0033          0.69               1.0000          0.01
    0.0066          0.71               1.0833          0.00
    0.0099          0.73               1.1667          0.00
    0.0133          0.69               1.2500          0.00
    0.0166          0.67               1.3333          0.00
    0.0200          0.62               1.4166          0.01
    0.0233          0.59               1.5000          0.01
    0.0266          0.54               1.5833          0.00
    0.0300          0.51               1.6667          0.00
    0.0333          0.47               1.7500          0.00
    0.0500          0.32               1.8333          0.00
    0.0666          0.23               1.9167          0.00
    0.0833          0.16               2.0000          0.00
    0.1000          0.11               2.5000          0.00
    0.1166          0.08               3.0000          0.00
    0.1333          0.07               3.5000          0.00
    0.1500          0.04               4.0000          0.00
    0.1666          0.04               4.5000          0.00
    0.1833          0.03               5.0000         -0.00
    0.2000          0.02               5.5000          0.00
    0.2166          0.02               6.0000          0.00
    0.2333          0.02               6.5000          0.00
    0.2500          0.02               7.0000          0.00
    0.2666          0.02               7.5000          0.00
    0.2833          0.01               8.0000         -0.00
    0.3000          0.02               8.5000          0.00
    0.3166          0.02               9.0000          0.00
    0.3333          0.01               9.5000          0.00
    0.4167          0.01              10.0000         -0.00
    0.5000          0.00
    0.5833          0.01
    0.6667          0.00
    0.7500          0.01
    0.8333          0.01
```

```
        SE1000B
  Environmental Logger
      04/28  06:16

  Unit# 00979  Test# 2

INPUT 1:  Level (F) TOC

Reference           0.00
Scale factor       15.05
Offset            - 0.03

Step# 0   04/22  07:03

Elapsed Time    Value              Elapsed Time    Value
------------  ---------            ------------  ---------
    0.0000        0.00                 0.9167        0.00
    0.0033        0.00                 1.0000       -0.00
    0.0066        0.00                 1.0833       -0.00
    0.0099        0.00                 1.1667       -0.00
    0.0133        0.00                 1.2500       -0.00
    0.0166        0.00                 1.3333       -0.00
    0.0200        0.00                 1.4166       -0.00
    0.0233        0.00                 1.5000       -0.00
    0.0266        0.00                 1.5833       -0.00
    0.0300        0.00                 1.6667       -0.00
    0.0333        0.00                 1.7500       -0.00
    0.0500        0.00                 1.8333       -0.00
    0.0666        0.00                 1.9167       -0.00
    0.0833        0.00                 2.0000       -0.00
    0.1000        0.00                 2.5000       -0.00
    0.1166        0.00                 3.0000       -0.00
    0.1333        0.00                 3.5000       -0.00
    0.1500        0.00                 4.0000       -0.00
    0.1666        0.00                 4.5000       -0.00
    0.1833        0.00                 5.0000       -0.00
    0.2000        0.00                 5.5000       -0.01
    0.2166        0.00                 6.0000       -0.01
    0.2333        0.00                 6.5000       -0.01
    0.2500        0.00                 7.0000       -0.00
    0.2666        0.00                 7.5000       -0.01
    0.2833        0.00                 8.0000       -0.01
    0.3000       -0.00                 8.5000       -0.01
    0.3166        0.00                 9.0000       -0.01
    0.3333        0.00                 9.5000       -0.00
    0.4167       -0.00                10.0000       -0.00
    0.5000       -0.00
    0.5833       -0.00
    0.6667       -0.00
    0.7500        0.00
    0.8333       -0.00
```

```
        SE1000B
   Environmental Logger
      04/28   06:18

   Unit# 00979   Test# 2

 INPUT 1:   Level (F) TOC

 Reference           0.00
 Scale factor       15.05
 Offset            - 0.03

 Step# 1    04/22   07:14

 Elapsed Time      Value                Elapsed Time      Value
 ------------      -----                ------------      -----
    0.0000         -0.01                   0.9167          0.49
    0.0033         -0.01                   1.0000          0.50
    0.0066          0.01                   1.0833          0.49
    0.0099          0.00                   1.1667          0.50
    0.0133          0.05                   1.2500          0.49
    0.0166          0.09                   1.3333          0.48
    0.0200          0.16                   1.4166          0.49
    0.0233          0.18                   1.5000          0.50
    0.0266          0.22                   1.5833          0.49
    0.0300          0.25                   1.6667          0.47
    0.0333          0.30                   1.7500          0.47
    0.0500          0.48                   1.8333          0.47
    0.0666          0.56                   1.9167          0.49
    0.0833          0.59                   2.0000          0.48
    0.1000          0.59                   2.5000          0.46
    0.1166          0.60                   3.0000          0.48
    0.1333          0.55                   3.5000          0.48
    0.1500          0.53                   4.0000          0.50
    0.1666          0.55                   4.5000          0.49
    0.1833          0.54                   5.0000          0.49
    0.2000          0.50                   5.5000          0.48
    0.2166          0.50                   6.0000          0.50
    0.2333          0.51                   6.5000          0.50
    0.2500          0.50                   7.0000          0.47
    0.2666          0.50                   7.5000          0.49
    0.2833          0.47                   8.0000          0.49
    0.3000          0.46                   8.5000          0.49
    0.3166          0.47                   9.0000          0.48
    0.3333          0.52                   9.5000          0.49
    0.4167          0.47                  10.0000          0.48
    0.5000          0.51                  12.0000          0.49
    0.5833          0.47                  14.0000          0.50
    0.6667          0.51
    0.7500          0.47
    0.8333          0.49
```

```
        SE1000B
  Environmental Logger
      04/28   06:19

  Unit# 00979   Test# 2

INPUT 1:   Level (F) TOC

Reference            0.00
Scale factor        15.05
Offset             - 0.03

Step# 2    04/22   07:30

Elapsed Time     Value                Elapsed Time     Value
------------   ----------             ------------   ---------
   0.0000          0.49                  0.9167         -0.02
   0.0033          0.50                  1.0000         -0.02
   0.0066          0.48                  1.0833         -0.02
   0.0099          0.48                  1.1667         -0.02
   0.0133          0.46                  1.2500         -0.02
   0.0166          0.41                  1.3333         -0.02
   0.0200          0.38                  1.4166         -0.02
   0.0233          0.35                  1.5000         -0.02
   0.0266          0.31                  1.5833         -0.02
   0.0300          0.27                  1.6667         -0.03
   0.0333          0.24                  1.7500         -0.02
   0.0500          0.09                  1.8333         -0.03
   0.0666          0.00                  1.9167         -0.02
   0.0833         -0.03                  2.0000         -0.02
   0.1000         -0.04                  2.5000         -0.02
   0.1166         -0.03                  3.0000         -0.02
   0.1333         -0.02                  3.5000         -0.03
   0.1500         -0.02                  4.0000         -0.02
   0.1666         -0.02                  4.5000         -0.02
   0.1833         -0.01                  5.0000         -0.02
   0.2000         -0.01                  5.5000         -0.02
   0.2166         -0.01                  6.0000         -0.02
   0.2333         -0.01                  6.5000         -0.02
   0.2500         -0.01                  7.0000         -0.02
   0.2666         -0.02                  7.5000         -0.02
   0.2833         -0.02                  8.0000         -0.02
   0.3000         -0.02                  8.5000         -0.01
   0.3166         -0.01                  9.0000         -0.02
   0.3333         -0.02                  9.5000         -0.02
   0.4167         -0.01                 10.0000         -0.02
   0.5000         -0.02
   0.5833         -0.01
   0.6667         -0.02
   0.7500          0.02
   0.8333         -0.02
```

```
        SE1000B
  Environmental Logger
      04/28  06:21

  Unit# 00979  Test# 3

INPUT 1:  Level (F) TOC

Reference           0.00
Scale factor       15.05
Offset            - 0.03

Step# 0    04/22  08:59

Elapsed Time     Value           Elapsed Time     Value
------------   ---------         ------------   ---------
   0.0000         0.03              0.9167         0.03
   0.0033         0.04              1.0000         0.03
   0.0066         0.04              1.0833         0.03
   0.0099         0.04              1.1667         0.03
   0.0133         0.04              1.2500         0.03
   0.0166         0.04              1.3333         0.03
   0.0200         0.04              1.4166         0.03
   0.0233         0.04              1.5000         0.03
   0.0266         0.04              1.5833         0.03
   0.0300         0.04              1.6667         0.03
   0.0333         0.05              1.7500         0.03
   0.0500         0.04              1.8333         0.03
   0.0666         0.04              1.9167         0.03
   0.0833         0.04              2.0000         0.03
   0.1000         0.04              2.5000         0.03
   0.1166         0.04              3.0000         0.03
   0.1333         0.04              3.5000         0.03
   0.1500         0.03              4.0000         0.02
   0.1666         0.03              4.5000         0.03
   0.1833         0.04              5.0000         0.03
   0.2000         0.03              5.5000         0.03
   0.2166         0.04              6.0000         0.03
   0.2333         0.03              6.5000         0.02
   0.2500         0.04              7.0000         0.03
   0.2666         0.03              7.5000         0.03
   0.2833         0.03              8.0000         0.03
   0.3000         0.03              8.5000         0.03
   0.3166         0.03              9.0000         0.03
   0.3333         0.03              9.5000         0.03
   0.4167         0.03             10.0000         0.03
   0.5000         0.03
   0.5833         0.03
   0.6667         0.03
   0.7500         0.03
   0.8333         0.03
```

```
          SE1000B
   Environmental Logger
       04/28  06:00

   Unit# 00979  Test# 0

 INPUT 1:  Level (F) TOC

 Reference         0.00
 Scale factor     15.05
 Offset          - 0.03

 Step# 0   04/21  12:58

 Elapsed Time    Value              Elapsed Time     Value
 ------------  ---------            ------------   ---------

    0.0000       -1.31                 0.9167        -1.32
    0.0033       -1.31                 1.0000        -1.32
    0.0066       -1.30                 1.0833        -1.31
    0.0099       -1.31                 1.1667        -1.31
    0.0133       -1.30                 1.2500        -1.31
    0.0166       -1.30                 1.3333        -1.31
    0.0200       -1.30                 1.4166        -1.32
    0.0233       -1.30                 1.5000        -1.31
    0.0266       -1.30                 1.5833        -1.32
    0.0300       -1.30                 1.6667        -1.32
    0.0333       -1.31                 1.7500        -1.31
    0.0500       -1.31                 1.8333        -1.31
    0.0666       -1.31                 1.9167        -1.31
    0.0833       -1.31                 2.0000        -1.32
    0.1000       -1.31                 2.5000        -1.32
    0.1166       -1.31                 3.0000        -1.32
    0.1333       -1.31                 3.5000        -1.32
    0.1500       -1.32                 4.0000        -1.32
    0.1666       -1.31                 4.5000        -1.32
    0.1833       -1.31                 5.0000        -1.32
    0.2000       -1.31                 5.5000        -1.32
    0.2166       -1.31                 6.0000        -1.32
    0.2333       -1.31                 6.5000        -1.32
    0.2500       -1.31                 7.0000        -1.32
    0.2666       -1.31                 7.5000        -1.31
    0.2833       -1.31                 8.0000        -1.33
    0.3000       -1.31                 8.5000        -1.32
    0.3166       -1.31                 9.0000        -1.32
    0.3333       -1.31                 9.5000        -1.32
    0.4167       -1.31                10.0000        -1.33
    0.5000       -1.31                12.0000        -1.32
    0.5833       -1.31
    0.6667       -1.31
    0.7500       -1.32
    0.8333       -1.31
```

302232

```
         SE1000B
   Environmental Logger
       04/28   06:21

   Unit# 00979   Test# 3

 INPUT 1:   Level  (F)  TOC

 Reference               0.00
 Scale factor           15.05
 Offset                - 0.03

 Step# 0    04/22   08:59

 Elapsed Time      Value              Elapsed Time      Value
 ------------    ----------           ------------    ----------
     0.0000          0.03                 0.9167          0.03
     0.0033          0.04                 1.0000          0.03
     0.0066          0.04                 1.0833          0.03
     0.0099          0.04                 1.1667          0.03
     0.0133          0.04                 1.2500          0.03
     0.0166          0.04                 1.3333          0.03
     0.0200          0.04                 1.4166          0.03
     0.0233          0.04                 1.5000          0.03
     0.0266          0.04                 1.5833          0.03
     0.0300          0.04                 1.6667          0.03
     0.0333          0.05                 1.7500          0.03
     0.0500          0.04                 1.8333          0.03
     0.0666          0.04                 1.9167          0.03
     0.0833          0.04                 2.0000          0.03
     0.1000          0.04                 2.5000          0.03
     0.1166          0.04                 3.0000          0.03
     0.1333          0.04                 3.5000          0.03
     0.1500          0.03                 4.0000          0.02
     0.1666          0.03                 4.5000          0.03
     0.1833          0.04                 5.0000          0.03
     0.2000          0.03                 5.5000          0.03
     0.2166          0.04                 6.0000          0.03
     0.2333          0.03                 6.5000          0.02
     0.2500          0.04                 7.0000          0.03
     0.2666          0.03                 7.5000          0.03
     0.2833          0.03                 8.0000          0.03
     0.3000          0.03                 8.5000          0.03
     0.3166          0.03                 9.0000          0.03
     0.3333          0.03                 9.5000          0.03
     0.4167          0.03                10.0000          0.03
     0.5000          0.03
     0.5833          0.03
     0.6667          0.03
     0.7500          0.03
     0.8333          0.03
```

```
          SE1000B
    Environmental Logger
       04/28  06:22

    Unit# 00979  Test# 3

  INPUT 1:  Level (F) TOC

  Reference          0.00
  Scale factor      15.05
  Offset           - 0.03

  Step# 1   04/22  09:11

  Elapsed Time     Value            Elapsed Time     Value
  ------------  ----------          ------------  ----------
     0.0000         0.03               0.9167         0.58
     0.0033         0.03               1.0000         0.58
     0.0066         0.04               1.0833         0.59
     0.0099         0.04               1.1667         0.59
     0.0133         0.04               1.2500         0.57
     0.0166         0.04               1.3333         0.61
     0.0200         0.02               1.4166         0.58
     0.0233         0.06               1.5000         0.61
     0.0266         0.09               1.5833         0.57
     0.0300         0.16               1.6667         0.59
     0.0333         0.20               1.7500         0.59
     0.0500         0.39               1.8333         0.56
     0.0666         0.54               1.9167         0.59
     0.0833         0.62               2.0000         0.60
     0.1000         0.66               2.5000         0.59
     0.1166         0.65               3.0000         0.58
     0.1333         0.63               3.5000         0.58
     0.1500         0.64               4.0000         0.59
     0.1666         0.61               4.5000         0.57
     0.1833         0.62               5.0000         0.59
     0.2000         0.62               5.5000         0.57
     0.2166         0.58               6.0000         0.62
     0.2333         0.62               6.5000         0.64
     0.2500         0.61               7.0000         0.60
     0.2666         0.60               7.5000         0.56
     0.2833         0.61               8.0000         0.57
     0.3000         0.61               8.5000         0.57
     0.3166         0.61               9.0000         0.63
     0.3333         0.60               9.5000         0.57
     0.4167         0.60              10.0000         0.57
     0.5000         0.56
     0.5833         0.59
     0.6667         0.60
     0.7500         0.56
     0.8333         0.59
```

```
        SE1000B
  Environmental Logger
      04/28   06:24

  Unit# 00979   Test# 3

INPUT 1:  Level (F) TOC

Reference           0.00
Scale factor       15.05
Offset            - 0.03

Step# 2   04/22   09:21

Elapsed Time     Value           Elapsed Time    Value
------------   ---------         ------------  ---------
    0.0000        0.63              0.9167        0.03
    0.0033        0.63              1.0000        0.03
    0.0066        0.63              1.0833        0.04
    0.0099        0.62              1.1667        0.03
    0.0133        0.55              1.2500        0.03
    0.0166        0.54              1.3333        0.03
    0.0200        0.53              1.4166        0.03
    0.0233        0.49              1.5000        0.03
    0.0266        0.44              1.5833        0.03
    0.0300        0.40              1.6667        0.04
    0.0333        0.36              1.7500        0.04
    0.0500        0.22              1.8333        0.03
    0.0666        0.14              1.9167        0.03
    0.0833        0.10              2.0000        0.03
    0.1000        0.08              2.5000        0.03
    0.1166        0.06              3.0000        0.03
    0.1333        0.05              3.5000        0.03
    0.1500        0.05              4.0000        0.03
    0.1666        0.05              4.5000        0.03
    0.1833        0.05              5.0000        0.03
    0.2000        0.04              5.5000        0.03
    0.2166        0.04              6.0000        0.03
    0.2333        0.04              6.5000        0.03
    0.2500        0.04              7.0000        0.03
    0.2666        0.03              7.5000        0.03
    0.2833        0.04              8.0000        0.03
    0.3000        0.04              8.5000        0.02
    0.3166        0.04              9.0000        0.02
    0.3333        0.04              9.5000        0.02
    0.4167        0.04             10.0000        0.02
    0.5000        0.03
    0.5833        0.04
    0.6667        0.03
    0.7500        0.04
    0.8333        0.03
```

```
        SE1000B
   Environmental Logger
      04/28   06:25

   Unit# 00979   Test# 4

 INPUT 1:   Level  (F) TOC

 Reference               0.00
 Scale  factor          15.05
 Offset                 -0.03

 Step# 0     04/22    10:14

 Elapsed Time      Value                Elapsed Time      Value
 ------------     -------               ------------    ---------
     0.0000        -0.03                   0.9167          -0.03
     0.0033        -0.03                   1.0000          -0.03
     0.0066        -0.03                   1.0833          -0.04
     0.0099        -0.02                   1.1667          -0.03
     0.0133        -0.02                   1.2500          -0.04
     0.0166        -0.03                   1.3333          -0.03
     0.0200        -0.02                   1.4166          -0.03
     0.0233        -0.02                   1.5000          -0.03
     0.0266        -0.02                   1.5833          -0.03
     0.0300        -0.03                   1.6667          -0.03
     0.0333        -0.03                   1.7500          -0.04
     0.0500        -0.03                   1.8333          -0.03
     0.0666        -0.03                   1.9167          -0.03
     0.0833        -0.03                   2.0000          -0.03
     0.1000        -0.03                   2.5000          -0.03
     0.1166        -0.03                   3.0000          -0.03
     0.1333        -0.03                   3.5000          -0.03
     0.1500        -0.03                   4.0000          -0.03
     0.1666        -0.03                   4.5000          -0.03
     0.1833        -0.03                   5.0000          -0.03
     0.2000        -0.03                   5.5000          -0.04
     0.2166        -0.03                   6.0000          -0.03
     0.2333        -0.03                   6.5000          -0.04
     0.2500        -0.03                   7.0000          -0.04
     0.2666        -0.03                   7.5000          -0.04
     0.2833        -0.03                   8.0000          -0.04
     0.3000        -0.04                   8.5000          -0.03
     0.3166        -0.03                   9.0000          -0.04
     0.3333        -0.03                   9.5000          -0.04
     0.4167        -0.03                  10.0000          -0.04
     0.5000        -0.03                  12.0000          -0.05
     0.5833        -0.03
     0.6667        -0.04
     0.7500        -0.03
     0.8333        -0.03
```

```
         SE1000B
   Environmental Logger
      04/28   06:26

   Unit# 00979   Test# 4

 INPUT 1:  Level (F) TOC

 Reference            0.00
 Scale factor        15.05
 Offset             - 0.03

 Step# 1    04/22   10:27

 Elapsed Time      Value               Elapsed Time      Value
                                       ------------    ---------
     0.0000         -0.05                 0.9167          0.63
     0.0033         -0.04                 1.0000          0.64
     0.0066         -0.04                 1.0833          0.64
     0.0099          0.04                 1.1667          0.64
     0.0133         -0.04                 1.2500          0.64
     0.0166         -0.04                 1.3333          0.64
     0.0200         -0.04                 1.4166          0.65
     0.0233         -0.04                 1.5000          0.64
     0.0266         -0.02                 1.5833          0.65
     0.0300         -0.07                 1.6667          0.64
     0.0333         -0.20                 1.7500          0.64
     0.0500          0.07                 1.8333          0.67
     0.0666          0.23                 1.9167          0.38
     0.0833          0.29                 2.0000          0.65
     0.1000          0.44                 2.5000          0.65
     0.1166          0.55                 3.0000          0.65
     0.1333          0.61                 3.5000          0.66
     0.1500          0.64                 4.0000          0.66
     0.1666          0.64                 4.5000          0.65
     0.1833          0.62                 5.0000          0.65
     0.2000          0.62                 5.5000          0.66
     0.2166          0.62                 6.0000          0.67
     0.2333          0.64                 6.5000          0.66
     0.2500          0.61                 7.0000          0.65
     0.2666          0.66                 7.5000          0.66
     0.2833          0.60                 8.0000          0.66
     0.3000          0.60                 8.5000          0.66
     0.3166          0.63                 9.0000          0.66
     0.3333          0.63                 9.5000          0.68
     0.4167          0.62                10.0000          0.67
     0.5000          0.62
     0.5833          0.61
     0.6667          0.62
     0.7500          0.64
     0.8333          0.64
```

```
          SE1000B
   Environmental Logger
       04/28  06:28

   Unit# 00979  Test# 4

 INPUT 1:  Level (F) TOC

 Reference           0.00
 Scale factor       15.05
 Offset              0.03

 Step# 2   04/22   10:38

 Elapsed Time     Value          Elapsed Time     Value
 ------------   ---------        ------------   ---------
    0.0000         0.54             0.9167        -0.03
    0.0033         0.50             1.0000        -0.04
    0.0066         0.44             1.0833        -0.04
    0.0099         0.40             1.1667        -0.04
    0.0133         0.35             1.2500        -0.04
    0.0166         0.31             1.3333        -0.04
    0.0200         0.27             1.4166        -0.03
    0.0233         0.24             1.5000        -0.04
    0.0266         0.21             1.5833        -0.03
    0.0300         0.18             1.6667        -0.05
    0.0333         0.16             1.7500        -0.04
    0.0500         0.08             1.8333        -0.04
    0.0666         0.03             1.9167        -0.04
    0.0833         0.01             2.0000        -0.04
    0.1000        -0.00             2.5000        -0.05
    0.1166        -0.01             3.0000        -0.04
    0.1333        -0.01             3.5000        -0.04
    0.1500        -0.01             4.0000        -0.04
    0.1666        -0.02             4.5000        -0.05
    0.1833         0.02             5.0000        -0.05
    0.2000        -0.02             5.5000        -0.05
    0.2166        -0.02             6.0000        -0.05
    0.2333        -0.02             6.5000        -0.04
    0.2500        -0.02             7.0000        -0.05
    0.2666        -0.02             7.5000        -0.05
    0.2833        -0.03             8.0000        -0.04
    0.3000        -0.02             8.5000        -0.04
    0.3166        -0.03             9.0000        -0.04
    0.3333        -0.02             9.5000        -0.04
    0.4167         0.03            10.0000        -0.04
    0.5000        -0.03
    0.5833         0.03
    0.6667        -0.04
    0.7500        -0.03
    0.8333        -0.03
```

APPENDIX I

Nassau County Well Listing

COMPONENT CODES

12* SEWER DISTRICT

NC01	NASSAU COUNTY DISTRICT #1
NC02	NASSAU COUNTY DISTRICT #2
NC03	NASSAU COUNTY DISTRICT #3
NC04	NASSAU COUNTY DISTRICT #4
CG05	GLEN COVE SEWER DISTRICT
FS06	FREEPORT SEWER DISTRICT
LB07	LONG BEACH SEWER DISTRICT
PW08	PORT WASHINGTON SEWER DISTRICT
BG09	BELGRAVE SEWER DISTRICT
RO10	ROSLYN SEWER DISTRICT
GN11	GREAT NECK SEWER DISTRICT
VG12	VILLAGE OF GREAT NECK SEWER DISTRICT
JB13	JONES BEACH SEWER DISTRICT
OB14	OYSTER BAY SEWER DISTRICT

22* STATUS

AB	ABANDONED
NU	NOT IN USE
OC	OCCASIONAL USE
SS	SEASONAL (SUMMER)
YR	YEAR-ROUND USE
OB	OBSERVATION

30* AQUIFER

1	GLACIAL
2	MAGOTHY
3	LLOYD
4	JAMECO AND GLACIAL (COMMON SUCTION)
5	MAGOTHY AND GLACIAL (COMMON SUCTION)
6	JAMECO
7	PORT WASHINGTON

21* Welltype

99	All Abandoned wells (wn C22 = AB ∴ C21 = 99)

302240

1/

2

NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-00010	JAMAICA W S CO	ELMONT	15	400	330	404224	734240	NC02	60	800	2	TOH	AB
N-00011	JAMAICA W S CO	ELMONT	15A	409	375	404224	734240	NC02	60	800	2	TOH	AB
N-00012	JAMAICA W S CO	ELMONT	15B	423	365	404224	734240	NC02	60	800	2	TOH	AB
N-00013A	JAMAICA W S CO	ELMONT	15C	287	229	404224	734240	NC02	60	800	2	TOH	AB
N-00014	JAMAICA W S CO	NO NHP	9	106	85	404411	734137	NC02	100	800	1	TOH	OC
N-00015	JAMAICA W S CO	NO NHP	16	102	81	404426	734150	NC02	115	700	1	TOH	AB
N-00016	KNICKERBOCKER ICE	G C PK	1	444	378	404419	734016	NC02	95	860	2	TNH	NU
N-00017	JAMAICA W S CO	NO NHP	20	465	405	404437	734023	NC02	100	800	2	TOH	YR
N-00022	GREAT NECK NORTH W A	MANHST	2	145	126	404650	734409	NC04	25	1050	2	TNH	SS
N-00023	GREAT NECK NORTH W A	MANHST	3	460	404	404650	734445	NC04	25	350	3	TNH	AB
N-00024	MANHASSET-LAKEVL W D	KENSGTN	5	419	359	404737	734235	GN11	20	1200	3	TNH	OB
N-00028	PLANDOME VILLAGE	PLANDOME	1	136	111	404834	734146	NC04	40	450	1	TNH	YR
N-00029	PLANDOME VILLAGE	PLANDOME	2	207	182	404830	734148	NC04	40	350	1	TNH	YR
N-00030	GREAT NECK NORTH W A	KNGS PT	1	203	164	404902	734430	NC04	45	700	7	TNH	NU
N-00031	GREAT NECK NORTH W A	KNGS PT	4	229	183	404856	734426	NC04	45	1050	7	TNH	SS
N-00036	SANDS POINT VILL	SNDS PT	1	214	200	405111	734302	NC04	60	650	7	TNH	YR
N-00037	SANDS POINT VILL	SNDS PT	2	140	120	405113	734302	NC04	60	650	1	TNH	YR
N-00038	FALAISE	SNDS PT	1	422	381	405132	734141	NC04	70	200	3	TNH	AB
N-00041	LONG BEACH CITY	LNG BCH	3	1255	1195	403534	733955	LB07	5	1000	3	TOH	AB
N-00042	LONG BEACH CITY	LNG BCH	4	1184	1144	403534	733955	LB07	5	510	3	TOH	AB
N-00043	LONG BEACH CITY	LNG BCH	5	1264	1193	403533	734010	LB07	5	1000	3	TOH	AB
N-00044	LONG BEACH CITY	LNG BCH	6	1259	1189	403530	734023	LB07	10	700	3	TOH	AB
N-00046	LIDO-PT LOOKOUT W D	LIO BCH	1	1260	1200	403534	733531	LB07	5	1000	3	TOH	YR
N-00048	ROCKVILLE CTR VILL	RVC	1	528	449	403924	733919	NC02	15	1200	2	TOH	AB
N-00049	ROCKVILLE CTR VILL	RVC	2	330	304	403924	733919	NC02	15	800	2	TOH	AB
N-00050	ROCKVILLE CTR VILL	RVC	3	513	442	403924	733919	NC02	15	1100	2	TOH	NU
N-00052	ROCKVILLE CTR VILL	RVC	4	539	469	403932	733827	NC02	20	1500	2	TOH	AB
N-00068	FREEPORT VILLAGE	FRPT	5	500	450	403924	733528	FS06	20	1100	2	TOH	YR
N-00069	FREEPORT VILLAGE	FRPT	6	494	444	403924	733535	FS06	20	1400	2	TOH	YR
N-00072	ROCKVILLE CTR VILL	RVC	5	604	544	404109	733742	NC02	40	1140	2	TOH	YR
N-00075	WEST HEMPSTEAD W D	W HEMP	1	181	133	404138	733842	NC02	60	1200	2	TOH	YR
N-00076	WEST HEMPSTEAD W D	W HEMP	2	193	145	404138	733842	NC02	60	1000	2	TOH	YR
N-00079	HEMPSTEAD VILLAGE	HEMPSTD	2	428	338	404253	733712	NC02	60	1275	2	TOH	YR
N-00080	HEMPSTEAD VILLAGE	HEMPSTD	3	478	428	404255	733712	NC02	60	900	2	TOH	YR
N-00081	HEMPSTEAD VILLAGE	HEMPSTD	4	420	360	404305	733712	NC02	60	1000	2	TOH	YR
N-00082	HEMPSTEAD VILLAGE	HEMPSTD	5	542	390	404305	733712	NC02	60	1000	2	TOH	YR
N-00083	HEMPSTEAD VILLAGE	HEMPSTD	6	403	363	404305	733712	NC02	60	1000	2	TOH	YR
N-00091	GARDEN CITY VILLAGE	GDN CTY	2	90	82	404357	733830	NC02	80	900	1	TOH	AB
N-00093	GARDEN CITY VILLAGE	GDN CTY	5	77	44	404357	733830	NC02	80	900	1	TOH	AB
N-00094	GARDEN CITY VILLAGE	GDN CTY	6	372	322	404357	733830	NC02	88	1050	2	TOH	NU
N-00095	GARDEN CITY VILLAGE	GDN CTY	7	534	474	404357	733830	NC02	88	1050	2	TOH	YR
N-00097	MINEOLA VILLAGE	MINEOLA	1	355	295	404446	733815	NC02	100	1200	2	TNH	YR
N-00098	MINEOLA VILLAGE	MINEOLA	2	366	308	404446	733815	NC02	100	750	2	TNH	AB
N-00100	ECONOMY FOOD	MINEOLA	1	87	72	404431	733810	NC02	105	85	1	TNH	YR
N-00101	WESTBURY W D	WSTBY	6	341	280	404526	733534	NC03	110	1000	2	TNH	YR
N-00102	WHEATLEY HILLS G C	E WILL	1	92	71	404538	733747	NC02	175	400	1	TNH	YR
N-00103	WILLISTON PK VILL	WIL PK	1	380	330	404556	733831	NC02	110	1000	2	TNH	YR
N-00104	WILLISTON PK VILL	WIL PK	2	376	325	404556	733831	NC02	110	750	2	TNH	YR
N-00105	OLD WESTBURY VILL	C WSTBY	2	452	412	404652	733727	NC04	170	500	2	TNH	AB
N-00107	OLD WESTBURY VILL	O WSTBY	3	491	455	404734	733526	NC04	220	500	2	TNH	NU
N-00115	LOCUST VALLEY W D	LOC VLY	1	407	340	405244	733509	NC04	100	400	3	TOB	AB
N-00118	LOCUST VALLEY W D	LOC VLY	4	471	412	405244	733509	NC04	100	1100	3	TOB	OC
N-00119	LOCUST VALLEY W D	LOC VLY	5	571	497	405244	733513	NC04	80	1600	3	TOB	NU
N-00121	THE CREEK CLUB	LATNGTN	1	218	149	405308	733551	NC04	120	400	2	TOB	YR

NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-00129	JONES BEACH ST PK	WANTAGH	2	951	882	403606	733016	JB13	10	1000	2	TOH	OB
N-00131	FREEPORT VILLAGE	FRPT	1	523	473	403952	733422	FS06	20	0	2	TOH	AB
N-00132	FREEPORT VILLAGE	FRPT	2	507	466	403952	733422	FS06	20	900	2	TOH	YR
N-00133	FREEPORT VILLAGE	FRPT	3	511	470	403952	733422	FS06	20	1000	2	TOH	YR
N-00134	FREEPORT VILLAGE	FRPT	4	517	467	403952	733422	FS06	20	1500	2	TOH	YR
N-00148	HICKSVILLE W D	HKSVL	2-1	153	133	404441	733209	NC03	120	650	2	TOB	AB
N-00149	HICKSVILLE W D	HKSVL	1-1	152	127	404639	733111	NC03	165	640	2	TOB	AB
N-00152	OLD WESTBURY VILL	O WSTBY	1	478	438	404628	733419	NC04	140	500	2	TNH	YR
N-00157	AHRC	BKVL	1	360	335	404831	733451	NC04	280	300	2	TOB	AB
N-00189	BETHPAGE ST PARK	FRMGDL	1	183	150	404451	732648	NC03	145	550	2	TOB	NU
N-00198	JERICHO W D	SYOSSET	3	617	567	404917	732931	NC04	230	1130	2	TOB	YR
N-00199	JERICHO W D	SYOSSET	4	600	544	404920	732931	NC04	220	1120	2	TOB	YR
N-00204	CTR ISLAND VILLAGE	CTR ISL	1	---	---	405409	733123	NC04	500	---	3	TOB	YR
N-00361	GARDEN CITY VILLAGE	GDN CTY	1	40	25	404357	733830	NC02	80	1000	1	TOH	AB
• N-00558	MANHASSET LAUNDRY	GT NK	1	182	172	404644	734400	BG09	44	40	2	TNH	YR
N-00570	JERICHO W D	SYOSSET	5	600	560	404922	732922	NC04	220	1200	2	TOB	YR
N-00578	MINEOLA VILLAGE	MINEOLA	3	407	357	404458	733913	NC02	100	1000	2	TNH	NU
N-00585	OYSTER BAY W D	O BAY	PLT1	77	57	405231	733230	OB14	20	750	1	TOB	YR
N-00590	SAGAMORE HILL	COV NK	2	150	140	405308	733000	NC04	130	35	1	TOB	YR
N-00617	BETHPAGE ST PARK	FRMGDL	3	180	150	404451	732648	NC03	146	1000	2	TOB	SS
N-00629	BETH-DAVID CEM	ELMONT	1	71	49	404144	734145	NC02	50	375	1	TOH	YR
N-00638	OLD WESTBURY C C	O WSTBY	1	560	545	404802	733615	NC03	295	500	2	TOB	YR
N-00650	GARDEN CITY PARK W D	HERRCKS	1	346	306	404534	733933	NC02	120	700	2	TNH	YR
N-00651	GARDEN CITY PARK W D	HERRCKS	2	340	300	404534	733933	NC02	120	500	2	TNH	YR
N-00660	POWERS CHEMCO	GLN CV	1	404	---	405153	733823	GC05	58	500	3	TOB	YR
N-00662	BAR BEACH	PT WASH	0	364	348	404940	733927	PW08	5	0	3	TNH	NU
N-00687	GREAT NECK NORTH W A	SAD RK	5	309	279	404744	734442	GN11	10	700	3	TNH	YR
N-00693	JAMAICA W S CO	ELMONT	15D	93	68	404224	734240	NC02	60	1700	1	TOH	OC
N-00700	GREAT NECK NORTH W A	MANHST	21A	70	46	404653	734409	NC04	20	1050	1	TNH	SS
N-00706	FARMINGDALE VILLAGE	FRMGDL	1-2	70	55	404400	732614	NC03	70	500	1	TOB	AB
N-00728	N Y WATER SERVICE	MSPQ E	1	37	32	404058	732605	NC03	25	520	1	TOB	AB
N-00729	N Y WATER SERVICE	MSPQ E	2	80	70	404058	732605	NC03	25	1400	1	TOB	AB
N-00735	OYSTER BAY W D	O BAY	4	100	85	405231	733230	OB14	20	150	1	TOB	YR
N-00736	OYSTER BAY W D	O BAY	5	70	56	405231	733230	OB14	20	150	1	TOB	YR
N-00801	GLEN COVE CITY	GLN CV	0	36	---	405128	733725	GC05	50	---	5	TOB	AB
N-00827	WESTBURY W D	WSTBY	3	240	---	404526	733534	NC03	110	100	2	TNH	AB
N-00828	WESTBURY W D	WSTBY	4	350	---	404526	733534	NC03	110	250	2	TNH	AB
N-00829	WESTBURY W D	WSTBY	5	252	238	404527	733523	NC03	110	300	2	TNH	AB
N-00835	GLEN COVE CITY	GLN CV	M	300	276	405347	733755	GC05	20	550	3	TOB	YR
N-00901	SEA CLIFF W S CO	SEA CLF	0	68	47	405018	733902	NC04	15	---	1	TOB	NU
N-01037C	SEA CLIFF W S CO	SEA CLF	--	---	---	------	------	NC04	---	---	--	TOB	NU
N-01102	NCDPW	LK SUCC	C2	166	161	404611	734216	NC02	184	0	1	TNH	OB
N-01118	NCDPW	SNDS PT	E2	82	77	405048	734043	NC04	147	0	1	TNH	OB
N-01120	NCDPW	FLWR HL	E4	100	95	404838	734040	PW08	116	0	2	TNH	OB
N-01139	NCDPW	GDN CTY	F-6	68	63	404419	733803	NC02	103	0	1	TOH	OB
N-01141	NCDPW	GDN CTY	F-8	43	38	404305	733749	NC02	77	0	1	TOH	OB
N-01147	NCDPW	RVC	F14	24	19	403944	733713	NC02	27	0	1	TOH	OB
N-01152	NCDPW	GLN CV	G4	130	125	405103	733753	GC05	154	0	1	TOB	OB
N-01160	NCDPW	GDN CTY	G12	58	53	404342	733626	NC03	91	0	1	TOH	OB
N-01168	NCDPW	FRPT	G20	28	23	403850	733509	FS06	14	0	1	TOH	OB
N-01176	NCDPW	O WSTBY	H7	198	193	404736	733530	NC03	195	0	2	TOB	OB
N-01177	NCDPW	O WSTBY	H8	146	141	404638	733513	NC03	183	0	2	TNH	AB
N-01185	NCDPW	MERRICK	H16	18	16	403958	733343	NC03	21	0	1	TOH	OB
N-01190	NCDPW	UPBRKVL	U4	99	94	405142	733406	NC04	128	0	1	TOB	OB

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-01194	NCDPW	JERICHO	08	100	95	404657	733322	NC03	168	0	1	TOB	OB
N-01195	NCDPW	HKSVL	09	116	111	404615	733304	NC03	148	0	2	TOB	OB
N-01197	NCDPW	HKSVL	011	69	64	404454	733239	NC03	116	0	1	TOB	OB
N-01204	NCDPW	BELMR	018	29	24	404014	733128	NC03	21	0	1	TOH	OB
N-01205	NCDPW	BELMR	019	29	24	403934	733119	NC03	8	0	1	TOH	OB
N-01209	NCDPW	E NORW	P4	64	59	405153	733216	NC04	122	0	1	TOB	AB
N-01219	NCDPW	NO WTGH	P14	32	27	404219	732934	NC03	55	0	1	TOH	AB
N-01222	NCDPW	SEAFORD	P17	28	23	404026	732904	NC03	20	0	1	TOH	AB
N-01231	NCDPW	PLNVIEW	T-8	81	76	------	------	NC03	143	0	2	TOB	OB
N-01232	NCDPW	BTHPG	T9	57	52	404542	732827	NC03	111	0	1	TOB	OB
N-01233	NCDPW	BTHPG	T-10A	40	35	------	------	NC03	95	0	1	TOB	OB
N-01236	NCDPW	NO MSPQ	T13	52	47	404301	732754	NC03	70	0	1	TOB	OB
N-01246	NCDPW	PLNVIEW	U5	125	120	404701	732652	NC03	185	0	2	TOB	OB
N-01298	GREAT NECK NORTH W A	G N EST	6	336	286	404659	734447	GN11	10	1200	3	TNH	SS
N-01300	LINKS G C	NO HLS	1	375	305	404553	734021	NC02	175	500	2	TNH	YR
N-01328	MANHASSET-LAKEVL W D	NO HLS	21	742	652	404713	734105	NC04	190	1500	3	TNH	YR
N-01346	LONG ISLAND W C	WOMR	5	160	---	403854	734239	NC02	10	9700	4	TOH	YR
N-01402	LONG ISLAND W C	BALDWIN	4(1-15)	35	---	403953	733617	NC02	20	1040	1	TOH	NU
N-01443	NCDPW	SO HEMP	X34	24	19	403950	733612	NC02	18	0	1	TOH	AB
N-01451	NCDPW	GDN CTY	X-43	35	30	404356	733711	NC02	84	0	--	TOH	OB
N-01495	LONG ISLAND W C	BALDWIN	B15	35	---	403953	733617	NC02	20	---	1	TOH	AB
N-01595C	SEA CLIFF W S CO	SEA CLF	--	---	---	------	------	NC04	---	---	--	TOB	NU
N-01601	LONG ISLAND W C	RSVLT	1-13	600	530	404048	733547	NC02	40	1200	2	TOH	YR
N-01602	LONG ISLAND W C	LAKVW	2-1	486	446	404031	733925	NC02	25	1200	2	TOH	SS
N-01603	LONG ISLAND W C	W HEMP	3-1	529	484	404115	733933	NC02	40	1200	2	TOH	YR
N-01618	MANHASSET-LAKEVL W D	MANHST	22	550	470	404631	734213	NC04	175	1500	3	TNH	YR
N-01631	EISENHOWER PARK	E MEAD	4	43	36	404351	733443	NC03	84	250	1	TOH	AB
N-01651	LOCUST VALLEY W D	LOC VLY	6	465	385	405232	733642	NC04	130	800	3	TOB	SS
N-01658	GRUMMAN	BTHPG	1	107	71	404448	732952	NC03	109	700	2	TOB	AB
N-01665	GRUMMAN	BTHPG	2	101	68	404454	732954	NC03	120	1000	2	TOB	AB
N-01666	GRUMMAN	BTHPG	3	108	81	404441	732952	NC03	122	1000	2	TOB	AB
N-01667	WESTBURY W D	WSTBY	7	237	207	404526	733534	NC03	110	1050	2	TNH	AB
N-01696	MITCHEL FIELD W D	MITCH F	5	468	438	404405	733527	NC03	80	800	2	TOH	AB
N-01697	GARDEN CITY VILLAGE	GDN CTY	8	518	478	404359	733832	NC02	88	1120	2	TOH	YR
N-01715	PORT WASHINGTON W D	PT WASH	N1	480	430	404906	734109	PW08	110	510	3	TNH	YR
N-01716	PORT WASHINGTON W D	PT WASH	N2	475	425	404909	734109	PW08	110	684	3	TNH	YR
N-01773	SUNY-OLD WESTBURY	O WSTBY	1	293	281	404703	733449	NC04	228	150	2	TNH	NU
N-01788	VILLAGE BATH	STHMORE	1	320	265	404039	734743	NC04	230	260	2	TNH	YR
N-01802	MANHASSET-LAKEVL W D	NO NHP	7	691	641	404511	734210	NC02	140	1050	3	TNH	SS
N-01804	SPERRY	LK SUCC	3	250	220	404530	734150	NC02	120	1300	2	TNH	YR
N-01840	LIBERTY INDUST PARK	S FMGDL	2	78	65	404337	732649	NC03	64	350	1	TOB	SS
N-01859	GRUMMAN	BTHPG	11	165	135	404516	732935	NC03	126	800	2	TOB	AB
N-01870	ROSLYN W D	ROSLYN	1-82	260	---	404808	733910	RS10	40	1499	2	TNH	YR
N-01922	GRUMMAN	BTHPG	14	187	157	404513	732956	NC03	125	800	2	TOB	AB
N-01923	GRUMMAN	BTHPG	4	359	293	404441	732952	NC03	114	1200	2	TOB	YR
N-01927	LONG BEACH CITY	LNG BCH	8	1219	1159	403534	733955	LB07	10	700	3	TOH	AB
N-01937	FARMINGDALE VILLAGE	FRMGDL	2-1	146	121	404409	732713	NC03	120	800	2	TOB	YR
N-01958	JAMAICA W S CO	NO NHP	16A	727	667	404426	734150	NC02	115	1115	3	TOH	YR
N-01965	LIBERTY INDUST PARK	S FMGDL	3	172	146	404332	732655	NC03	61	250	2	TOB	NU
N-02028	MANHASSET-LAKEVL W D	NO HLS	5T	485	425	404725	734007	NC04	260	1450	2	TNH	YR
N-02030	PORT WASHINGTON W D	PT WASH	N3	215	190	404907	734109	PW08	110	510	1	TNH	YR
N-02048	DAURENHEIM CORP	WANTAGH	2	60	44	404144	733016	NC03	42	400	1	TOH	AB
N-02052	PORT WASHINGTON W D	FLWR HL	H4	325	275	404829	733953	PW08	100	1200	2	TNH	YR
N-02060	GLEN CV BOTTLING	GLN CV	1	82	60	405142	733805	GC05	26	98	1	TOB	NU

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-02072	HICKSVILLE W D	HKSVL	1-3	159	138	404639	733111	NC03	165	600	2	TOB	AB
N-02087	COLUMBIA RIBBON	GLN CV	1	345	334	405146	733813	GC05	50	200	3	TOB	AB
N-02112	DOUBLEDAY INC	GDN CTY	2	61	45	404319	733753	NC02	82	300	1	TOH	YR
N-02115	JAMAICA W S CO	NU V S	25	84	64	404109	734329	NC02	40	1200	1	TOH	AB
N-02169	FRESH MEADOWS C C	LK SUCC	1	239	221	404651	734215	NC04	58	600	2	TNH	AB
N-02214	GREAT NECK NORTH W A	GT NK	7	286	236	404826	734510	VG12	50	1300	3	TNH	NU
N-02236	WESTBURY W D	NEW CSL	8	565	520	404517	733436	NC03	110	750	2	TNH	AB
N-02239	WEST HEMPSTEAD W D	W HEMP	3	178	138	404138	733842	NC02	60	1200	2	TOH	YR
N-02240	CATAPANO NURSERY	HKSVL	1	89	73	404437	732954	NC03	105	250	1	TOB	YR
N-02316	PALL CORP	GLN CV	1	170	---	405106	733725	GC05	60	250	2	TOB	YR
N-02400	ROSLYN W D	E HILLS	2	439	399	404708	733805	RS10	165	1000	2	TNH	YR
N-02402	LEVITTOWN W D	LEVITT	1	206	164	404400	733148	NC03	100	800	2	TOH	AB
N-02403	LEVITTOWN W D	LEVITT	2	84	59	404000	733148	NC03	100	800	1	TOH	AB
N-02409	COLD SPRING HATCHERY	LRL HLW	5	86	66	405125	732805	NC04	15	0	1	TOB	SS
N-02413	JAMAICA W S CO	ELMONT	28A	508	478	404125	734209	NC02	55	900	2	TOH	SS
N-02414	JAMAICA W S CO	ELMONT	28	89	69	404125	734209	NC02	55	1200	1	TOH	SS
N-02487	WILLISTON PK VILL	WIL PK	3	338	298	404547	733901	NC02	125	1000	2	TNH	NU
N-02565	GARDEN CITY PARK W D	G C PK	3	405	370	404434	733944	NC02	100	1000	2	TNH	NU
N-02578	LONG ISLAND W C	V S	7-2A	---	---	404036	734314	NC02	30	1200	1	TOH	YR
N-02578A	LONG ISLAND W C	V S	7-2	88	56	404036	734314	NC02	30	1200	1	TOH	AB
N-02580	LEVITTOWN W D	LEVITT	3	357	321	404325	733145	NC03	85	800	2	TOH	YR
N-02581	LEVITTOWN W D	LEVITT	4	80	54	404325	733145	NC03	85	800	1	TOH	AB
N-02597	LONG BEACH CITY	LNG BCH	9	1235	1175	403529	734033	LB07	5	1200	3	TOH	YR
N-02602	WESTBURY W D	NEW CSL	9	800	760	404519	733427	NC03	110	950	3	TNH	SS
N-02603	N Y WATER SERVICE	MSPQ E	3	66	34	404058	732605	NC03	25	700	1	TOB	AB
N-02613	LONG ISLAND W C	BALDWIN	4-16	500	460	403953	733617	NC02	20	1215	2	TOH	YR
N-02616	GLEN HEAD C C	SEA CLF	1	232	184	405052	733708	NC04	75	500	1	TOB	YR
N-02635	NCDPW	PT WASH	X76	154	150	404943	734152	PW08	42	0	2	TNH	OB
N-02747	CARLE PLACE W D	CAR PL	1	328	278	404446	733650	NC03	110	1000	2	TNH	NU
N-02748	CARLE PLACE W D	CAR PL	2	510	460	404446	733650	NC03	110	1200	2	TNH	YR
N-02748A	CARLE PLACE W D	CAR PL	2	326	276	404446	733650	NC03	110	1000	2	TNH	AB
N-02847	NO STAGE THEATER	GLN CV	1	116	82	405209	733751	GC05	80	425	1	TOB	YR
N-02920	SWAN COVE W S	COV NK	1	515	505	405252	732928	NC04	20	100	3	TOB	YR
N-02923	EISENHOWER PARK	E MEAD	1	122	80	404403	733350	NC03	96	750	2	TOH	SS
N-03081	ROCKVILLE LINKS	RVC	1	60	44	404029	733744	NC02	38	310	1	TOH	SS
N-03129	EISENHOWER PARK	E MEAD	2	142	97	404356	733351	NC03	95	1000	2	TOH	NU
N-03142	BETHPAGE W D	BTHPG	4	163	122	404547	732849	NC03	137	700	2	TOB	AB
N-03147	BETHPAGE W D	BTHPG	5	233	192	404547	732849	NC03	137	500	2	TOB	AB
N-03185	MINEOLA VILLAGE	MINEOLA	4	463	423	404418	733847	NC02	100	1050	2	TNH	YR
N-03193	LEVITTOWN W D	LEVITT	5	316	274	404337	733046	NC03	90	1000	2	TOH	AB
N-03194	LEVITTOWN W D	LEVITT	6	256	219	404337	733046	NC03	90	1000	2	TOH	AB
N-03243	EISENHOWER PARK	E MEAD	3	303	248	404345	733356	NC03	89	1000	2	TOH	YR
N-03312	LEVITTOWN W D	LEVITT	8	304	252	404309	733026	NC03	75	800	2	TOH	AB
N-03313	LEVITTOWN W D	LEVITT	7	95	64	404309	733026	NC03	75	1000	2	TOH	AB
N-03327	LONG ISLAND W C	V S	7-1	450	398	404036	734314	NC02	30	1400	2	TOH	AB
N-03427	N Y WATER SERVICE	WANTAGH	10	161	126	404052	732948	NC03	20	700	2	TOH	AB
N-03443	GREAT NECK NORTH W A	GT NK	8	464	424	404818	734346	VG12	115	1200	3	TNH	YR
N-03448	LONG BEACH CITY	LNG BCH	7	1238	1180	403520	734153	LB07	10	1400	3	TOH	AB
N-03456	EAST MEADOW W D	E MEAD	1	555	494	404310	733316	NC03	85	1500	2	TOH	YR
N-03456A	EAST MEADOW W D	E MEAD	1	320	280	404310	733316	NC03	85	910	2	TOH	AB
N-03457	EAST MEADOW W D	E MEAD	2	320	280	404310	733316	NC03	85	1050	2	TOH	YR
N-03463	N Y WATER SERVICE	NO WTGH	10	299	247	404130	733114	NC03	40	1400	2	TOH	AB
N-03465	EAST MEADOW W D	E MEAD	3	580	519	404310	733316	NC03	85	1500	2	TOH	YR
N-03465A	EAST MEADOW W D	E MEAD	3	297	268	404310	733316	NC03	85	810	2	TOH	AB

NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-03466	GLEN COVE CITY	GLN CV	20	173	148	405116	733729	GC05	160	485	2	TOB	AB
N-03474	JERICHO W D	BKVL	6	512	452	404846	733440	NC04	195	1200	2	TOB	YR
N-03475	JERICHO W D	BKVL	7	482	432	404849	733445	NC04	195	1200	2	TOB	YR
N-03486	OYSTER BAY W D	O BAY	6	102	70	405231	733230	OB14	20	500	1	TOB	YR
N-03488	HICKSVILLE W D	HKSVL	3-1	169	116	404446	733057	NC03	115	1200	2	TOB	AB
N-03498	TOBAY BEACH	WANTAGH	1	335	315	403632	732552	NC03	10	125	2	TOB	YR
N-03520	LONG ISLAND W C	W HEMP	3-2	178	126	404115	733933	NC02	40	800	2	TOH	SS
N-03523	MANHASSET-LAKEVL W D	MNSY PK	8	320	290	404808	734112	NC04	200	950	2	TNH	YR
N-03540	PLANDOME VILLAGE	PLANOME	3	207	155	404826	734148	NC04	40	650	1	TNH	YR
N-03552	HICKSVILLE W D	HKSVL	4-1	169	116	404455	733203	NC03	120	1200	2	TOB	AB
N-03553	HICKSVILLE W D	HKSVL	5-1	152	99	404455	733249	NC03	115	1200	2	TOB	AB
N-03561	OYSTER BAY W D	O BAY	7	118	88	405231	733230	OB14	20	800	1	TOB	YR
N-03562	MARION ST SCH	E RCKWY	1	55	40	403848	734034	NC02	22	100	1	TOH	SS
N-03564	N Y WATER SERVICE	WANTAGH	2	69	28	404052	732948	NC03	20	1000	1	TOH	AB
N-03569	CERRO WIRE	LOC GRV	1	402	353	404756	733054	NC03	181	1000	2	TOB	YR
N-03603	FRANKLIN SQUARE W D	FR SQ	1	493	443	404247	734023	NC02	65	1000	2	TOH	SS
N-03604	FRANKLIN SQUARE W D	FR SQ	2	498	438	404247	734023	NC02	65	1000	2	TOH	SS
N-03605	FRANKLIN SQUARE W D	FR SQ	3	438	398	404152	734103	NC02	60	1200	2	TOH	SS
N-03618	LEVITTOWN W D	LEVITT	6A	418	317	404337	733039	NC03	90	1000	2	TOH	YR
N-03668	HEMPSTEAD VILLAGE	HEMPSTD	7	500	450	404149	733731	NC02	50	1200	2	TOH	YR
N-03672	GARDEN CITY PARK W D	G C PK	4	447	407	404459	734021	NC02	100	1000	2	TNH	NU
N-03673	GARDEN CITY PARK W D	G C PK	5	429	389	404459	734021	NC02	100	1000	2	TNH	NU
N-03680	N Y WATER SERVICE	NO WTGH	2J	328	270	404130	733114	NC03	40	1400	2	TOH	AB
N-03687	LONG BEACH CITY	LNG BCH	10	1245	1195	403534	733955	LB07	5	1200	3	TOH	YR
N-03695	MITCHEL FIELD W D	MITCH F	1	500	470	404350	733512	NC03	80	350	2	TOH	AB
N-03697	MITCHEL FIELD W D	MITCH F	3	432	402	404350	733612	NC03	80	350	2	TOH	AB
N-03698	MITCHEL FIELD W D	MITCH F	4	472	442	404350	733612	NC03	80	350	2	TOH	AB
N-03699	CONSOL LITHO	CAR PL	1	89	67	404451	733652	NC02	107	400	2	TNH	SS
N-03700	CONSOL LITHO	CAR PL	2	73	51	404451	733652	NC02	107	400	1	TNH	YR
N-03704	WEST HEMPSTEAD W D	W HEMP	4	159	106	404138	733840	NC02	60	1200	2	TOH	YR
N-03720	JAMAICA W S CO	NO V S	30	516	476	404113	734039	NC02	40	1200	2	TOH	YR
N-03722	LONG ISLAND W C	RSVLT	1-15	81	55	404048	733547	NC02	40	750	1	TOH	SS
N-03732	ALBERTSON W D	ROS HTS	1	350	310	404621	733829	NC02	145	1000	2	TNH	YR
N-03733	ALBERTSON W D	ROS HTS	2	450	410	404626	733829	NC02	145	1000	2	TNH	YR
N-03742	NO HEMPSTEAD C C	PT WASH	1	260	230	404842	734044	PW08	140	600	1	TNH	NU
N-03745	ROCKVILLE CTR VILL	RVC	6	592	542	404109	733749	NC02	40	1200	2	TOH	YR
N-03780	N Y WATER SERVICE	NO WTGH	1S	142	89	404228	732933	NC03	60	1400	2	TOH	AB
N-03781	LONG ISLAND W C	V S	9-2	430	370	404031	734144	NC02	25	1600	2	TOH	AB
N-03782	LONG ISLAND W C	V S	9-1	408	347	404031	734144	NC02	25	1400	2	TOH	AB
N-03832	LONG ISLAND W C	RSVLT	1-16	95	65	404048	733547	NC02	40	750	1	TOH	YR
N-03859	MERRICK-STEWART RLTY	E MEAD	1	279	259	404403	733457	NC03	77	150	2	TOH	YR
N-03876	BETHPAGE W D	PLNEDGE	6-1	386	328	404354	732912	NC03	90	1400	2	TOB	NU
N-03878	HICKSVILLE W D	HKSVL	6-2	428	375	404626	733231	NC03	145	1200	2	TOB	SS
N-03881	GARDEN CITY VILLAGE	GDN CTY	9	466	426	404320	734021	NC02	85	1100	2	TOH	YR
N-03886	N Y WATER SERVICE	MSPQ E	4	75	42	404058	732605	NC03	25	1400	1	TOB	AB
N-03892	GLEN COVE CITY	GLN CV	1S	246	138	405230	733721	GC05	130	700	1	TOB	NU
N-03893	N Y WATER SERVICE	NO WTGH	2S	151	98	404228	732933	NC03	60	1400	2	TOH	SS
N-03894	N Y WATER SERVICE	NO MRK	1C	362	312	404101	733419	NC03	30	1400	2	TOH	AB
N-03895	N Y WATER SERVICE	NO BLMR	1N	349	312	404117	733230	NC03	40	2100	2	TOH	SS
N-03905	MANHASSET-LAKEVL W D	LK SUCC	12	254	214	404544	734151	BG09	150	1050	1	TNH	YR
N-03912	PRATT RESIDENCE	SNDS PT	1	104	101	405044	734055	NC04	165	18	1	TNH	YR
N-03926	WOODMERE CLUB	WDSBRGH	1	67	54	404303	733721	NC02	17	350	1	TOH	YR
N-03934	GARDEN CITY VILLAGE	GDN CTY	10	417	377	404406	733707	NC02	90	1400	2	TOH	YR
N-03935	GARDEN CITY VILLAGE	GDN CTY	11	410	370	404406	733707	NC02	90	1400	2	TOH	YR

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-03937	LONG ISLAND W C	MLVRN	8-1	462	389	404003	734019	NC02	25	1400	2	TOH	YR
N-03953	HICKSVILLE W D	HKSVL	6-1	419	169	404626	733231	NC03	145	1200	2	TOB	YR
N-03993	HENRYS LUNCH	GLN CV	1	70	65	405153	733759	GC05	40	20	2	TOB	YR
N-04009	DAURENHEIM CORP	WANTAGH	1	50	35	404144	733015	NC03	42	250	1	TOH	AB
N-04010	LIBERTY INDUST PARK	S FMGDL	4	268	238	404339	732658	NC03	64	500	2	TOB	YR
N-04042	SO FARMINGDALE W D	PLNEDGE	1-1	154	96	404307	732745	NC03	60	1100	2	TOB	AB
N-04043	SO FARMINGDALE W D	PLNEDGE	1-2	374	322	404307	732751	NC03	60	1200	2	TOB	YR
N-04063	BETHPAGE W D	BTHPG	7	233	145	404532	732848	NC03	125	1400	2	TOB	AB
N-04077	JAMAICA W S CO	FL PK	35	90	70	404324	734142	NC02	85	1250	1	TOH	SS
N-04082	MINEOLA VILLAGE	MINEOLA	5	462	422	404523	733732	NC02	110	1020	2	TNH	YR
N-04095	PLAINVIEW W D	PLNVIEW	1-1	490	440	404639	732802	NC03	170	1200	2	TOB	YR
N-04096	PLAINVIEW W D	PLNVIEW	1-2	494	444	404639	732802	NC03	170	1200	2	TOB	YR
N-04097	PLAINVIEW W D	PLNVIEW	3-1	465	413	404631	732939	NC03	175	1200	2	TOB	YR
N-04118	WEST HEMPSTEAD W D	W HEMP	5	204	146	404129	733844	NC02	50	1200	2	TOH	YR
N-04132	LONG ISLAND W C	BALDWIN	12-1	619	549	403941	733645	NC02	40	1400	2	TOH	YR
N-04133	JERICHO W D	LOC GRV	8	450	400	404810	733036	NC04	195	1200	2	TOB	AB
N-04146	BETHPAGE W D	BTHPG	8	235	153	404533	732848	NC03	125	1400	2	TOB	AB
N-04173	SPERRY	LK SUCC	2	255	225	404528	734159	NC02	120	1000	2	TNH	YR
N-04206	CARLE PLACE W D	CAR PL	3	355	305	404525	733632	NC03	110	1000	2	TNH	YR
N-04215	ROSLYN C C	ROS HTS	1	104	87	404638	733753	NC02	132	150	1	TNH	AB
N-04223	PORT WASHINGTON W D	FLWR HL	S5	326	273	404856	734033	PW08	190	700	1	TNH	YR
N-04243	MANHASSET-LAKEVL W D	LK SUCC	4T	255	205	404543	734151	BG09	150	1050	1	TNH	SS
N-04245	JERICHO W D	JERICHO	9	565	525	404736	733213	NC03	220	1200	2	TOB	YR
N-04246	JERICHO W D	JERICHO	10	453	403	404802	733130	NC04	195	1200	2	TOB	OB
N-04265	ROSLYN W D	E HILLS	3	485	435	404757	733722	RS10	200	1200	2	TNH	YR
N-04298	JAMAICA W S CO	FL PK	35A	394	344	404324	734142	NC02	85	1250	2	TOH	YR
N-04298A	JAMAICA W S CO	FL PK	35A	384	349	404324	734142	NC02	85	1250	2	TOH	AB
N-04310	WAVERLY PK SCH	E RCKWY	1	64	54	403813	734035	NC02	10	68	1	TOH	SS
N-04327	ALBERTSON W D	SRNGTWN	3	425	365	404621	733923	NC02	140	1000	2	TNH	YR
N-04329	MASSAPEQUA H S	MSPQ PK	1	50	30	404013	732715	NC03	13	160	1	TOB	SS
N-04334	E J KORVETTES	W HEMP	1	161	89	404217	733830	NC02	65	800	2	TOH	YR
N-04388	GREAT NECK NORTH W A	MANHST	9	145	125	404650	734403	NC04	25	1250	2	TNH	YR
N-04389	SANDS POINT VILL	SNDS PT	3	225	150	405100	734058	NC04	120	500	2	TNH	YR
N-04390	JAMAICA W S CO	NO NHP	40	296	261	404513	734124	NC02	120	1400	2	TOH	YR
N-04390A	JAMAICA W S CO	NO NHP	40	105	84	404513	734124	NC02	120	1400	1	TOH	AB
N-04393	LONG ISLAND W C	V S	10-1	472	419	403932	734124	NC02	25	1400	2	TOH	YR
N-04394	LONG ISLAND W C	MLVRN	8-2	180	132	404003	734019	NC02	25	1400	2	TOH	OC
N-04400	OYSTER BAY W D	OBAY CO	PLT2	302	214	405157	733003	OB14	50	1200	2	TOB	YR
N-04405	LONG ISLAND W C	ATL BCH	6-1	1075	1005	403515	734310	LB07	10	1390	3	TOH	SS
N-04410	H O PENN	G C EST	1	115	99	404416	733507	NC03	88	200	2	TOH	AB
N-04411	LONG ISLAND W C	LYNBRK	14-1	550	480	403921	734044	NC02	20	1390	2	TOH	SS
N-04425	HEMPSTEAD VILLAGE	HEMPSTD	1R	365	325	404300	733712	NC02	60	1200	2	TOH	YR
N-04447	EAST MEADOW W D	E MEAD	4	330	280	404310	733316	NC03	85	1200	2	TOH	YR
N-04448	EAST MEADOW W D	E MEAD	5	550	500	404310	733316	NC03	85	1200	2	TOH	YR
N-04450	LEVITTOWN W D	LEVITT	9	466	415	404325	733135	NC03	85	1200	2	TOH	YR
N-04451	LEVITTOWN W D	LEVITT	10	403	353	404429	733053	NC03	110	1200	2	TOH	SS
N-04461	N Y WATER SERVICE	WANTAGH	30	174	107	404052	732948	NC03	20	1700	2	TOH	AB
N-04462	NORTH SHORE C C	GLN HD	1	181	149	405003	733844	NC04	69	500	2	TOB	YR
N-04512	JAMAICA W S CO	NO V S	34	504	454	404102	734111	NC02	40	1700	2	TOH	YR
N-04602	MASSAPEQUA W D	MSPQ PK	1	444	382	404154	732618	NC03	40	1400	2	TOB	YR
N-04603	MASSAPEQUA W D	MSPQ PK	2	184	113	404154	732618	NC03	40	1400	2	TOB	AB
N-04623	ROSLYN W D	ROS EST	4	498	440	404721	733951	RS10	260	1200	2	TNH	YR
N-04633	MEADOWBROOK CLUB	JERICHO	1	216	163	404723	733323	NC03	176	1000	2	TOB	SS
N-04708	GRUMMAN	BTHPG	12	169	149	404524	732956	NC03	122	400	2	TOB	AB

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-04756	UNIONDALE W D	UNDALE	1	313	245	404206	733448	NC02	55	1100	2	TOH	YR
N-04756A	UNIONDALE W D	UNDALE	1	307	245	404206	733448	NC02	55	1100	2	TOH	AB
N-04757	UNIONDALE W D	UNDALE	2	628	529	404206	733448	NC02	55	1200	2	TOH	YR
N-04757A	UNIONDALE W D	UNDALE	2	319	267	404206	733448	NC02	55	1100	2	TOH	AB
N-04758	UNIONDALE W D	UNDALE	3	625	550	404206	733448	NC02	55	1200	2	TOH	YR
N-04758A	UNIONDALE W D	UNDALE	3	441	363	404206	733448	NC02	55	1100	2	TOH	AB
N-04759	UNIONDALE W D	UNDALE	4	625	539	404206	733448	NC02	55	1200	2	TOH	YR
N-04759A	UNIONDALE W D	UNDALE	4	355	283	404206	733448	NC02	55	1100	2	TOH	AB
N-04760	PINE HOLLOW C C	E NORW	1	247	215	405108	733120	NC04	220	500	1	TOB	NU
N-04772	GREAT NECK SAW CO	MINEOLA	1	232	192	404440	733737	NC02	110	45	2	TNH	YR
N-04859	PORT WASHINGTON W D	BAX EST	SH3	385	355	405008	734147	PW08	20	350	7	TNH	NU
N-04860	PORT WASHINGTON W D	BAX EST	SH1	89	60	405010	734147	PW08	20	550	1	TNH	YR
N-05007	WESTBURY W D	WSTBY	10	494	428	404552	733420	NC03	120	1350	2	TNH	YR
N-05071	NASSAU G C	GLN CV	1	242	210	405218	733625	GC05	143	500	2	TOB	YR
N-05099	MANHASSET-LAKEVL W D	THOMSTN	1	393	345	404646	734235	GN11	175	1050	2	TNH	SS
N-05121	LONG ISLAND W C	LYNBRK	15-1	542	481	403956	734104	NC02	20	1390	2	TOH	YR
N-05129	JONES BEACH ST PK	WANTAGH	3	965	895	403606	733030	JB13	9	1200	2	TOH	YR
N-05145	LONG ISLAND W C	V S	7-3	460	405	404036	734314	NC02	30	1400	2	TOH	YR
N-05147	SO FARMINGDALE W D	MSPQ E	2-1	219	165	404214	732622	NC03	40	1200	2	TOB	YR
N-05148	SO FARMINGDALE W D	PLNEDGE	1-3	369	298	404307	732747	NC03	60	1200	2	TOB	YR
N-05152	LOCUST VALLEY W D	LATNGTN	7	355	305	405328	733514	NC04	45	1100	3	TOB	YR
N-05153	LONG ISLAND W C	BALDWIN	12-2	323	268	403941	733645	NC02	26	1390	2	TOH	SS
N-05155	JAMAICA W S CO	ELMONT	44	90	70	404239	734202	NC02	75	1200	1	TOH	SS
N-05156	JAMAICA W S CO	ELMONT	44A	331	291	404239	734202	NC02	75	1400	2	TOH	YR
N-05163	GARDEN CITY VILLAGE	GDN CTY	12	475	425	404359	733902	NC02	85	1400	2	TOH	YR
N-05187	LONG ISLAND W C	RSVLT	16-1	495	445	404039	733439	NC02	35	1400	2	TOH	YR
N-05193	ROCKVILLE CTR VILL	RVC	7	550	490	403931	733811	NC02	20	1200	2	TOH	YR
N-05194	ROCKVILLE CTR VILL	RVC	8	515	455	403924	733919	NC02	15	1200	2	TOH	YR
N-05195	ROCKVILLE CTR VILL	RVC	9	505	444	403924	733919	NC02	15	1200	2	TOH	YR
N-05195A	ROCKVILLE CTR VILL	RVC	9	335	305	403924	733919	NC02	15	1200	2	TOH	AB
N-05201	JERICHO W D	ROS HBR	11	504	434	404928	733820	NC04	48	1200	3	TOB	SS
N-05209	PORT WASHINGTON W D	PT WASH	B6	300	260	404939	734030	PW08	200	900	2	TNH	YR
N-05227	LIDO-PT LOOKOUT W D	LID BCH	2	1260	1200	403532	733534	LB07	5	1200	3	TOH	YR
N-05259	N Y WATER SERVICE	NO BLMR	2N	308	241	404117	733230	NC03	40	2100	2	TOH	AB
N-05260	WEST HEMPSTEAD W D	W HEMP	6	514	452	404142	733840	NC02	60	1200	2	TOH	YR
N-05261	GLEN COVE CITY	GLN CV	2S	230	129	405230	733721	GC05	130	1400	1	TOB	YR
N-05301	LEVITTOWN W D	LEVITT	11	377	324	404428	733152	NC03	105	1200	2	TOH	NU
N-05302	LEVITTOWN W D	LEVITT	12	484	431	404247	733145	NC03	65	1200	2	TOH	YR
N-05303	LEVITTOWN W D	NO WTGH	13	506	454	404257	733006	NC03	65	1200	2	TOH	AB
N-05303A	LEVITTOWN W D	NO WTGH	13	512	454	404257	733006	NC03	65	1200	2	TOH	SS
N-05304	LEVITTOWN W D	LEVITT	14	467	415	404225	733042	NC03	60	1200	2	TOH	NU
N-05305	GRUMMAN	BTHPG	13	167	115	404524	732949	NC03	129	800	2	TOB	AB
N-05306	GRUMMAN	BTHPG	15	256	173	404523	732935	NC03	129	800	2	TOB	AB
N-05308	LONG BEACH CITY	LNG BCH	11	1220	1160	403518	733827	LB07	10	1250	3	TOH	NU
N-05318	EAST MEADOW W D	UNDALE	6	633	581	404159	733450	NC03	55	1200	2	TOH	YR
N-05318A	EAST MEADOW W D	UNDALE	6	310	260	404159	733450	NC03	55	1200	2	TOH	AB
N-05319	EAST MEADOW W D	UNDALE	7	438	388	404159	733450	NC03	55	1200	2	TOH	NU
N-05320	EAST MEADOW W D	UNDALE	8	643	593	404159	733450	NC03	55	1200	2	TOH	YR
N-05320A	EAST MEADOW W D	UNDALE	8	379	319	404159	733450	NC03	55	1200	2	TOH	AB
N-05321	EAST MEADOW W D	E MEAD	9	509	449	404243	733158	NC03	65	1200	2	TOH	YR
N-05322	EAST MEADOW W D	E MEAD	10	510	470	404243	733201	NC03	65	1200	2	TOH	YR
N-05334	INWOOD C C	INWOOD	1	117	21	403712	734503	NC01	18	500	6	TOH	YR
N-05336	HICKSVILLE W D	HKSVL	2-2	523	472	404441	733209	NC03	120	1200	2	TOB	YR
N-05441	SALISBURY C C	STW MNR	1	86	76	404320	734106	NC02	84	50	1	TOH	SS

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-05450	ENGINEERS C C	ROS HBR	1	80	58	404908	733812	NC04	57	500	1	TNH	YR
N-05457	HEMPSTEAD GEN HOSP	HEMPSTD	1	52	46	404234	733609	NC02	70	40	1	TOH	YR
N-05484	ROOSEVELT FIELD W D	G C EST	1	575	503	404420	733643	NC02	90	1400	2	TOH	YR
N-05484A	ROOSEVELT FIELD W D	G C EST	1	287	101	404420	733643	NC02	90	1200	2	TOH	AB
N-05485	ROOSEVELT FIELD W D	G C EST	2	557	475	404424	733653	NC02	92	1400	2	TOH	YR
N-05485A	ROOSEVELT FIELD W D	G C EST	2	278	92	404424	733653	NC02	92	1500	2	TOH	AB
N-05486	ROOSEVELT FIELD W D	G C EST	3	556	450	404415	733653	NC02	87	1400	2	TOH	AB
N-05486A	ROOSEVELT FIELD W D	G C EST	3	263	92	404415	733653	NC02	87	1200	2	TOH	AB
N-05507	MACY'S	G C EST	1	330	107	404415	733658	NC03	92	1200	2	TOH	SS
N-05528	MANHASSET-LAKEVL W D	NO HLS	6T	490	430	404730	734007	NC04	260	1050	2	TNH	YR
N-05535	DEEPDALE C C	NO HLS	1	390	330	404620	734141	NC02	250	500	2	TNH	YR
N-05589	HEWLETT H S	HEWLETT	1	78	69	403822	734148	NC02	20	44	1	TOH	SS
N-05596	MINEOLA VILLAGE	MINEOLA	6	468	403	404453	733725	NC02	100	1200	2	TNH	YR
N-05603	GARDEN CITY PARK W D	HERRCKS	6	415	365	404517	734023	NC02	110	1200	2	TNH	NU
N-05653	LONG ISLAND W C	SO HEMP	18-1	581	529	404112	733716	NC02	50	1400	2	TOH	YR
N-05654	WESTBURY W D	WSTBY	11	538	275	404453	733526	NC03	100	1350	2	TNH	YR
N-05654A	WESTBURY W D	WSTBY	11	335	275	404453	733526	NC03	100	1050	2	TNH	AB
N-05655	WESTBURY W D	NEW CSL	12	255	205	404543	733336	NC03	130	1050	2	TNH	YR
N-05656	LONG ISLAND W C	LAKVW	17-1	495	445	403948	733929	NC02	20	1390	2	TOH	YR
N-05663	HIP OF GTR NY	HKSVL	1	99	89	404540	733100	NC03	140	160	1	TOB	YR
N-05672	BEAVER DAM CLUB	LOC VLY	1	121	101	405259	733413	NC04	25	250	1	TOB	YR
N-05695	FREEPORT VILLAGE	FRPT	7	526	466	403923	733543	FS06	20	1500	2	TOH	YR
N-05696	FREEPORT VILLAGE	FRPT	8	518	468	403952	733422	FS06	20	1500	2	TOH	YR
N-05703	MASSAPEQUA W D	MSPQ PK	3	459	384	404154	732618	NC03	40	1400	2	TOB	YR
N-05705	TOB INCINERATOR	O BTHPG	2	492	450	404551	732742	NC03	145	500	2	TOB	NU
N-05708	PALL CORP	E HILLS	2	243	212	404824	733806	NC04	210	250	2	TNH	YR
N-05710	MANHASSET-LAKEVL W D	NO HLS	6	385	325	404558	734150	NC02	160	1400	1	TNH	SS
N-05725	NEWSDAY	GDN CTY	1	56	46	404352	733647	NC02	79	250	1	TOH	YR
N-05762	GLEN COVE CITY	GLN CV	R	280	221	405230	733721	GC05	145	1400	2	TOB	YR
N-05767	N Y WATER SERVICE	WANTAGH	4D	384	305	404055	732948	NC03	20	2100	2	TOH	YR
N-05792	SEA CLIFF W S CO	GLN HD	GH	295	255	405016	733735	NC04	120	1050	2	TOB	YR
N-05848	N Y WATER SERVICE	MSPQ E	5M	335	204	404058	732605	NC03	25	1400	2	TOB	AB
N-05851	SUNY-OLD WESTBURY	O WSTBY	3	177	171	404703	733449	NC04	218	8	2	TNH	YR
N-05852	ROSLYN W D	E HILLS	5	482	432	404808	733746	RS10	190	1200	2	TNH	YR
N-05876	PORT WASHINGTON W D	FLWR HL	R7	238	168	404858	734115	PW08	125	500	2	TNH	YR
N-05884	GREAT NECK NORTH W A	GT NK	10	163	92	404758	734259	VG12	20	1250	2	TNH	YR
N-05947	ALBERTSON W D	SRNGTWN	4	365	295	404645	733905	NC02	155	1200	2	TNH	YR
N-05994	GLEN COVE COMM HOSP	GLN CV	1	226	173	405211	733718	GC05	130	1080	1	TOB	YR
N-06003	GREAT NK EST VILL	G N EST	1	94	79	404711	734455	BG09	10	100	1	TNH	SS
N-06018	SUN GLOW CAR WASH	NHP	1	91	85	404518	734119	NC02	---	44	--	TNH	SS
N-06042	MILL NECK EST W S	MILL NK	[illegible]	340	328	405400	733319	NC04	20	60	3	TOB	NU
N-06045	EUR-AMER BANK	G C EST	6	328	277	404433	733656	NC03	97	870	2	TOH	SS
N-06046	ROOSEVELT FIELD W D	G C EST	4	175	145	404415	733551	NC02	101	570	2	TOH	NU
N-06076	PLAINVIEW W D	PLNVIEW	4-1	358	296	404651	732913	NC03	160	1200	2	TOB	YR
N-06077	PLAINVIEW W D	PLNVIEW	4-2	460	398	404651	732913	NC03	160	1200	2	TOB	YR
N-06078	BETHPAGE W D	BTHPG	9	275	225	404537	732848	NC03	125	1400	2	TOB	YR
N-06087	PORT WASHINGTON W D	BAX EST	SH2	92	62	405010	734147	PW08	20	500	1	TNH	YR
N-06092	JERICHO W D	WDBRY	12	631	561	404910	732751	NC04	240	1200	2	TOB	YR
N-06093	JERICHO W D	WDBRY	13	606	546	404910	732751	NC04	240	1200	2	TOB	YR
N-06119	ABILITIES INC	SRNGTWN	1	181	156	404611	733929	NC02	123	350	2	TNH	SS
N-06146	LONG ISLAND W C	LAKVW	19-1	498	448	404004	733922	NC02	20	1400	2	TOH	YR
N-06148	SO FARMINGDALE W D	NO MSPQ	4-1	561	462	404215	732732	NC03	45	1200	2	TOB	YR
N-06149	SO FARMINGDALE W D	MSPQ E	2-2	640	585	404214	732622	NC03	40	1200	2	TOB	YR
N-06150	SO FARMINGDALE W D	MSPQ E	3-1	607	545	404246	732903	NC03	60	1400	2	TOB	YR

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-06190	HICKSVILLE W D	HKSVL	7-1	600	550	404706	733052	NC03	175	1200	2	TOB	SS
N-06191	HICKSVILLE W D	HKSVL	7-2	550	489	404706	733052	NC03	175	1200	2	TOB	SS
N-06192	HICKSVILLE W D	HKSVL	8-1	626	575	404521	733102	NC03	135	1400	2	TOB	YR
N-06193	HICKSVILLE W D	HKSVL	8-2	467	396	404521	733102	NC03	135	1400	2	TOB	YR
N-06202	PRO ELECTRONICS	NEW CSL	1	265	237	404550	733304	NC03	132	200	2	TNH	AB
N-06289	PIPING ROCK C C	LOC VLY	1	219	122	405121	733515	NC04	162	500	1	TOB	SS
N-06297	ANZIANO BLDG	MINEOLA	1	92	72	404437	733826	NC02	109	200	1	TNH	AB
N-06302	ROCKVILLE LINKS	RVC	2	61	51	404049	733749	NC02	39	200	1	TOH	YR
N-06315	CARLE PLACE W D	CAR PL	4	348	298	404525	733632	NC03	110	1200	2	TNH	YR
N-06320	CHAMINADE H S	MINEOLA	1	76	67	404443	733859	NC02	100	44	1	TNH	SS
N-06376	NASSAU SANATORIUM	PLNVIEW	--	240	199	404629	732706	NC03	190	200	1	TOB	AB
N-06413	SALK JR H S	LEVITT	1	51	41	404219	733023	NC03	53	150	1	TOH	SS
N-06417	LEVITTOWN DIV H S	LEVITT	1	60	49	404346	733130	NC03	93	105	1	TOH	SS
N-06439	ABBEY LANE SCH	LEVITT	1	59	54	404308	733034	NC03	70	45	1	TOH	SS
N-06442	MASSAPEQUA W D	MASPQA	4	612	524	404123	732850	NC03	40	1400	2	TOB	OC
N-06443	MASSAPEQUA W D	MASPQA	5	268	190	404123	732850	NC03	40	1400	2	TOB	OC
N-06450	LONG BEACH CITY	LNG BCH	12	1275	1215	403533	734010	LB07	5	1250	3	TOH	YR
N-06455	SHELL GAS	V S	1	84	80	403944	734248	NC02	10	12	1	TOH	YR
N-06502	GARDEN CITY G C	GDN CTY	1	90	69	404353	733906	NC02	97	500	1	TOH	SS
N-06521	PLAINEDGE H S	NO MSPQ	1	35	29	404202	732826	NC03	50	45	1	TOB	SS
N-06530	DOUBLEDAY INC	GDN CTY	1	56	45	404319	733753	NC02	81	200	1	TOH	YR
N-06531	METCO INC	HKSVL	1	174	161	404714	733100	NC03	178	40	1	TOB	YR
N-06579	AUGUST THOMSEN CO	GLN CV	1	146	130	405108	733725	GC05	60	44	2	TOB	YR
N-06580	PLAINVIEW W D	PLNVIEW	3-2	596	523	404631	732939	NC03	175	1200	2	TOB	YR
N-06610	LAWRENCE G C	LAWRENC	1	230	199	403641	734332	NC02	9	500	2	TOH	SS
N-06620	NATL METAL PROC	HKSVL	1	87	82	404508	733015	NC03	120	18	2	TOB	YR
N-06644	FARMINGDALE VILLAGE	FRMGDL	2-2	222	175	404409	732713	NC03	120	1400	2	TOB	YR
N-06651	JERICHO W D	JERICHO	14	610	560	404756	733153	NC04	240	1200	2	TOB	YR
N-06657	TOBAY BEACH	WANTAGH	2	294	271	403632	732552	NC03	10	150	2	TOB	YR
N-06692	MAIMONIDES SCH	SNDS PT	1	320	---	405118	734135	NC04	112	60	3	TNH	AB
N-06741	CERRO WIRE	LOC GRV	2	423	373	404755	733053	NC03	191	1000	2	TOB	YR
N-06744	JAMAICA W S CO	ELMONT	44B	94	74	404239	734202	NC02	75	1200	1	TOH	SS
N-06745	JAMAICA W S CO	ELMONT	44C	344	304	404239	734202	NC02	75	1400	2	TOH	YR
N-06768	PLANTING FIELDS ARBR	O BAY	1	175	163	405203	733311	NC03	208	80	1	TOB	NU
N-06769	WOODMERE CLUB	WDSBRGH	2	37	26	404257	733712	NC02	14	350	1	TOH	YR
N-06780	PENINSULA G C	MSPQ PK	1	51	35	403927	732624	NC03	8	250	1	TOB	SS
N-06806	CEDARBROOK G C	O BKVL	1	323	287	405028	733625	NC04	154	650	2	TOB	YR
N-06817	ROCKVILLE CTR VILL	RVC	10	558	508	403931	733811	NC02	20	1200	2	TOH	YR
N-06819	WESTBURY W D	NEW CSL	12A	265	215	404543	733336	NC03	130	1050	2	TNH	YR
N-06841	ISLAND INN	G C EST	1	337	311	404443	733613	NC03	100	252	2	TOH	AB
N-06842	ISLAND INN	G C EST	2	158	143	404443	733613	NC03	100	100	2	TOH	SS
N-06848	W T CLARKE H S	BOWL GR	1	104	93	404445	733326	NC03	105	45	2	TOH	SS
N-06858	MAIMONIDES SCH	SNDS PT	2	356	338	405118	734135	NC04	109	157	3	TNH	AB
N-06866	MASSAPEQUA W D	MASPQA	6	626	566	404043	732836	NC03	20	1400	2	TOB	YR
N-06067	MASSAPEQUA W D	MASPQA	7	492	414	404043	732836	NC03	20	1400	2	TOB	YR
N-06893	LONG ISLAND W C	RSVLT	1-17	560	500	404048	733547	NC02	40	1400	2	TOH	YR
N-06905	CHERRY VLY G C	GDN CTY	1	78	56	404258	733855	NC02	70	500	1	TOH	SS
N-06915	BETHPAGE W D	BTHPG	10	608	558	404403	732831	NC03	85	1400	2	TOB	YR
N-06915A	BETHPAGE W D	BTHPG	10	475	422	404403	732831	NC03	85	1400	2	TOB	AB
N-06916	BETHPAGE W D	BTHPG	11	611	563	404403	732831	NC03	85	1400	2	TOB	YR
N-06945	GARDEN CITY PARK W D	SRNGTWN	7	401	351	404547	734011	NC02	145	1200	2	TNH	YR
N-06949	NEWSDAY	GDN CTY	2	51	46	404352	733647	NC02	79	75	1	TOH	YR
N-06953	DEFOREST DR ASSOC	LRL HLW	1	153	145	405143	732808	NC04	30	44	2	TOB	YR
N-06956	PLAINVIEW W D	PLNVIEW	5-1	597	514	404557	732705	NC03	180	1400	2	TOB	YR

NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-06965	SEAWANE CLUB	HEW HAR	1	137	121	403802	734041	NC02	12	500	6	TOH	SS
N-06985	SEAFORD H S	SEAFORD	1	46	36	404108	732931	NC03	35	40	1	TOH	SS
N-06994	GEORGIAN WEB OFFSET	G C EST	1	270	250	404421	733617	NC03	89	350	2	TOH	SS
N-07000	LAWRENCE STP	LAWRENC	1	110	99	403620	734418	NC01	9	100	6	TOH	YR
N-07006	V S AUTO LAUNDRY	V S	1	30	25	403934	734210	NC02	15	44	1	TOH	YR
N-07022	PATTERSON HOME	UNDALE	1	60	55	404153	733506	NC02	50	55	1	TOH	SS
N-07030	JERICHO W D	JERICHO	15	530	480	404635	733310	NC04	160	1200	2	TOB	YR
N-07034	WOODCREST C C	MUTNTWN	1	232	201	404922	733031	NC03	210	1000	1	TOB	AB
N-07045	SUNY-OLD WESTBURY	O WSTBY	2	151	136	404703	733449	NC04	241	48	2	TNH	NU
N-07047	PLANTING FIELDS ARBR	O BAY	2	264	212	405203	733311	NC03	185	500	2	TOB	NU
N-07052	SYOSSET HOSP	SYOSSET	1	254	234	404839	733030	NC03	220	530	2	TOB	YR
N-07053	NO HILLS C C	NO HLS	1	286	246	404631	734101	NC02	209	500	2	TNH	YR
N-07058	GARDEN CITY VILLAGE	GDN CTY	13	440	380	404320	734012	NC02	80	1400	2	TOH	YR
N-07076	LEVITTOWN W D	LEVITT	5A	674	569	404337	733039	NC03	90	1200	2	TOH	YR
N-07087	STERNS	GT NK	1	203	172	404607	734342	BG09	100	300	2	TNH	YR
N-07101	CENT GENERAL HOSP	PLNVIEW	1	100	90	404630	732848	NC03	143	45	2	TOB	SS
N-07104	ROSLYN W D	E HILLS	6	431	361	404832	733722	RS10	170	1200	2	TNH	YR
N-07114	MIDDLE BAY C C	OCNSIDE	1	200	138	403755	733738	NC02	11	500	2	TOH	SS
N-07115	MUTTONTOWN C C	MUTNTWN	1	274	234	405031	733314	NC04	205	700	2	TOB	YR
N-07117	FRANKLIN SQUARE W D	FR SQ	4	486	426	404213	734058	NC02	60	1400	2	TOH	YR
N-07126	MANHASSET-LAKEVL W D	NO HLS	1T	458	378	404651	734006	NC04	210	1400	2	TNH	YR
N-07133	SEAWANE CLUB	HEW HAR	2	150	134	403759	734122	NC02	8	500	6	TOH	SS
N-07157	SANDS POINT VILL	SNDS PT	4	240	135	405100	734100	NC04	120	500	2	TNH	YR
N-07174	ERDMAN BEVERAGES	UNDALE	1	67	57	404321	733629	NC02	76	150	1	TOH	NU
N-07216	GREAT NK SEWER DIST	GT NK	1	117	107	404745	734247	GN11	15	45	1	TNH	YR
N-07280	GLEN COVE COMM HOSP	GLN CV	2	193	173	405216	733718	GC05	140	180	2	TOB	YR
N-07289	ST RAPHAELS	E MEAD	1	37	30	404230	733247	NC03	60	45	1	TOH	AB
N-07298	HEMPSTEAD VILLAGE	HEMPSTD	8	444	394	404305	733712	NC02	60	1000	2	TOH	YR
N-07327	NORTH JR H S	LYNBRK	1	56	46	403937	734049	NC02	20	75	1	TOH	SS
N-07353	WESTBURY W D	NEW CSL	14	390	300	404555	733411	NC03	120	1400	2	TNH	YR
N-07362	ST PIUS X SEM	UNDALE	1	58	48	404235	733505	NC02	67	100	1	TOH	SS
N-07377	SO FARMINGDALE W D	PLNEDGE	1-4	758	607	404315	732751	NC03	60	1400	2	TOB	SS
N-07397	NCDPW	O BTHPG	U6A	101	96	404544	732656	NC03	154	0	1	TOB	OB
N-07407	N Y WATER SERVICE	MERRICK	11J	645	479	404002	733333	NC03	25	2100	2	TOH	SS
N-07408	EUR-AMER BANK	S FMGDL	1	36	30	404203	732557	NC03	40	25	1	TOB	YR
N-07414	N Y WATER SERVICE	MSPQ E	6M	530	378	404056	732611	NC03	25	2100	2	TOB	YR
N-07420	AMER INST PHYSICS	WDBRY	1	265	250	404802	732808	NC03	283	100	2	TOB	AB
N-07421	PLAINVIEW W O	PLNVIEW	5-2	559	482	404557	732705	NC03	180	1400	2	TOB	YR
N-07427	PHOTOCIRCUITS	GLN CV	1	161	120	405108	733714	GC05	58	750	1	TOB	YR
N-07433	CAMP BAUMAN	RSVLT	1	92	73	403957	733417	NC02	11	300	1	TOH	YR
N-07438	BETHPAGE ST PARK	FRMGDL	4	555	486	404454	732742	NC03	130	127	2	TOB	SS
N-07439	CRESCENT BEACH	GLN CV	1	212	206	405259	733851	GC05	20	30	3	TOB	SS
N-07445	JAMAICA W S CO	NO NHP	40A	448	388	404513	734124	NC02	120	1200	2	TOH	SS
N-07446	JERICHO W D	BKVL	16	493	443	404848	733443	NC04	195	1200	2	TOB	SS
N-07450	NCDPW	O WSTBY	H6A	134	129	404853	733600	NC03	176	0	1	TOB	OB
N-07464	BERNER H S	MASPQA	1	35	19	404039	732559	NC03	13	250	1	TOB	SS
N-07465	BERNER H S	MASPQA	2	35	19	404037	732559	NC03	9	250	1	TOB	SS
N-07482	JAMAICA W S CO	NO V S	25A	435	395	404109	734329	NC02	40	1200	2	TOH	YR
N-07500	EISENHOWER PARK	E MEAD	6	458	405	404419	733451	NC03	92	1400	2	TOH	YR
N-07512	GARDEN CITY PARK W D	HERRCKS	8	375	325	404536	734103	NC02	120	1200	2	TNH	YR
N-07513	OLD WESTBURY VILL	O WSTBY	2A	470	420	404652	733727	NC04	170	1200	2	TNH	YR
N-07515	SO FARMINGDALE W O	S FMGDL	5-1	347	289	404337	732711	NC03	65	1400	2	TOB	OC
N-07516	SO FARMINGDALE W O	S FMGDL	5-2	584	494	404337	732711	NC03	65	1400	2	TOB	OC
N-07518	GRUMMAN	BTHPG	16	375	314	404529	732934	NC03	133	1200	2	TOB	YR

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NASSAU COUNTY WELL LISTING

AREAS 249

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-07521	LONG ISLAND W C	LAKVW	17-2	555	445	403948	733929	NC02	20	1400	2	TOH	YR
N-07522	LONG ISLAND W C	LAKVW	19-2	555	495	404004	733922	NC02	20	1400	2	TOH	YR
N-07523	LEVITTOWN W D	LEVITT	8A	684	590	404309	733029	NC03	75	1200	2	TOH	YR
N-07526	PLAINVIEW W D	PLNVIEW	2-1	688	570	404703	732801	NC03	240	1400	2	TOB	YR
N-07529	HEMPSTEAD G C	HEMPSTD	1	62	44	404200	733805	NC02	45	600	1	TOH	YR
N-07534	GRUMMAN	BTHPG	6	366	288	404506	733009	NC03	120	1200	2	TOB	YR
N-07535	GRUMMAN	BTHPG	8	357	281	404518	732956	NC03	122	1200	2	TOB	YR
N-07536	GRUMMAN	BTHPG	9	436	375	404518	732949	NC03	125	1200	2	TOB	YR
N-07548	LONG ISLAND W C	V S	20-1	511	461	404012	734255	NC02	20	1400	2	TOH	YR
N-07549	OLD WESTBURY VILL	O WSTBY	4	499	449	404737	733531	NC04	195	1200	2	TNH	YR
N-07551	PORT WASHINGTON W D	NO HLS	MP8	469	375	404657	733957	PW08	170	1400	2	TNH	YR
N-07552	PORT WASHINGTON W D	NO HLS	MP9	454	360	404650	733953	PW08	160	1400	2	TNH	YR
N-07560	LI JEWISH HOSP	N H P	1	242	221	404532	734220	NC02	150	310	2	TNH	YR
N-07561	HICKSVILLE W D	HKSVL	5-2	550	463	404455	733249	NC03	115	1400	2	TOB	YR
N-07562	HICKSVILLE W D	HKSVL	1-4	545	458	404639	733111	NC03	165	1400	2	TOB	YR
N-07569	E ROCKAWAY H S	E RCKWY	1	35	30	403853	733925	NC02	10	45	1	TOH	SS
N-07593	JERICHO W D	LRL HLW	17	468	408	405045	732833	NC04	235	1200	2	TOB	YR
N-07613	ATLASS RESIDENCE	KNGS PT	1	235	221	404814	734518	NC04	36	75	3	TNH	YR
N-07614	POWERS CHEMCO	GLN CV	3	393	351	405151	733819	GC05	32	500	3	TOB	YR
N-07620	BAYVILLE VILLAGE	BAYVL	1-1	480	412	405424	733400	NC04	120	1000	3	TOB	YR
N-07632	CALHOUN H S	MERRICK	1	65	60	404028	733350	NC03	25	100	2	TOH	SS
N-07635	GRUMMAN	BTHPG	5	394	314	404500	733004	NC03	120	1200	2	TOB	YR
N-07636	GRUMMAN	BTHPG	10	373	312	404517	732943	NC03	125	1200	2	TOB	YR
N-07637	GRUMMAN	BTHPG	11	490	429	404516	732935	NC03	126	1200	2	TOB	YR
N-07643	BAYVILLE VILLAGE	BAYVL	1-2	218	159	405424	733400	NC04	120	1000	1	TOB	NU
N-07649	JAMAICA W S CO	N H P	57	205	165	404345	734119	NC02	95	1200	2	TOH	SS
N-07650	JAMAICA W S CO	N H P	57A	440	400	404345	734119	NC02	95	1200	2	TOH	SS
N-07651	MANHASSET-LAKEVL W D	NO HLS	23	405	321	404612	734012	NC04	160	1400	2	TNH	YR
N-07664	ENGINEERS C C	ROS HBR	2	79	58	404910	733812	NC04	53	500	1	TNH	YR
N-07665	LOCUST VALLEY W D	MATINCK	8	370	320	405203	733500	NC04	220	1200	2	TOB	YR
N-07719	BAYVILLE VILLAGE	BAYVL	1	400	398	405452	733430	NC04	10	10	3	TOB	NU
N-07720	WEST HEMPSTEAD W D	W HEMP	7	506	435	404236	733954	NC02	70	1400	2	TOH	YR
N-07729	SILL RESIDENCE	LRL HLW	1	356	348	405102	732821	NC04	240	18	2	TOB	YR
N-07732	AUTRONICS PLASTICS	NEW CSL	1	108	96	404517	733339	NC03	120	45	2	TNH	YR
N-07744	PLATTDEUTSCHE HOME	FR SQ	1	60	54	404234	734058	NC02	65	45	1	TOH	YR
N-07747	MANHASSET-LAKEVL W D	KENSGTN	42	138	117	404737	734235	GN11	20	1600	1	TNH	SS
N-07772	JERICHO W D	SYOSSET	18	563	503	405012	733055	NC04	240	1200	2	TOB	YR
N-07773	JERICHO W D	SYOSSET	19	560	500	405012	733055	NC04	240	1200	2	TOB	YR
N-07776	LONG BEACH CITY	LNG BCH	13	1233	1183	403534	733955	LB07	5	1200	3	TOH	YR
N-07781	JERICHO W D	JERICHO	22	454	394	404751	733220	NC04	215	1200	2	TOB	SS
N-07785	WESTBURY W D	WSTBY	7A	400	330	404526	733534	NC03	110	1400	2	TNH	YR
N-07796	FREEPORT VILLAGE	FRPT	1A	585	525	403952	733422	FS06	20	1400	2	TOH	YR
N-07797	EAST MEADOW W D	E MEAD	11	545	485	404310	733316	NC03	85	1450	2	TOH	YR
N-07799	GARDEN CITY C C	GDN CTY	1	81	60	404319	734016	NC02	65	500	1	TOH	SS
N-07830	MILL RIVER CLUB	O BAY	1	197	166	405116	733257	NC04	118	600	2	TOB	SS
N-07831	LONG ISLAND W C	BALDWIN	22-1	585	515	404023	733713	NC02	40	1400	2	TOH	YR
N-07834	GLEN HEAD C C	SEA CLF	2	202	171	405043	733716	NC04	150	500	2	TOB	YR
N-07847	CAMP BAUMAN	RSVLT	3	40	36	403957	733417	NC02	15	15	1	TOH	YR
N-07849	ATLASS BLDG	MINEOLA	1	251	221	404425	733817	NC02	104	530	2	TNH	AB
N-07852	FARMINGDALE VILLAGE	FRMGDL	1-3	450	400	404411	732618	NC03	70	1200	2	TOB	YR
N-07853	MAJESTIC PROD	UNDALE	1	30	25	404345	733641	NC02	80	10	1	TOH	NU
N-07855	LONG ISLAND W C	MLVRN	23-1	600	530	404042	734037	NC02	35	1400	2	TOH	YR
N-07856	EAST MEADOW H S	E MEAD	1	70	50	404349	733333	NC03	90	45	1	TOH	SS
N-07857	SEA CLIFF W S CO	SEA CLF	SC	614	560	405059	733841	NC04	180	1300	3	TOB	SS

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-07858	TAM O'SHANTER C C	BKVL	1	375	310	404834	733326	NC03	218	1000	2	TOB	YR
N-07873	ROSLYN W D	E HILLS	7	530	470	404815	733639	NC02	220	1200	2	TNH	YR
N-07892	MANHASSET-LAKEVL W D	NO HLS	3T	451	366	404651	734006	NC04	210	1400	2	TNH	SS
N-07957	ROOSEVELT FIELD W D	G C EST	5	519	433	404420	733532	NC02	85	1400	2	TOH	YR
N-07997	BELLMORE ANIMAL HOSP	BELMR	1	94	88	404001	733200	NC03	15	20	2	TOH	YR
N-08004	BETHPAGE W D	BTHPG	5-1	740	679	404343	732843	NC03	80	1400	2	TOB	YR
N-08007	WESTBURY W D	WSTBY	15	564	490	404543	733549	NC03	120	1400	2	TNH	YR
N-08008	NEWBRIDGE RD PK	BELMR	1	385	364	403911	733209	NC03	11	140	2	TOH	SS
N-08010	ROSLYN W D	ROS EST	8	448	378	404741	733920	RS10	220	1200	2	TNH	YR
N-08011	LONG BEACH CITY	LNG BCH	14	1265	1205	403533	734010	LB07	5	1250	3	TOH	YR
N-08021	COVE NK TENNIS	COV NK	1	200	194	405241	733018	NC04	10	20	2	TOB	YR
N-08026	PARISI HARDWARE	N H P	1	119	111	404422	734000	NC02	105	22	1	TNH	AB
N-08031	N Y WATER SERVICE	WANTAGH	10	509	388	404052	732948	NC03	20	2100	2	TOH	YR
N-08038	LAKE SUCCESS G C	LK SUCC	1	295	271	404604	734244	NC04	210	700	2	TNH	YR
N-08039	HARDER EXTERM	HEMPSTD	1	55	44	404204	733703	NC02	65	48	1	TOH	YR
N-08043	JERICHO W D	WDBRY	23	688	515	404757	732833	NC03	220	1200	2	TOB	NU
N-08044	COMM CHURCH-E WILL	E WILL	1	94	86	404532	733812	NC02	118	45	1	TNH	SS
N-08046	NCDPW	KNGS PT	--	189	184	404947	734503	NC04	12	0	1	TNH	OB
N-08050	INA BUILDING	G C EST	1	328	300	404422	733700	NC03	91	225	2	TOH	SS
N-08052	NCDPW	KNGS PT	--	94	89	404947	734502	NC04	13	0	1	TNH	OB
N-08054	PLAINVIEW W D	PLNVIEW	5-3	580	510	404557	732705	NC03	180	1400	2	TOB	SS
N-08068	RELIANCE FED	G C EST	1	291	265	404359	733646	NC03	80	500	2	TOH	YR
N-08093	CYCO PLASTICS	MINEOLA	1	90	77	404437	733807	NC02	105	45	1	TNH	AB
N-08124	GRUMMAN	BTHPG	3	543	483	404500	732955	NC03	116	1200	2	TOB	YR
N-08129	AURORA PLASTICS	W HEMP	1	64	45	404225	733923	NC02	54	300	1	TOH	YR
N-08136	FARMINGDALE H S	FRMGDL	1	70	55	404226	732700	NC03	43	300	1	TOB	SS
N-08153	MEPHAM H S	NO BLMR	1	84	73	404036	733235	NC03	31	100	1	TOH	SS
N-08154	GRUMMAN	BTHPG	2	520	424	404454	732954	NC03	120	1200	2	TOB	YR
N-08162	BELLMORE-KENNEDY H S	BELMR	1	154	133	403904	733136	NC03	9	150	2	TOH	SS
N-08171	TOH GOLF COURSE	MERRICK	1	378	341	403855	733337	NC03	9	360	2	TOH	SS
N-08181	O WESTBURY GDNS	O WSTBY	1	240	230	404616	733547	NC03	141	150	2	TNH	YR
N-08183	OYSTER BAY W D	O BAY	6-1	230	181	405146	733134	OB14	80	1100	2	TOB	YR
N-08195	LONG ISLAND W C	LYNBRK	24-1	507	447	403934	734107	NC02	20	1400	2	TOH	YR
N-08196	LONG ISLAND W C	BALDWIN	4-17	620	560	403953	733617	NC02	20	1400	2	TOH	YR
N-08214	MASSAPEQUA W D	MASPQA	8	686	605	404145	732618	NC03	40	1400	2	TOB	YR
N-08216	ROCKVILLE CTR VILL	RVC	11	660	600	404004	733714	NC02	32	1400	2	TOH	YR
N-08217	ROCKVILLE CTR VILL	RVC	12	503	483	404004	733714	NC02	32	1400	2	TOH	YR
N-08218	ROCKVILLE CTR VILL	RVC	13	460	400	404109	733742	NC02	40	1400	2	TOH	YR
N-08221	ARYEH RESIDENCE	KNGS PT	1	290	250	404922	734500	NC04	70	45	3	TNH	SS
N-08224	PHOTOCIRCUITS	GLN CV	2	155	104	405108	733714	GC05	58	1000	1	TOB	YR
N-08228	WEST HEMPSTEAD H S	W HEMP	1	69	51	404137	733945	NC02	48	100	1	TOH	SS
N-08233	LONG BEACH CITY	LNG BCH	15	1226	1176	403518	733820	LB07	10	1250	3	TOH	YR
N-08246	SANDS POINT G C	SNDS PT	1	350	320	405121	734159	NC04	101	500	7	TNH	SS
N-08248	WILLISTON PK VILL	WIL PK	4	400	315	404525	733825	NC02	115	1400	2	TNH	YR
N-08249	HICKSVILLE W D	HKSVL	1-5	490	400	404639	733111	NC03	165	1400	2	TOB	NU
N-08250	LONG ISLAND W C	SO HEMP	18-2	480	420	404112	733716	NC02	50	1400	2	TOH	YR
N-08251	LONG ISLAND W C	LYNBRK	15-2	495	430	403956	734104	NC02	20	1400	2	TOH	YR
N-08253	N Y WATER SERVICE	MERRICK	12J	597	562	404002	733333	NC03	25	1250	2	TOH	YR
N-08264	HEMPSTEAD VILLAGE	HEMPSTD	9	510	460	404149	733731	NC02	50	1500	2	TOH	YR
N-08265	BROOKSIDE JR H S	NO MRK	1	68	63	404118	733414	NC03	40	100	2	TOH	SS
N-08279	LEVITTOWN W D	LEVITT	7A	547	389	404309	733029	NC03	75	1200	2	TOH	YR
N-08305	BOCES	CAR PL	1	59	51	404457	733710	NC02	91	120	1	TNH	SS
N-08313	SANDS POINT VILL	SNDS PT	5	165	114	405111	734302	NC04	60	650	1	TNH	YR
N-08321	LEVITTOWN W D	LEVITT	2A	674	574	X 404400	733148	NC03	100	1200	2	TOH	YR

Same is 7562?

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-08326	GLEN COVE CITY	GLN CV	21	165	120	405116	733729	GC05	80	1400	2	TOB	YR
N-08327	GLEN COVE CITY	GLN CV	22	165	115	405116	733729	GC05	80	1400	2	TOB	AB
N-08339	GARDEN CITY VILLAGE	GDN CTY	14	358	308	404320	734012	NC02	80	1500	2	TOH	YR
N-08342	GREAT NECK NORTH W A	GT NK	11	434	373	404650	734445	GN11	25	1050	3	TNH	SS
N-08343	GLEN COVE COMM HOSP	GLN CV	3	420	380	405216	733721	GC05	140	650	3	TOB	YR
N-08344	TOH HOUSING AUTH	V S	1	58	33	403936	734312	NC02	10	45	1	TOH	SS
N-08354	LIDO-PT LOOKOUT W D	LID BCH	3	1270	1215	403522	733659	LB07	5	1200	3	TOH	YR
N-08355	JERICHO W D	MUTNTWN	25	590	530	404837	733158	NC04	260	1400	2	TOB	YR
N-08383	CHESTER RESIDENCE	CTR ISL	1	105	99	405339	733127	NC04	30	15	1	TOB	YR
N-08389	JIM DANDY CLEANERS	ELMONT	1	93	83	404117	734215	NC02	40	25	1	TOH	AB
N-08409	GARDEN CITY PARK W D	G C PK	9	400	340	404420	733939	NC02	100	1200	2	TNH	NU
N-08412	NCDPW	WANTAGH	X41A	28	23	404041	733032	NC03	26	0	1	TOH	OB
N-08413	GREAT NK VILL STP	GT NK	1	133	123	404750	734253	GN11	100	45	2	TNH	YR
N-08414	JONES BEACH ST PK	WANTAGH	4	1080	1005	403558	733029	JB13	9	1200	2	TOH	YR
N-08420	LONG ISLAND W C	V S	9-1R	420	350	404031	734145	NC02	20	1400	2	TOH	YR
N-08423	EUR-AMER BANK	WSTBY	1	98	81	404446	733516	NC03	90	150	2	TOH	YR
N-08426	MILL NECK EST W S	MILL NK	2	360	345	405400	733319	NC04	20	160	3	TOB	YR
N-08430	NCDPW	E NORW	P5A	145	140	405009	733147	NC04	190	0	2	TOB	OB
N-08432	C W POST	GRNVL	1	250	220	404939	733525	NC03	165	450	2	TOB	SS
N-08436	METCO INC	HKSVL	2	188	159	404712	733058	NC03	175	300	2	TOB	YR
N-08454	GRUMMAN	BTHPG	13	560	499	404524	732949	NC03	129	1200	2	TOB	YR
N-08457	CARLE PLACE W D	CAR PL	5	435	375	404457	733607	NC03	100	1200	2	TNH	YR
N-08458	GIMBELS	G C EST	1	350	290	404422	733645	NC03	90	1200	2	TOH	SS
N-08471	NCDPW	BELMR	X38A	18	13	403939	733222	NC03	5	0	1	TOH	AB
N-08472	ADVANCED FOODS	NEW CSL	1	195	177	404541	733309	NC03	125	30	2	TNH	YR
N-08474	UNIONDALE W D	UNDALE	5	556	485	404325	733631	NC02	80	1450	2	TOH	YR
N-08475	UNIONDALE W D	UNDALE	6	481	409	404325	733631	NC02	80	1450	2	TOH	YR
N-08477	ROSLYN PLAZA	ROSLYN	1	710	668	404752	733953	NC04	180	250	3	TNH	YR
N-08478	PUB CLEAR HOUSE	P WSH N	1	156	145	405041	734158	PW08	20	45	1	TNH	YR
N-08480	N Y WATER SERVICE	NO WTGH	3S	656	569	404228	732933	NC03	60	1550	2	TOH	YR
N-08481	OCEANSIDE SCH #4	OCNSIDE	4	55	44	403800	733810	NC02	8	100	1	TOH	SS
N-08482	OCEANSIDE SCH #6	OCNSIDE	6	80	69	403842	733806	NC02	17	100	2	TOH	SS
N-08483	OCEANSIDE SCH #8	OCNSIDE	8	55	44	403756	733848	NC02	8	100	1	TOH	SS
N-08487	WANTAGH H S	WANTAGH	1	49	39	404126	733051	NC03	35	200	1	TOH	SS
N-08497	WESTBURY W D	NEW CSL	16	539	456	404519	733429	NC03	110	1400	2	TNH	YR
N-08499	WERE ASSOC	LK SUCC	1	270	160	404542	734126	NC02	140	1200	2	TNH	SS
N-08512	HEMP TOWN HALL	HEMPSTD	1	52	40	404224	733715	NC02	60	125	1	TOH	AB
N-08522	MID ISLAND HOSP	BTHPG	1	125	105	404338	732843	NC03	86	500	1	TOB	SS
N-08525	HICKSVILLE W D	HKSVL	3-2	503	432	404446	733057	NC03	115	1400	2	TOB	YR
N-08526	HICKSVILLE W D	HKSVL	4-2	601	520	404455	733203	NC03	120	1400	2	TOB	YR
N-08530	C D HDQTS	GDN CTY	[illegible]	80	70	404414	733825	NC02	100	100	1	TOH	SS
N-08531	C D HDQTS	GDN CTY	2	84	73	404414	733825	NC02	100	100	1	TOH	SS
N-08534	LAWRENCE G C	LAWRENC	2	265	247	403557	734337	NC02	13	240	2	TOH	SS
N-08542	MUTTONTOWN C C	MUTNTWN	2	335	294	405031	733314	NC04	175	700	2	TOB	YR
N-08550	NCDPW	LEVITT	012A	66	61	404403	733220	NC03	102	0	1	TOH	OB
N-08554	KLEER-PAK	INWOOD	1	25	21	403647	734452	NC01	20	16	1	TOH	YR
N-08557	LONG BEACH CITY	LNG BCH	16	1253	1203	403534	733955	LB07	5	1250	3	TOH	YR
N-08558	ALBERTSON W D	ALBTSN	5	410	350	404626	733829	NC02	145	1200	2	TNH	YR
N-08564	WERE ASSOC	LK SUCC	2	320	170	404542	734124	NC02	140	1200	2	TNH	SS
N-08576	MINEOLA VILLAGE	MINEOLA	7	505	445	404455	733753	NC02	110	1000	2	TNH	YR
N-08585	NCDPW	N H P	--	107	102	404443	734044	NC02	108	0	1	TNH	OB
N-08595	PLAINVIEW W D	PLNVIEW	5-4	610	540	404557	732705	NC03	180	1350	2	TOB	SS
N-08598	NCDPW	UNDALE	G14A	45	---	404239	733556	NC02	70	0	--	TOH	OB
N-08601	HOLY ROOD CEM	WSTBY	1	340	319	404505	733453	NC03	104	150	2	TNH	YR

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-08602	SUTTER MONUMENTS	HKSVL	1	84	80	404536	733201	NC03	130	6	1	TOB	AB
N-08603	N Y WATER SERVICE	MSPQ E	7M	893	802	404056	732611	NC03	25	2100	2	TOB	YR
N-08622	HASSET MERCURY	WANTAGH	1	200	191	404017	733010	NC03	22	20	2	TOH	YR
N-08623	AMER IMPR PRODUCTS	N H P	2	96	86	404430	733938	NC02	100	69	1	TNH	YR
N-08625	COMMANDER OIL	UNDALE	1	35	32	404352	733624	NC02	80	7	1	TOH	AB
N-08627	OCEANSIDE SCH #7	OCNSIDE	7	84	73	403814	733751	NC02	9	250	1	TOH	SS
N-08629	NCDPW	MSPQ E	U15	183	178	403920	732534	NC03	3	0	2	TOB	OB
N-08641	NCDPW	ATL BCH	X122	67	62	403513	734413	NC02	9	0	1	TOH	AB
N-08643	GRUMMAN	BTHPG	14	467	416	404513	732956	NC03	122	1200	2	TOB	YR
N-08645	NCDPW	LAWRENC	D19	90	85	403553	734331	NC01	6	0	1	TOH	OB
N-08657	FREEPORT VILLAGE	FRPT	9	635	565	403927	733550	FS06	20	1400	2	TOH	YR
N-08658	OLD WESTBURY VILL	O WSTBY	5	610	550	404820	733440	NC04	300	1400	2	TOB	YR
N-08659	GIM METALS	CAR PL	1	70	63	404448	733712	NC02	100	30	1	TNH	AB
N-08664	SO FARMINGDALE W D	MASPQA	6-1	606	529	404221	732545	NC03	45	1400	2	TOB	YR
N-08665	SO FARMINGDALE W D	MASPQA	6-2	576	506	404221	732545	NC03	45	1400	2	TOB	YR
N-08666	LEVER BLDG 100	GDN CTY	2	67	43	404411	733701	NC03	91	150	1	TOH	OB
N-08668	TOB INCINERATOR	O BTHPG	1	485	434	404551	732742	NC03	141	700	2	TOB	YR
N-08672	N Y WATER SERVICE	NO WTGH	3J	570	518	404130	733114	NC03	40	700	2	TOH	AB
N-08674	GATEWAY TIRE	FRPT	1	24	20	403904	733407	FS06	5	16	1	TOH	AB
N-08681	FOX RUN C C	MUTNTWN	1	370	317	405016	733246	NC04	200	50	2	TOB	SS
N-08682	NYS DMV	WSTBY	1E	110	90	404442	733533	NC03	100	200	2	TOH	SS
N-08713	JERICHO W D	O BKVL	27	372	312	404919	733733	NC04	180	1400	2	TOB	YR
N-08714	EUR-AMER BANK	WSTBY	2	282	272	404446	733516	NC03	90	200	2	TNH	YR
N-08753	NYS DMV	WSTBY	2W	109	99	404442	733534	NC03	100	200	2	TOH	SS
N-08761	NO HEMPSTEAD C C	PT WASH	2	255	224	404843	734040	PW08	140	600	1	TNH	YR
N-08767	BETHPAGE W D	BTHPG	7-A	640	579	404532	732848	NC03	125	1400	2	TOB	YR
N-08768	BETHPAGE W D	BTHPG	8-A	678	605	404533	732848	NC03	125	1400	2	TOB	YR
N-08774	OCEANSIDE SCH #5	OCNSIDE	5	36	26	403911	733758	NC02	22	70	1	TOH	SS
N-08775	BOARDMAN JR H S	OCNSIDE	9	130	100	403728	733742	NC02	8	200	2	TOH	SS
N-08776	BAYVILLE VILLAGE	BAYVL	1-3	459	399	405427	733355	NC04	120	1000	3	TOB	YR
N-08778	HICKSVILLE W D	HKSVL	9-1	590	529	404537	733046	NC03	140	1400	2	TOB	YR
N-08779	HICKSVILLE W D	HKSVL	9-2	585	524	404537	733046	NC03	140	1400	2	TOB	YR
N-08789	NCDPW	MASPQA	T15A	30	25	404152	732730	NC03	40	0	1	TOB	OB
N-08790	PLANDOME C C	PLANOME	1	430	400	404858	734125	NC04	100	400	3	TNH	SS
N-08799	WHEATLEY HILLS G C	E WILL	2	221	190	404540	733742	NC02	111	600	2	TNH	YR
N-08807	CERTIFIED INDUST	HKSVL	1	140	109	404543	733247	NC03	118	350	2	TOB	AB
N-08808	MARIA REGINA H S	UNDALE	1	67	55	404245	733453	NC02	60	45	1	TOH	SS
N-08816	GRUMMAN	BTHPG	15	500	449	404523	732935	NC03	129	1200	2	TOB	YR
N-08818	FRANKLIN SQUARE W D	FR SQ	5	480	420	404213	734058	NC02	60	1200	2	TOH	SS
N-08830	HEMPSTEAD H S	HEMPSTD	1	84	44	404153	733754	NC02	38	850	1	TOH	YR
N-08836	VALLEY TOWNE HOUSE	V S	1	62	56	404037	734106	NC02	34	20	1	TOH	YR
N-08837	N Y WATER SERVICE	WANTAGH	5D	681	614	404052	732948	NC03	20	1200	2	TOH	YR
N-08842	GRUMMAN	BTHPG	1	570	519	404439	732954	NC03	111	1200	2	TOB	YR
N-08855	CROSS CTRY PAPER	HEMPSTD	1	32	29	404216	733739	NC02	55	10	1	TOH	YR
N-08875	NCDPW	MASPQA	X120	37	32	403929	732745	NC03	5	0	1	TOB	OB
N-08876	NCDPW	MASPQA	X128	35	30	403925	732611	NC03	5	0	1	TOB	OB
N-08877	NCDPW	MANHST	X129	76	71	404750	734231	NC04	12	0	1	TNH	OB
N-08879	NCDPW	PLDM MR	X130	63	50	404853	734210	NC04	10	0	1	TNH	OB
N-08880	METCO INC	HKSVL	1	247	221	404535	733244	NC03	122	300	2	TOB	YR
N-08881	ROCKVILLE LINKS	RVC	3	71	51	404029	733745	NC02	38	325	1	TOH	YR
N-08882	ROCKVILLE LINKS	RVC	4	61	41	404036	733735	NC02	39	325	1	TOH	YR
N-08885	OLD WESTBURY C C	O WSTBY	2	298	257	404745	733614	NC03	196	750	2	TNH	YR
N-08887	SLATER ELECT	SEA CLF	1	130	105	405058	733811	NC04	65	240	1	TOB	YR
N-08888	NCDPW	HKSVL	P8A	111	106	404705	733056	NC03	175	0	1	TOB	OB

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-08891	NCDPW	SNDS PT	X132	72	67	405055	734307	NC04	80	0	1	TNH	OB
N-08896	VALLEY STR INCIN	V S	1	88	68	404041	734311	NC02	31	300	1	TOH	YR
N-08941	BETHPAGE W D	PLNEDGE	6-2	770	700	404354	732912	NC03	90	1400	2	TOB	YR
N-08956	BOWLING GREEN W D	BOWL GR	1	530	470	404509	733334	NC03	120	1400	2	TOH	YR
N-08957	BOWLING GREEN W D	BOWL GR	2	584	524	404509	733334	NC03	120	1400	2	TOH	YR
N-08958	NCDPW	UNDALE	X140	35	30	404302	733541	NC02	77	0	1	TOH	OB
N-08961	FLAGG RESIDENCE	OBAY CO	1	143	138	405200	733000	NC04	---	18	1	TOB	YR
N-08969	HEMPSTEAD VILL HALL	HEMPSTD	1	33	30	404235	733725	NC02	60	7	1	TOH	NU
N-08971	NCDPW	----	X-144	38	33	------	------	----	---	---	1	TOH	OB
N-08976	N Y WATER SERVICE	NO BLMR	3N	700	619	404117	733230	NC03	40	2100	2	TOH	YR
N-08979	LONG ISLAND W C	LYNBRK	24-2	435	375	403934	734107	NC02	22	1400	2	TOH	YR
N-08984	NCDPW	BOWL GR	H10A	48	43	404454	733447	NC03	104	0	1	TOH	OB
N-08994	SANDS POINT VILL	SNDS PT	0000	308	298	405153	734206	NC04	20	0	3	TNH	NU
N-08998	V S CENTRAL H S	V S	1	42	34	404011	734203	NC02	20	60	1	TOH	SS
N-09005	TNH LANDFILL	PT WASH	2	45	40	404900	733959	NC04	170	0	1	TNH	NU
N-09006	TNH LANDFILL	PT WASH	1	85	80	404900	733959	NC04	170	0	1	TNH	NU
N-09007	TNH LANDFILL	PT WASH	4	95	85	404902	734018	NC04	170	0	1	TNH	AB
N-09008	TNH LANDFILL	PT WASH	3	180	170	404902	734018	NC04	170	0	2	TNH	AB
N-09018	NAT WESTMINSTER BANK	HKSVL	1	405	380	404600	733134	NC03	148	120	2	TOB	YR
N-09019	TNH LANDFILL	PT WASH	5	411	369	404853	733959	NC04	170	45	3	TNH	YR
N-09020	NC MEDICAL CTR	E MEAD	1	72	52	404334	733312	NC03	91	800	1	TOH	YR
N-09021	NC MEDICAL CTR	E MEAD	1	74	54	404335	733314	NC03	91	800	1	TOH	YR
N-09023	PINE HOLLOW C C	E NORW	2	247	217	405108	733146	NC04	219	700	1	TOB	NU
N-09029	NAS-SUFF VETER HOSP	FRMGDL	1	69	64	404355	732720	NC03	70	40	1	TOB	YR
N-09030	NASSAU COLISEUM	UNDALE	1	63	43	404320	733512	NC02	82	800	1	TOH	YR
N-09054	NCDPW	BALDWIN	X95A	40	35	403823	733633	NC02	14	0	1	TOH	OB
N-09057	NCDPW	E MEAD	H13A	47	42	404244	733423	NC03	70	0	1	TOH	OB
N-09059	NCDPW	BKVL	O7A	175	170	404832	733323	NC03	228	0	2	TOB	OB
N-09068	SAGAMORE HILL	COV NK	1	170	160	405306	733000	NC04	130	25	1	TOB	NU
N-09072	ANZIANO BLDG	MINEOLA	2	84	65	404437	733826	NC02	109	200	1	TNH	SS
N-09073	MCELHINNEY RESIDENCE	MILL NK	1	134	129	405221	733324	NC04	120	20	2	TOB	YR
N-09076	SAGAMORE HILL	COV NK	1R	200	189	405308	733000	NC04	130	75	1	TOB	YR
N-09077	NCDPW	NO BLMR	O14A	52	47	404242	733158	NC03	69	0	1	TOH	OB
N-09078	NCDPW	E MEAD	H12A	65	60	404321	733425	NC03	84	0	1	TOH	OB
N-09079	NCDPW	HKSVL	P10A	70	65	404508	733013	NC03	119	0	1	TOB	OB
N-09087	NCDPW	UPBRKVL	O5A	111	106	405020	733357	NC03	157	0	1	TOB	OB
N-09088	NCDPW	BTHPG	X93A	68	63	404415	732824	NC03	78	0	1	TOB	OB
N-09089	NCDPW	PLNVIEW	T7A	178	173	404718	732859	NC03	173	0	2	TOB	OB
N-09098	NCDPW	GT NK	X67A	72	67	404831	734446	VG12	59	0	1	TNH	OB
N-09099	NCDPW	GT NK	X68A	71	66	404802	734407	VG12	60	0	1	TNH	OB
N-09100	NCDPW	GLN CV	G2A	70	65	405202	733752	GC05	54	0	1	TOB	OB
N-09102	STIEHLER GDN CTR	OCNSIDE	1	24	20	403915	733731	NC02	25	25	1	TOH	YR
N-09106	MCLELLAN STORE	FRMGDL	1	59	53	404356	732646	NC03	73	50	1	TOB	YR
N-09115	NCDPW	GLN CV	H4A	110	105	405115	733614	NC04	145	0	1	TOB	OB
N-09116	NCDPW	SNDS PT	E1A	31	26	405135	734101	NC04	15	0	1	TNH	OB
N-09117	NCDPW	O BKVL	H5A	73	68	405022	733559	NC04	112	0	1	TOB	OB
N-09118	NCDPW	SNDS PT	X133A	100	95	405147	734331	NC04	50	0	1	TNH	OB
N-09127	NCDPW	BAYVL	P1B	41	36	405419	733259	NC04	9	0	1	TOB	OB
N-09151	JAMAICA W S CO	ELMONT	15A	420	380	404224	734240	NC02	60	1200	2	TOH	YR
N-09152	NCDPW	LKL HLW	U1A	58	53	405125	732758	NC04	40	0	1	TOB	OB
N-09154	NCDPW	OBAY CO	T3A	66	61	405159	733002	NC04	34	0	1	TOB	OB
N-09168	NCDPW	O WSTBY	H8A	217	212	404632	733451	NC03	165	0	2	TNH	OB
N-09173	MASSAPEQUA W D	MASPQA	2R	850	810	404154	732618	NC03	40	2100	2	TOB	YR
N-09180	HICKSVILLE W D	HKSVL	8-3	630	545	404521	733102	NC03	135	1400	2	TOB	YR

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-09188	NCDPW	MANHST	01A	80	75	404654	734216	NC04	45	0	1	TNH	OB
N-09189	NCDPW	O BAY	P3A	42	37	405150	733201	OB14	59	0	1	TOB	OB
N-09190	NCDPW	O WSTBY	G8A	133	128	404703	733701	NC03	156	0	1	TNH	OB
N-09191	NCDPW	O WSTBY	G9A	135	130	404620	733646	NC03	176	0	1	TNH	OB
N-09210	GLEN COVE CITY	GLN CV	30	275	209	405205	733628	GC05	150	1400	2	TOB	YR
N-09211	GLEN COVE CITY	GLN CV	31	269	209	405205	733628	GC05	150	1400	2	TOB	YR
N-09212	HICKSVILLE W D	HKSVL	5-3	604	538	404455	733249	NC03	115	1400	2	TOB	YR
N-09261	PATTERSON HOME	UNDALE	1	522	469	404155	733507	NC02	50	1000	2	TOH	YR
N-09262	PATTERSON HOME	UNDALE	2	552	500	404152	733510	NC02	50	1000	2	TOH	YR
N-09271	NCDPW	KNGS PT	X-148	89	84	404838	734537	NC04	47	0	1	TNH	OB
N-09308	MANHASSET-LAKEVL W D	KENSGTN	5R	410	308	404737	734235	NC04	20	1400	3	TNH	YR
N-09310	LEVER BLDG 300	GDN CTY	1	230	180	404405	733658	NC03	95	850	2	TOH	SS
N-09311	LEVER BLDG 100	GDN CTY	1	229	190	404410	733701	NC03	95	850	2	TOH	SS
N-09313	NCDPW	ROSLYN	F1AA	58	53	404747	733855	RS10	60	0	1	TNH	OB
N-09314	NCDPW	MILL NK	02A	54	49	405354	733453	NC04	32	0	1	TOB	OB
N-09316	NCDPW	COV NK	T1A	58	53	405329	733023	NC04	25	0	1	TOB	OB
N-09317	NCDPW	MUTNTWN	P6A	194	189	404929	733147	NC03	217	0	2	TOB	OB
N-09334	GLEN COVE CITY	GLN CV	K	298	248	405132	733704	GC05	140	1200	2	TOB	YR
N-09338	N Y WATER SERVICE	NO WTGH	4S	649	585	404228	732933	NC03	60	2100	2	TOH	YR
N-09341	PRD ELECTRONICS	NEW CSL	2	265	231	404550	733304	NC03	130	200	2	TNH	YR
N-09353	NCDPW	BKVL	06A	101	96	404933	733348	NC04	140	0	2	TOB	OB
N-09354	NCDPW	WSTBY	H9A	89	84	404540	733500	NC03	118	0	2	TNH	OB
N-09355	NCDPW	UNDALE	G13B	58	53	404336	733546	NC02	77	0	1	TOH	OB
N-09356	NCDPW	SNDS PT	X151	104	99	405124	734205	NC04	100	0	1	TNH	OB
N-09357	NCDPW	FRPT	X35A	27	22	403907	733608	FS06	8	0	1	TOH	OB
N-09358	NCDPW	WDMR	X8A	20	15	403758	734328	NC02	5	0	1	TOH	OB
N-09359	NCDPW	V S SO	X7A	26	21	403910	734323	NC02	5	0	1	TOH	OB
N-09398	-------	GDN CTY	--	22	16	404311	733716	NC02	---	0	--	TOH	--
N-09403	-------	----	--	---	---	------	------	----	---	---	--	---	--
N-09446	SANDS POINT VILL	SNDS PT	6	368	328	405115	734306	NC04	61	600	3	TNH	YR
N-09452	WEST HEMPSTEAD W D	W HEMP	8	595	521	404142	733840	NC02	60	1400	2	TOH	YR
N-09453	TOB INCINERATOR	O BTHPG	1	65	500	404540	732655	NC03	160	0	1	TOB	AB
N-09463	HICKSVILLE W D	HKSVL	10-1	638	560	404605	733143	NC03	140	1380	2	TOB	YR
N-09468	NCDPW	INWOOD	X11A	25	20	403721	734434	NC01	7	0	1	TOH	OB
N-09469	NCDPW	MSPQ PK	T16A	46	41	404113	732715	NC03	31	0	1	TOB	OB
N-09470	NCDPW	FRPT	G18A	29	24	404002	733527	FS06	28	0	1	TOH	OB
N-09471	NCDPW	SEAFORD	P15A	41	36	404127	732909	NC03	32	0	1	TOH	OB
N-09472	NCDPW	MLVRN	E15A	36	31	404020	733941	NC02	25	0	1	TOH	OB
N-09473	NCDPW	NO BLMR	X36A	42	37	404127	733247	NC03	41	0	1	TOH	OB
N-09475	NCDPW	ATL BCH	X122A	68	63	403526	734413	NC02	8	0	1	TOH	OB
N-09478	NCDPW	BAYVL	01C	24	19	405423	733505	NC04	9	0	1	TOB	OB
N-09480	TNH LANDFILL	PT WASH	5	268	258	404018	734902	PW08	170	0	2	TNH	OB
N-09481	TNH LANDFILL	PT WASH	6	166	156	404018	734902	PW08	170	0	2	TNH	OB
N-09482	TNH LANDFILL	PT WASH	7	53	43	404004	734906	PW08	---	0	1	TNH	OB
N-09483	TNH LANDFILL	PT WASH	8	57	52	404004	734906	PW08	---	0	1	TNH	OB
N-09488	HICKSVILLE W D	HKSVL	1-6	575	515	404639	733111	NC03	165	1380	2	TOB	YR
N-09514	N Y WATER SERVICE	NO WTGH	4J	660	569	404130	733114	NC03	40	2100	2	TOH	YR
N-09520	OYSTER BAY W D	O BAY	6-2	512	451	405146	733134	OB14	80	1200	2	TOB	YR
N-09521	ROOSEVELT FIELD W D	G C EST	7	603	475	404413	733611	NC03	80	1350	2	TOH	YR
N-09589	LEVITTOWN MEM H S	LEVITT	4	63	53	404308	733034	NC03	70	200	1	TOH	SS
N-09590	LEVITTOWN DIV H S	LEVITT	2	51	41	404219	733023	NC03	60	200	1	TOH	SS
N-09591	BETHPAGE W D	BTHPG	BGD-1	682	616	404524	732825	NC03	120	1380	2	TOB	YR
N-09613	LONG ISLAND W C	V S	7-1A	480	424	404036	734314	NC02	27	1400	2	TOH	YR
N-09657	NCDPW	SEAFORD	P17A	32	27	404026	732904	NC03	20	0	1	TOH	OB

HAN SITE

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-09658	NCDPW	NO WTGH	P14A	53	48	404219	732937	NC03	55	0	1	TOH	OB
N-09661	NCDPW	FRMGDL	T-12A	57	52	------	------	NC03	---	0	2	TOB	OB
N-09667	-------	----	--	---	---	------	------	----	---	---	--	---	--
N-09687	FRESH MEADOWS C C	LK SUCC	1	243	203	404651	734215	NC04	90	800	2	TNH	SS
N-09703	NCDPW	GDN CTY	X156	106	96	404337	733626	NC03	87	0	2	TOH	OB
N-09709	TOH RES RECOVERY	G C EST	1	363	305	404416	733521	NC03	89	1000	2	TOH	OB
N-09711	NCDPW	ROS HTS	F2A	146	141	404712	733850	RD10	160	0	2	TNH	OB
N-09712	NCDPW	ALBTSN	F3A	158	153	404620	733837	NC02	140	0	2	TNH	OB
N-09713	NCDPW	GDN CTY	X157	215	205	404337	733626	NC03	87	0	2	TOH	OB
N-09751	TOH RES RECOVERY	G C EST	2	215	205	404416	733521	NC03	88	1000	2	TOH	OB
N-09753	TOB LANDFILL	O BTHPG	G9	81	76	404516	732643	NC03	116	0	1	TOB	OB
N-09754	TOB LANDFILL	O BTHPG	G10	64	59	404516	732643	NC03	116	0	1	TOB	OB
N-09757	TOB LANDFILL	O BTHPG	I13	60	55	404529	732643	NC03	117	0	1	TOB	OB
N-09768	GARDEN CITY PARK W D	G C PK	10	477	404	404547	734011	NC02	145	1200	2	TNH	YR
N-09792	ROCKVILLE CTR VILL	RVC	4	537	466	403932	733827	NC02	20	1500	2	TOH	YR
N-09802	NCDPW	----	X-175	146	141	------	------	----	---	---	2	---	OB
N-09803	NCDPW	----	X-176	62	57	------	------	----	---	---	1	---	OB
N-09804	NCDPW	----	X-177	143	138	------	------	----	---	---	2	---	OB
N-09805	NCDPW	----	X-178	61	56	------	------	----	---	---	1	---	OB
N-09806	WOODCREST C C	MUTNTWN	2	239	196	404922	733031	NC03	220	1000	1	TOB	YR
N-09809	PORT WASHINGTON W D	FLWR HL	S10	524	438	404840	734045	PW08	120	1350	1	TNH	YR
N-09846	ROOSEVELT FIELD W D	G C EST	10	594	508	404414	733513	NC03	82	1350	2	TOH	YR
N-09878	N Y WATER SERVICE	NO BLMR	4N	664	560	404117	733230	NC03	40	2100	2	TOH	YR
N-09892	NORTHWEST 201	G N EST	NW-1	45	35	404713	734454	NC04	32	0	1	TNH	OB
N-09893	NORTHWEST 201	KNGS PT	NW-2	70	60	404814	734509	NC04	59	0	1	TNH	OB
N-09894	NORTHWEST 201	KNGS PT	NW-3	35	25	404908	734437	NC04	42	0	1	TNH	OB
N-09895	NORTHWEST 201	KNGS PT	NW-4	44	34	404916	734346	NC04	37	0	1	TNH	OB
N-09896	NORTHWEST 201	KNGS PT	NW-5	90	80	404843	734327	NC04	106	0	1	TNH	OB
N-09897	NORTHWEST 201	MANHST	NW-6	90	80	404726	734155	NC04	96	0	1	TNH	OB
N-09898	NORTHWEST 201	MANHST	NW-7	110	100	404744	734208	NC04	118	0	1	TNH	OB
N-09899	NORTHWEST 201	PLANDME	NW-8	35	25	404827	734223	NC04	43	0	1	TNH	OB
N-09900	NORTHWEST 201	PLDM MR	NW-9	35	15	404840	734143	NC04	51	0	1	TNH	OB
N-09901	NORTHWEST 201	PLDM HT	NW-10	50	30	404810	734149	NC04	55	0	1	TNH	OB
N-09902	NORTHWEST 201	PLANDME	NW-11	100	80	404817	734135	NC04	133	0	1	TNH	OB
N-09903	NORTHWEST 201	FLWR HL	NW-12	120	110	404838	734047	NC04	140	0	1	TNH	OB
N-09904	NORTHWEST 201	MNSY PK	NW-13	160	140	404744	734051	NC04	166	0	1	TNH	OB
N-09905	NORTHWEST 201	FLWR HL	NW-14	130	110	404831	733954	NC04	141	0	1	TNH	OB
N-09906	NORTHWEST 201	FLWR HL	NW-15	125	95	404805	734010	NC04	168	0	1	TNH	OB
N-09907	NORTHWEST 201	ROS EST	NW-16	145	115	404747	733956	NC04	176	0	1	TNH	OB
N-09910	N Y WATER SERVICE	WANTAGH	60	774	695	404052	732948	NC03	20	2100	2	TOH	YR
N-09914	NCDPW	GDN CTY	X183	57	53	404409	733741	NC02	99	0	1	TOH	OB
N-09917	NCDPW	HKSVL	O10A	76	71	404525	733251	NC03	125	0	1	TOB	OB
N-09918	NCDPW	HKSVL	X97A	77	72	404436	733057	NC03	112	0	1	TOB	OB
N-09919	NCDPW	HKSVL	X98B	84	79	404537	733148	NC03	136	0	1	TOB	OB
N-09920	NCDPW	HKSVL	P9D	89	84	404609	733030	NC03	146	0	1	TOB	OB
N-09921	NCDPW	BTHPG	P11C	62	57	404421	732945	NC03	94	0	1	TOH	OB
N-09922	NCDPW	LEVITT	P23	41	36	404341	732950	NC03	77	0	1	TOH	OB
N-09923	NCDPW	FRMGDL	U16	43	38	404349	732608	NC03	67	0	1	TOB	OB
N-09924	NCDPW	LEVITT	UP4	45	40	404322	733055	NC03	77	0	1	TOH	OB
N-09925	NCDPW	E MEAD	U13B	51	46	404326	733220	NC03	86	0	1	TOH	OB
N-09926	NCDPW	JERICHO	UP1	130	125	404720	733151	NC03	168	0	1	TOB	OB
N-09927	NCDPW	HKSVL	UP3	94	89	404632	733121	NC03	162	0	1	TOB	OB
N-09928	NCDPW	HKSVL	UP2	86	81	404626	733216	NC03	145	0	1	TOB	OB
N-09929	NCDPW	BTHPG	PT6	40	35	404341	732839	NC03	86	0	1	TOB	OB

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-09930	NCDPW	FRMGDL	TU4	46	41	404345	732731	NC03	76	0	1	TOB	OB
N-09931	NCDPW	BTHPG	PT5	73	68	404501	732918	NC03	119	0	1	TOB	OB
N-09932	NCDPW	PLNVIEW	PT4	105	100	404604	732920	NC03	146	0	1	TOB	OB
N-09933	NCDPW	PLNVIEW	PT2	115	110	404720	733000	NC03	179	0	1	TOB	OB
N-09934	NCDPW	PLNVIEW	TU1	127	122	404729	732933	NC03	174	0	1	TOB	OB
N-09935	NCDPW	O BTHPG	TU2	135	130	404624	732734	NC03	179	0	1	TOB	OB
N-09936	NCDPW	O BTHPG	TU3	63	58	404459	732701	NC03	93	0	1	TOB	OB
N-09937	NCDPW	SYOSSET	PT1	98	93	404742	733017	NC03	190	0	1	TOB	OB
N-09938	NCDPW	WSTBY	HO1	80	75	404528	733336	NC03	124	0	1	TNH	OB
N-09939	NCDPW	BOWL GR	HO2	74	69	404437	733344	NC03	105	0	1	TOH	OB
N-09940	NCDPW	CAR PL	G23	53	48	404525	733630	NC03	107	0	1	TNH	OB
N-09941	NCDPW	CAR PL	G22	50	45	404443	733625	NC03	86	0	1	TNH	OB
N-09942	NCDPW	MINEOLA	F5A	69	64	404455	733814	NC02	113	0	1	TNH	OB
N-09943	NCDPW	GDN CTY	F7A	69	64	404343	733805	NC02	90	0	1	TOH	OB
N-09944	NCDPW	G C PK	E9B	80	75	404412	734004	NC02	96	0	1	TNH	OB
N-09945	NCDPW	GDN CTY	E11A	67	62	404241	733956	NC02	76	0	1	TOH	OB
N-09946	NCDPW	HERRCKS	X20A	60	55	404533	733935	NC02	106	0	1	TNH	OB
N-09947	NCDPW	BEL TER	X1A	109	104	404321	734329	NC02	81	0	1	TOH	OB
N-09948	NCDPW	N H P	DE1	114	109	404508	734057	NC02	125	0	1	TNH	OB
N-09949	NCDPW	N H P	DE2	100	95	404417	734059	NC02	107	0	1	TNH	OB
N-09950	NCDPW	WSTBY	GH1	69	64	404514	733535	NC03	112	0	1	TNH	OB
N-09951	R FIELD-NCDH	GDN CTY	RF-1	44	38	404431	733703	NC02	95	0	1	TOH	OB
N-09951A	R FIELD-NCDH	GDN CTY	RF-1	55	49	404431	733703	NC02	95	0	1	TOH	AB
N-09952	R FIELD-NCDH	GDN CTY	RF-2	44	38	404427	733704	NC02	91	0	1	TOH	OB
N-09952A	R FIELD-NCDH	GDN CTY	RF-2	55	49	404427	733704	NC02	91	0	1	TOH	AB
N-09953	R FIELD-NCDH	GDN CTY	RF-3	55	49	404421	733705	NC02	94	0	1	TOH	OB
N-09954	R FIELD-NCDH	GDN CTY	RF-4	55	49	404407	733704	NC02	87	0	1	TOH	OB
N-09955	R FIELD-NCDH	GDN CTY	RF-5	55	49	404406	733657	NC02	83	0	1	TOH	OB
N-09956	R FIELD-NCDH	G C EST	RF-6	55	49	404420	733644	NC03	91	0	1	TOH	OB
N-09957	R FIELD-NCDH	G C EST	RF-7	55	49	404423	733654	NC03	93	0	1	TOH	OB
N-09958	R FIELD-NCDH	G C EST	RF-8	55	49	404416	733654	NC03	90	0	1	TOH	OB
N-09959	R FIELD-NCDH	G C EST	RF-9	55	49	404412	733634	NC03	83	0	1	TOH	OB
N-09960	R FIELD-NCDH	G C EST	RF-10	55	49	404423	733639	NC03	89	0	1	TOH	OB
N-09961	R FIELD-NCDH	G C EST	RF-11	55	49	404433	733643	NC03	84	0	1	TOH	OB
N-09962	R FIELD-NCDH	CAR PL	RF-13	65	59	404446	733724	NC03	101	0	1	TNH	OB
N-09963	R FIELD-NCDH	CAR PL	RF-14	55	49	404442	733649	NC03	91	0	1	TNH	OB
N-09964	R FIELD-NCDH	GDN CTY	RF-15	55	49	404403	733706	NC02	87	0	1	TOH	OB
N-09965	R FIELD-NCDH	GDN CTY	RF-16	44	38	404413	733704	NC02	93	0	1	TOH	OB
N-09965A	R FIELD-NCDH	GDN CTY	RF-16	55	49	404413	733707	NC02	90	0	1	TOH	AB
N-09966	R FIELD-NCDH	GDN CTY	RF-17	55	49	404358	733656	NC02	82	0	1	TOH	OB
N-09967	R FIELD-NCDH	G C EST	RF-18	55	49	404404	733631	NC03	83	0	1	TOH	OB
N-09968	R FIELD-NCDH	GDN CTY	RF-19	65	49	404432	733712	NC02	112	0	1	TOH	OB
N-09971	LEVER BLDG	GDN CTY	1	41	36	404348	733713	NC02	83	--	1	TOH	OB
N-09972	LEVER BLDG	GDN CTY	2	41	36	404348	733713	NC02	83	0	1	TOH	OB
N-09973	LEVER BLDG	GDN CTY	3	41	36	404348	733713	NC02	83	0	1	TOH	OB
N-09974	LEVER BLDG	GDN CTY	4	41	36	404348	733713	NC02	83	0	1	TOH	OB
N-09976	N Y WATER SERVICE	NO MRK	2C	567	440	404101	733419	NC03	30	2100	2	TOH	YR
N-09977	TNH LANDFILL	PT WASH	10	196	172	404028	734859	PW08	180	0	2	TNH	OB
N-09978	TNH LANDFILL	PT WASH	9	246	222	404028	734859	PW08	180	0	2	TNH	OB
N-09979	NCDPW	ELMONT	X2A	95	90	404233	734326	NC02	71	0	1	TOH	OB
N-09980	NCDPW	FRMGDL	U7A	58	53	404423	732623	NC03	80	0	1	TOB	OB
N-09981	NCDPW	PLNVIEW	PT3	108	103	404644	732955	NC03	166	0	1	TOB	OB
N-09982	NCDPW	N H P	D4A	112	107	404443	734203	NC02	120	0	1	TNH	OB
N-09983	NCDPW	FL PK	D5A	99	94	404408	734156	NC02	107	0	1	TOH	OB

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-09984	NCDPW	FR SQ	UE3	60	55	404252	734046	NC02	78	0	1	TOH	OB
N-10011	NCDPW	SO HEMP	X34A	26	21	403950	733616	NC02	18	0	1	TOH	OB
N-10019	R FIELD-USGS	GDN CTY	--	227	223	404407	733704	NC02	87	0	2	TOH	OB
N-10020	R FIELD-USGS	GDN CTY	--	190	185	404406	733657	NC02	83	0	2	TOH	OB
N-10024	TOB LANDFILL	O BTHPG	3-1	102	97	404514	732652	NC03	112	10	2	TOB	OB
N-10025	TOB LANDFILL	O BTHPG	3-2	110	105	404521	732643	NC03	116	10	2	TOB	OB
N-10026	TOB LANDFILL	O BTHPG	3-3	110	105	404529	732643	NC03	130	10	2	TOB	OB
N-10027	TOB LANDFILL	O BTHPG	3-4	118	113	404531	732716	NC03	146	10	2	TOB	OB
N-10033	GARDEN CITY VILLAGE	GDN CTY	15	541	439	404303	733808	NC02	75	1380	2	TOH	YR
N-10034	GARDEN CITY VILLAGE	GDN CTY	16	570	489	404303	733808	NC02	75	1380	2	TOH	YR
N-10035	R FIELD-USGS	GDN CTY	--	56	51	404338	733715	NC02	80	0	1	TOH	OB
N-10038	MITCHEL FIELD W D	E MEAD	1	605	201	404403	733350	NC03	96	1404	2	TOH	YR
N-10039	MITCHEL FIELD W D	E MEAD	2	588	262	404356	733351	NC03	95	1397	2	TOH	YR
N-10040	MITCHEL FIELD W D	E MEAD	3	606	270	404345	733356	NC03	89	1336	2	TOH	YR
N-10043	GEORGIAN WEB OFFSET	G C EST	2	185	165	404421	733617	NC03	89	350	2	TOH	SS
N-10045	TOB LANDFILL-NCDH	LOC GRV	SY-1	135	125	404757	733037	NC03	195	0	2	TOB	OB
N-10046	TOB LANDFILL-NCDH	LOC GRV	SY-2	125	115	404801	733033	NC03	182	0	2	TOB	OB
N-10047	TOB LANDFILL-NCDH	LOC GRV	SY-3	145	135	404805	733044	NC03	191	0	2	TOB	OB
N-10048	TOB LANDFILL-NCDH	LOC GRV	SY-4	153	143	404759	733048	NC03	193	0	2	TOB	OB
N-10049	TOB LANDFILL-NCDH	LOC GRV	SY-5	135	125	404756	733037	NC03	178	0	2	TOB	OB
N-10050	TOB LANDFILL-NCDH	LOC GRV	SY-6	145	135	404751	733031	NC03	186	0	2	TOB	OB
N-10051	TOB LANDFILL-NCDH	LOC GRV	SY-7	145	135	404750	733025	NC03	199	0	2	TOB	OB
N-10056	TNH LANDFILL-NCDH	N H P	DA-1	120	110	404459	734041	NC02	109	0	1	TNH	OB
N-10057	TNH LANDFILL-NCDH	N H P	DA-2	95	85	404457	734041	NC02	110	0	1	TNH	OB
N-10058	TNH LANDFILL-NCDH	N H P	DA-3	100	90	404458	734049	NC02	122	0	1	TNH	OB
N-10059	TNH LANDFILL-NCDH	N H P	DA-4	95	85	404442	734043	NC02	109	0	1	TNH	OB
N-10060	TNH LANDFILL-NCDH	N H P	DA-5	114	104	404439	734042	NC02	110	0	1	TNH	OB
N-10076	ISLAND INN	G C EST	1R	334	207	404443	733613	NC03	120	350	2	TOH	SS
N-10084	NCDPW	BELMR	X38B	21	16	403939	733222	NC03	5	0	1	TOH	OB
N-10094	R FIELD-USGS	GDN CTY	--	68	63	404416	733720	NC02	80	0	1	TOH	OB
N-10095	R FIELD-USGS	GDN CTY	--	50	47	404417	733711	NC02	99	0	1	TOH	OB
N-10095A	R FIELD-USGS	GDN CTY	--	66	61	404417	733711	NC02	99	0	1	TOH	AB
N-10096	R FIELD-USGS	GDN CTY	--	36	35	404403	733658	NC02	81	0	1	TOH	OB
N-10096A	R FIELD-USGS	GDN CTY	--	56	51	404403	733658	NC02	81	0	1	TOH	AB
N-10097	R FIELD-USGS	GDN CTY	--	36	35	404359	733701	NC02	82	0	1	TOH	OB
N-10097A	R FIELD-USGS	GDN CTY	--	56	51	404359	733701	NC02	82	0	1	TOH	AB
N-10103	LONG ISLAND W C	MLVRN	23-2	518	448	404023	733713	NC02	35	1380	2	TOH	YR
N-10144	BAYVILLE VILLAGE	BAYVL	2-1	374	329	405421	733239	NC04	5	1000	3	TOB	YR
N-10145	BAYVILLE VILLAGE	BAYVL	0	---	---	------	------	NC04	---	---	3	TOB	OB
N-10149	JERICHO W D	SYOSSET	20	625	535	405018	732845	NC04	220	1380	2	TOB	YR
N-10195	N Y WATER SERVICE	NO WTGH	5J	585	512	404130	733114	NC03	40	2100	2	TOH	YR
N-10202	R FIELD-USGS	GDN CTY	--	45	42	404348	733724	NC02	88	0	1	TOH	OB
N-10204	R FIELD-USGS	GDN CTY	--	44	41	404352	733657	NC02	80	0	1	TOH	OB
N-10205	R FIELD-USGS	GDN CTY	--	44	41	404340	733707	NC02	83	0	1	TOH	OB
N-10206	JAMAICA W S CO	ELMONT	15C	440	374	404224	734240	NC02	60	1380	2	TOH	YR
N-10207	JAMAICA W S CO	ELMONT	15E	450	379	404224	734240	NC02	60	1380	2	TOH	YR
N-10208	HICKSVILLE W D	HKSVL	9-3	649	572	404537	733046	NC03	140	1750	2	TOB	YR
N-10211	JAMAICA W S CO	ELMONT	28B	494	423	404125	734209	NC02	55	1380	2	TOH	YR
N-10252	NCDPW	G C PK	EBA	104	99	------	------	NC02	---	0	--	TNH	OB
N-10286	LONG ISLAND W C	V S	9-2A	535	484	404031	734144	NC02	25	1600	2	TOH	YR
N-10299	R FIELD-USGS	GDN CTY	--	54	51	404435	733656	NC02	94	0	1	TOH	OB
N-10306	NCDH	NO HKSVL	NH-1S	116	106	404729	733059	NC03	173	0	--	TOB	OB
N-10307	NCDH	NO HKSVL	NH-1D	212	192	404729	733059	NC03	172	0	--	TOB	OB
N-10308	NCDH	NO HKSVL	NH-2S	99	89	404650	733114	NC03	166	0	--	TOB	OB

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-10309	NCDH	NO HKSVL	NH-2D	210	190	404650	733114	NC03	167	0	--	TOB	OB
N-10310	NCDH	NO HKSVL	NH-3S	105	95	404650	733117	NC03	163	0	--	TOB	OB
N-10311	NCDH	NO HKSVL	NH-3D	225	205	404650	733117	NC03	164	0	--	TOB	OB
N-10312	NCDH	W HKSVL	WH-1	60	50	------	------	NC03	125	0	--	TOB	OB
N-10313	NCDH	W HKSVL	WH-2	63	53	404537	733236	NC03	131	0	--	TOB	OB
N-10314	NCDH	W HKSVL	WH-3	64	54	404538	733228	NC03	140	0	--	TOB	OB
N-10315	NCDH	W HKSVL	WH-4	66	56	404553	733226	NC03	134	0	--	TOB	OB
N-10316	NCDH	W HKSVL	WH-5	72	62	404552	733211	NC03	135	0	--	TOB	OB
N-10317	NCDH	W HKSVL	WH-6	64	54	404526	733243	NC03	126	0	--	TOB	OB
N-10318	NCDH	NEW CSL	NC-1	60	50	404525	733402	NC03	119	0	--	TNH	OB
N-10319	NCDH	NEW CSL	NC-2S	57	47	404526	733355	NC03	126	0	--	TNH	OB
N-10320	NCDH	NEW CSL	NC-3	60	50	404527	733349	NC03	122	0	--	TNH	OB
N-10321	NCDH	NEW CSL	NC-4	62	52	404528	733340	NC03	124	0	--	TNH	OB
N-10322	NCDH	NEW CSL	NC-5	67	57	404532	733331	NC03	126	0	--	TNH	OB
N-10323	NCDH	NEW CSL	NC-6	62	52	404536	733313	NC03	127	0	--	TNH	OB
N-10324	NCDH	NEW CSL	NC-7	57	47	404514	733405	NC03	117	0	--	TNH	OB
N-10325	NCDH	NEW CSL	NC-8	57	47	------	------	NC03	189	0	--	TNH	OB
N-10326	NCDH	NEW CSL	NC-9	59	49	------	------	NC03	119	0	--	TNH	OB
N-10327	NCDH	NEW CSL	NC-10	58	48	------	------	NC03	119	0	--	TNH	OB
N-10328	NCDH	NEW CSL	NC-11	58	48	------	------	NC03	118	0	--	TNH	OB
N-10329	NCDH	NEW CSL	NC-12	54	44	------	------	NC03	123	0	--	TNH	OB
N-10330	NCDH	G C PK	GCP-1	59	49	------	------	NC03	90	0	--	TNH	OB
N-10331	NCDH	G C PK	GCP-2	59	49	------	------	NC03	101	0	--	TNH	OB
N-10332	NCDH	N H P	NHP-1	60	50	------	------	NC03	79	0	--	TNH	OB
N-10333	NCDH	N H P	NHP-2	63	53	------	------	NC03	97	0	--	TNH	OB
N-10334	NCDH	N H P	NHP-3	50	40	------	------	NC03	79	0	--	TNH	OB
N-10401	WEST HEMPSTEAD W D	W HEMP	10	625	600	404140	733841	NC02	---	1438	2	TNH	YR
N-10408	WEST HEMPSTEAD W D	W HEMP	9	615	600	404140	733841	NC02	---	1515	2	TNH	YR
N-10451	WESTBURY W D	WSTBY	17	512	418	404548	733609	NC03	---	1400	2	TNH	YR
N-10458	NCDH	NEW CSL	NC-2D	120	100	404526	733355	NC03	121	0	--	TNH	OB
N-10459	NCDH	NEW CSL	NC-13	68	58	------	------	NC03	129	0	--	TNH	OB
N-10460	NCDH	NEW CSL	NC-14	68	58	404542	733323	NC03	131	0	--	TNH	OB
N-10461	NCDH	NEW CSL	NC-15	66	56	404535	733343	NC03	125	0	--	TNH	OB
N-10462	NCDH	NEW CSL	NC-16	64	54	404533	733350	NC03	123	0	--	TNH	OB
N-10463	NCDH	NEW CSL	NC-17	64	54	404526	733352	NC03	122	0	--	TNH	OB
N-10464	NCDH	NEW CSL	NC-18	60	50	404518	733401	NC03	117	0	--	TNH	OB
N-10465	NCDH	NEW CSL	NC-19	62	52	404526	733352	NC03	121	0	--	TNH	OB
N-10466	NCDH	NEW CSL	NC-20	60	50	404515	733353	NC03	118	0	--	TNH	OB
N-10467	NCDH	NEW CSL	NC-21	63	53	404526	733343	NC03	125	0	--	TNH	OB
N-10468	NCDH	NEW CSL	NC-22D	125	105	404532	733337	NC03	125	0	--	TNH	OB
N-10469	NCDH	NEW CSL	NC-23	64	54	404527	733330	NC03	123	0	--	TNH	OB
N-10470	NCDH	NEW CSL	NC-24	65	55	404513	733335	NC03	120	0	--	TNH	OB
N-10471	NCDH	NEW CSL	NC-25	60	50	404527	733334	NC03	119	0	--	TNH	OB
N-10472	NCDH	NEW CSL	NC-26S	62	52	404509	733334	NC03	113	0	--	TNH	OB
N-10473	NCDH	NEW CSL	NC-26D	120	100	404509	733334	NC03	111	0	--	TNH	OB
N-10474	NCDH	NEW CSL	NC-27	60	50	404511	733334	NC03	118	0	--	TNH	OB
N-10475	NCDH	NEW CSL	NC-28S	57	47	------	------	NC03	110	0	--	TNH	OB
N-10476	NCDH	NEW CSL	NC-28D	130	110	------	------	NC03	111	0	--	TNH	OB
N-10477	NCDH	NEW CSL	NC-29S	57	47	404504	733416	NC03	111	0	--	TNH	OB
N-10478	NCDH	NEW CSL	NC-29D	121	101	404504	733416	NC03	111	0	--	TNH	OB
N-10479	NCDH	NEW CSL	NC-30S	40	30	404447	733347	NC03	93	0	--	TNH	OB
N-10480	NCDH	NEW CSL	NC-30D	118	98	404447	733347	NC03	93	0	--	TNH	OB
N-10481	NCDH	G C PK	GCP-3	40	30	------	------	NC02	89	0	--	TNH	OB
N-10482	NCDH	G C PK	GCP-4	61	51	------	------	NC02	100	0	--	TNH	OB

12

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NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-10483	NCDH	G C PK	GCP-5	59	49	------	------	NC02	95	0	--	TNH	OB
N-10484	NCDH	G C PK	GCP-6	55	45	------	------	NC02	95	0	--	TNH	OB
N-10485	NCDH	G C PK	GCP-7	65	55	------	------	NC02	98	0	--	TNH	OB
N-10486	NCDH	G C PK	GCP-8	60	50	------	------	NC02	95	0	--	TNH	OB
N-10487	NCDH	G C PK	GCP-9	61	51	------	------	NC02	93	0	--	TNH	OB
N-10555	HICKSVILLE W D	HKSVL	11-1	---	---	------	------	NC03	---	---	--	TOB	YR
N-10557	MANHASSET-LAKEVL W D	NO HLS	25	408	321	404632	734017	NC04	---	1400	2	TNH	YR
N-10588	BETHPAGE USGS	HKSVL	--	76	71	404457	733124	NC03	---	0	--	TOB	OB
N-10589	BETHPAGE USGS	HKSVL	--	76	73	404527	733052	NC03	---	0	--	TOB	OB
N-10590	BETHPAGE USGS	BTHPG	--	76	73	404548	732935	NC03	---	0	--	TOB	OB
N-10591	BETHPAGE USGS	BTHPG	--	78	72	404543	732914	NC03	---	0	--	TOB	OB
N-10592	BETHPAGE USGS	BTHPG	--	73	67	404521	732833	NC03	---	0	--	TOB	OB
N-10593	BETHPAGE USGS	HKSVL	--	77	73	------	------	NC03	---	0	--	TOB	OB
N-10594	BETHPAGE USGS	HKSVL	--	76	73	404518	733007	NC03	---	0	--	TOB	OB
N-10595	BETHPAGE USGS	BTHPG	--	67	63	404500	732938	NC03	---	0	--	TOB	OB
N-10596	BETHPAGE USGS	HKSVL	--	71	68	404502	733014	NC03	---	0	--	TOB	OB
N-10597	BETHPAGE USGS	HKSVL	--	66	63	404454	733023	NC03	---	0	--	TOB	OB
N-10598	BETHPAGE USGS	HKSVL	--	77	73	404441	733021	NC03	---	0	--	TOB	OB
N-10599	BETHPAGE USGS	HKSVL	--	67	63	404432	732950	NC03	---	0	--	TOB	OB
N-10600	BETHPAGE USGS	BTHPG	--	61	57	404425	733002	NC03	---	0	--	TOB	OB
N-10601	BETHPAGE USGS	BTHPG	--	67	63	404426	732929	NC03	---	0	--	TOB	OB
N-10602	BETHPAGE USGS	BTHPG	--	56	52	404404	732937	NC03	---	0	--	TOB	OB
N-10603	BETHPAGE USGS	LEVITT	--	61	57	404357	732948	NC03	---	0	--	TOH	OB
N-10612	GARDEN CITY PARK W D	----	11	400	---	------	------	NC02	---	1200	--	TNH	YR
N-10623	BETHPAGE USGS	BTHPG	--	72	71	404508	732933	NC03	---	0	--	TOB	OB
N-10624	BETHPAGE USGS	BTHPG	--	194	71	404415	732941	NC03	122	0	--	TOB	OB
N-10625	BETHPAGE USGS	BTHPG	--	67	63	404454	732930	NC03	116	0	--	TOB	OB
N-10626	BETHPAGE USGS	BTHPG	--	67	63	404446	732914	NC03	119	0	--	TOB	OB
N-10627	BETHPAGE USGS	BTHPG	--	310	290	404415	732941	NC03	94	0	--	TOB	OB
N-10628	BETHPAGE USGS	BTHPG	--	67	63	404420	732903	NC03	101	0	--	TOB	OB
N-10629	BETHPAGE USGS	BTHPG	--	109	105	404504	732937	NC03	116	0	--	TOB	OB
N-10630	BETHPAGE USGS	HKSVL	--	300	290	404454	733023	NC03	111	0	--	TOB	OB
N-10631	BETHPAGE USGS	BTHPG	--	67	63	404428	732945	NC03	103	0	--	TOB	OB
N-10632	BETHPAGE USGS	BTHPG	--	67	63	404427	732936	NC03	104	0	--	TOB	OB
N-10633	BETHPAGE USGS	BTHPG	--	67	63	404423	732922	NC03	104	0	--	TOB	OB
N-10634	BETHPAGE USGS	BTHPG	--	67	63	404414	732924	NC03	101	0	--	TOB	OB
N-10635	BETHPAGE USGS	BTHPG	--	45	45	404403	732944	NC03	89	0	--	TOB	OB
N-10636	BETHPAGE USGS	LEVITT	--	56	52	404413	733024	NC03	96	0	--	TOB	OB
N-10662	BETHPAGE ST PARK	BTHPG	6	---	---	------	------	NC03	---	---	2	TOB	--
N-10812	BETHPAGE USGS	HKSVL	--	93	---	------	------	NC03	---	0	--	---	OB
N-10813	BETHPAGE USGS	LEVITT	--	67	---	------	------	NC03	---	0	--	---	OB
N-10814	BETHPAGE USGS	BTHPG	--	72	---	------	------	NC03	---	0	--	---	OB
N-10815	BETHPAGE USGS	LEVITT	--	61	---	------	------	NC03	---	0	--	---	OB
N-10816	BETHPAGE USGS	BTHPG	--	130	---	------	------	NC03	---	0	--	---	OB
N-10817	BETHPAGE USGS	BTHPG	--	51	---	------	------	NC03	---	0	--	---	OB
N-10818	BETHPAGE USGS	BTHPG	--	56	---	------	------	NC03	---	0	--	---	OB
N-10819T	BETHPAGE USGS	BTHPG	--	0	---	------	------	NC03	---	0	--	---	OB
N-10820	BETHPAGE USGS	BTHPG	--	72	---	------	------	NC03	---	0	--	---	OB
N-10821	BETHPAGE USGS	BTHPG	--	56	---	------	------	NC03	---	0	--	---	OB
N-10822	BETHPAGE USGS	LEVITT	--	130	---	------	------	NC03	---	0	--	---	OB
N-10863	N Y WATER SERVICE	MSPQ E	UM	685	604	404056	732611	NC03	25	2100	2	TOB	YR
N-10889	MANHASSET-LAKEVL W D	----	26	---	---	------	------	----	---	---	--	TNH	YR
N-10997	BETHPAGE USGS	----	--	---	---	------	------	NC03	---	0	--	---	OB
N-10998	BETHPAGE USGS	----	--	---	---	------	------	NC03	---	0	--	---	OB

NASSAU COUNTY WELL LISTING

NYS WELL NO.	WATER DISTRICT	COMMUNITY	LOCAL NO.	WELL DEPTH	SCREEN DEPTH	LATUDE	LGTUDE	SEWER DIST	L.S. ELEV	CAP	AQ	TOWN	ST
N-10999	BETHPAGE USGS	----	--	---	---	------	------	NC03	---	0	--	---	OB
N-11UNK1	SEAWANHAKA Y C	CTR ISL	1	450	---	405409	733054	NC04	20	30	3	TOB	YR
N-11UNK2	SPLITROCK W S	LRL HLW	1	---	---	------	------	NC04	---	---	2	TOB	YR
N-11000	BETHPAGE USGS	----	--	---	---	------	------	NC03	---	0	--	---	OB
N-11107	JERICHO W D	----	29	585	520	------	------	NC04	---	1200	2	TOB	YR

APPENDIX J

Waste Manifests

ANSON
ENVIRONMENTAL LTD.

33 Gerard Street • Suite 100
Huntington, New York 11743
516•351•3555
Fax 516•351•3615

July 1, 1993

Tom Taccone
New York/Caribbean Compliance Branch
U.S. Environmental Protection Agency
26 Federal Plaza, Room 747
New York, New York 10278

Re: Monthly Report for June, 1993
Anchor Chemical Superfund Site
Administrative Order No. II CERCLA-90208

Dear Mr. Taccone,

In accordance with the above referenced Administrative Order, Anson Environmental Ltd. and Blasland Bouck & Lee (BB&L), are submitting this monthly progress report on activities conducted at the Anchor Chemical Site in Hicksville, New York during the month of June.

On June 1st, the water in the remaining drums on site were pumped out by Long Island Cesspool Sewer and Drain under the auspices of NCDPW Permit #41 and NYSDEC permit #1A209. A total of 1400 gallons of water was pumped and removed by E&B Industrial Cleaning. Please find enclosed the waste manifest documents for the above referenced disposal.

One drum of material remains on site which contains oil-sorb used in the cleaning of Tank 10 by Enroserve and Roux Associates in 1991. It is being classified for proper disposal.

During June, additional clarification of the groundwater direction was forwarded to your office for evaluation of the Remedial Investigation report. Anson Environmental has cntinued to respond to verbal questions from the New York State Department of Environmental Conservation. Report modifications will be made upon receipt of comments from all regulatory agencies.

Efforts to keep this project moving forward are the goals of Anson Environmental and BB&L.

Very truly yours,

Dean Anson II
Co-Facility Coordinator

cc: M. Chen, NYSDEC (Albany)
A. Candella, NYSDEC (Stony Brook)
F. Werfel, Spiegel Associates
A. Sanders, Spiegel Associates
R. Leland, Esq., Rosenman and Colin
J. Doyle, Esq., USEPA
J. O'Brien, Esq., Anchor Chemical
J. Tillotson, Newsday

302266

NON-HAZARDOUS WASTE MANIFEST

1. Generator's US EPA ID No. EXEMPT
Manifest Document No.
2. Page 1 of 1

GENERATOR

3. Generator's Name and Mailing Address K.B. Co.
500 West John Street
Hicksville, NY
4. Generator's Phone ( )

5. Transporter 1 Company Name E+B Industrial Cleaning
6. US EPA ID Number NY-1A-209
7. Transporter 2 Company Name
8. US EPA ID Number
9. Designated Facility Name and Site Address Nassau County WPCP
Bay Park Plant
East Rockaway, NY
10. US EPA ID Number
A. Transporter's Phone 516-491-4301
B. Transporter's Phone
C. Facility's Phone

11. Waste Shipping Name and Description	12. Containers No.	Type	13. Total Quantity	14. Unit Wt/Vol
a. waste Non-hazardous Water	0.0.1	T.T	-.1.4.00	G
b.				
c.				
d.				

D. Additional Descriptions for Materials Listed Above
Pre-Approved as per letter dated May 24, 1993

E. Handling Codes for Wastes Listed Above

15. Special Handling Instructions and Additional Information

16. **GENERATOR'S CERTIFICATION:** I certify the materials described above on this manifest are not subject to federal regulations for reporting proper disposal of Hazardous Waste.
Printed/Typed Name Dean Anson for K.B. Co.
Signature
Month 06 Day 01 Year 93

TRANSPORTER

17. Transporter 1 Acknowledgement of Receipt of Materials
Printed/Typed Name ERIC HELMECKE
Signature Erich Helmecke
Month 06 Day 01 Year 93

18. Transporter 2 Acknowledgement of Receipt of Materials
Printed/Typed Name
Signature
Month Day Year

19. Discrepancy Indication Space

20. Facility Owner or Operator: Certification of receipt of waste materials covered by this manifest except as noted in Item 19.
Printed/Typed Name GEORGE C. HALLAM JR.
Signature George C. Hallam Jr
Month 6 Day 01 Year 93

2-BLS-C5 Rev. 9/92

ORIGINAL-RETURN TO GENERATOR

WASTE SYSTEMS, INC. Fax (716) 833-5670

NON-HAZARDOUS SOLID WASTE MANIFEST

TRANSPORTER Environmental Transport Group, Inc. Goldmine Road Flanders, New Jersey	**DATE** 05/18/93	**TIME IN**	**OUT**
	EWS # 1011		
TRUCK # 223	**TRAILER #** 403		

CONSIGNEE:
SUBURBAN RDF
STATE ROUTE 668-P.O. BOX 17
BROWNSVILLE, OHIO 43721
PHONE # (614) 787-2327

SHIPPER
~~ANSON ENVIRONMENTAL, LTD.~~
~~83 GARARD STREET, SUITE 100~~
~~HUNTINGTON, NEW YORK 11743~~
K.B. Co.
500 W. John St., Hicksville, N.Y.

NO. PIECES	**ARTICLES OR DESCRIPTION**		**WEIGHT**
		WEIGHT IN	
1 (63 Drums)	TRUCKLOAD NON-HAZARDOUS DRUMS FILLED WITH SOIL FROM DRILL CUTTING PROCESS. (SUBURBAN RDF APPROVAL NUMBER: 111980)	**WEIGHT OUT**	
		BILLED WEIGHT	

SHIPPER SIGNATURE ____________ **PRINT NAME** Dean Anson

DRIVER SIGNATURE ____________ **PRINT NAME** Steven C [illegible]

SPECIAL INSTRUCTIONS:

FOR APPROVAL:

CONSIGNEE PRINT NAME Steph Wilson 5-19-93

CONSIGNEE SIGN HERE (NO INITIALS) S Wilson

RECEIVED ABOVE MATERIAL IN GOOD CONDITION

FIRM ____________ DATE ____________

BY ____________ TIME ______ ☐ AM ______ ☐ PM ______

Solid waste being interpreted to mean only solid waste or waste containing animal and vegetable matter, rubbish, trash, debris, ashes and metal non-toxic sludge and other waste materials which is not a radioactive volatile, highly flammable explosive toxic or hazardous nature as listed.

The transporter herein named agrees to hold harmless and to indemnify EWS against all losses and claims as a result of shipment of any material not listed on this manifest.

1st Copy - Earthwatch 2nd Copy - Driver 3rd Copy - Land Fill 4th Copy Shipper

2

20244

Earthwatch
WASTE SYSTEMS, INC.

3527 Harlem Road
Buffalo, NY 14225
(716) 833-3266
Fax (716) 833-5670

NON-HAZARDOUS SOLID WASTE MANIFEST

TRANSPORTER Environmental Transport Group, Inc. Goldmine Road Flanders, New Jersey	**DATE** 05/18/93	**TIME IN**	**/ OUT**
	EWS # 1011		
TRUCK # 224	**TRAILER #** 147		

CONSIGNEE: SUBURBAN RDF STATE ROUTE 668-P.O. BOX 17 BROWNSVILLE, OHIO 43721 **PHONE #** (614) 787-2327	**SHIPPER** ~~ANSON ENVIRONMENTAL, LTD.~~ ~~33 GARARD STREET, SUITE 100~~ ~~HUNTINGTON, NEW YORK 11743~~ K.B. Co. 500 W. John St. Hicksville, New York

NO. PIECES	ARTICLES OR DESCRIPTION		WEIGHT
		WEIGHT IN	
1 (77 Drums)	TRUCKLOAD NON-HAZARDOUS DRUMS FILLED WITH SOIL FROM DRILL CUTTING PROCESS. (SUBURBAN RDF APPROVAL NUMBER: 111980)	**WEIGHT OUT**	
		BILLED WEIGHT	

SHIPPER SIGNATURE ________ **PRINT NAME** Dean Anson

DRIVER SIGNATURE Andrew T. Gancarcik **PRINT NAME** Andrew T. Gancarcik

SPECIAL INSTRUCTIONS:

FOR APPROVAL:

CONSIGNEE PRINT NAME Steph L Wilson 5-19-93

CONSIGNEE SIGN HERE (NO INITIALS) S. Wilson 5-19-93

RECEIVED ABOVE MATERIAL IN GOOD CONDITION

FIRM ________ DATE ________

BY ________ TIME ________ ☐ AM ________ ☐ PM ________

Solid waste being interpreted to mean only solid waste or waste containing animal and vegetable matter, rubbish, trash, debris, ashes and metal non-toxic sludge and other waste materials which is not a radioactive volatile, highly flammable explosive toxic or hazardous nature as listed.

The transporter herein named agrees to hold harmless and to indemnify EWS against all losses and claims as a result of shipment of any material not listed on this manifest.

APPENDIX K

Laboratory Resources Correspondence

Laboratory
Resources INC.

Teterboro Division
0 Hollister Road
terboro, New Jersey 07608
AX: 201-288-5311
201-288-3700 800-729-0852

September 23, 1993

Anson Environmental
33 Gerard Street
Huntington, N.Y. 11743

ATTN. Ms. Fritzi Gros-Daillon

Dear Fritzi,

This letter is in response to your inquiry concerning the compound bis (2-ethlyhexyl) phalate. This compound is associated with plastics and is also known as a common laboratory contaminant. This contaminant, when it occurs in the laboratory, is usually found inconsistently and at low concentrations. If you have questions, please contact me at 201-288-3700.

Sincerely,

Daniel Glenn
Senior Project Manager
LRI New Jersey Division

APPENDIX L

NYSDEC Bioremediation Protocol

New York State Department of Environmental Conservation
Building 40—SUNY, Stony Brook, New York 11790-2356
(516) 751-7725
Fax # (516) 751-3839

Thomas C. Jorling
Commissioner

September 15, 1992

CERTIFIED-RETURN RECEIPT REQUESTED

Anson Environmental
256 Main Street
Northport, NY 11768
Attn: Dean Anson II

Re:

Dear Mr. Anson:

As per our telephone conversation on September 8, 1992, enclosed is a copy of the "Draft In Situ Bioremediation--Minimum Specifications" Guide.

In order for this Department to consider your proposal for the above referenced site, the requirements in the draft guide must be adhered to in all aspects, or submit a revised proposal which utilizes a different type of remediation system.

Please call me at the above number and let me know what you plan to do to resolve this matter.

Sincerely,

N Acampora

Nick Acampora
Environmental Engineering
Technician III

Enc.
NA:ic
cc: W. Parish, NYSDEC
J. Jovino, Gus' Marina

DRAFT

RECEIVED
SEP 10 1992
REG. 1
OIL SPILLS

IN-SITU BIOREMEDIATION BID SPECIFICATION

Christopher O'Neill
NYSDEC
Bureau of Spill Prevention and Response
May 31, 1991

NOTE:

- Information contained in brackets, [ ], shall be determined by the NYSDEC representative preparing the bid package, considering the specific characteristics of the spill site and recommended values which may be identified in the brackets.
- Appendix V is not available at this time.

BID REQUEST FOR IN-SITU BIOLOGICAL TREATMENT TO REMEDIATE A SPILL SITE FOR THE NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION

Site Location:

DRAFT

Site History: [Appendix I]

I. SCOPE OF WORK

A bid is requested to conduct an in-situ bioremediation project at the above spill site for the NYSDEC, in accordance with the specifications described below. The project shall consist of separately identified Feasibility, Design, Implementation, and Closure Phases.

1. Feasibility Phase

The purpose of the Feasibility Phase is to determine whether the site is suited for bioremediation treatment, in terms of the biological, chemical, and hydrogeological parameters of the site. This phase shall consist of soil and groundwater sample collection, analyses and bench-scale studies to determine the appropriate parameters, and to verify the information provided by the NYSDEC in the Site History.

The Feasibility Phase shall specifically include, but is not limited to, the following items:

a. soil sampling and laboratory analyses in accordance with Appendix II;
b. groundwater sampling and laboratory analyses in accordance with Appendix III;
c. sampling of separate phase contaminant (if present), to determine its identity and degradability;
d. geologic and hydrogeologic data, including grain size distributions, soil permeability, and drilling logs;
e. groundwater velocity and direction;
f. chemical composition of soils and groundwater, such as adsorbed contaminant concentrations, dissolved contaminant concentrations, and inorganic nutrient and ion concentrations, with particular attention to nitrogen, phosphorous, potassium, iron, manganese and magnesium, using standard methods identified in 40 CFR Part 136;
g. total quantity of indigenous micro-organisms in each soil and groundwater sample using standard plate counting methods;
h. quantity of indigenous contaminant-utilizing bacteria in each soil and groundwater sample using standard in-house methods;
i. response of the soil and groundwater bacteria populations to oxygen and nutrient addition by plate growth methods over 3-7 day period;
j. optimization of the nutrient mixture concentrations;
k. rate of degradation of the contaminant under laboratory conditions;
l. identification of any rate limiting factors which may hinder the bioremediation, including toxicity or rate-limiting interactions;
m. treatment needed to address rate limiting factors identified above;

n. aquifer pump test for determining site-specific hydraulic parameters; and
o. any other work the bidder believes to be necessary for proper evaluation of the site parameters (work must be identified and described in bid).

The Feasibility Phase shall conclude with a full report submitted to the NYSDEC Regional Spill Project Manager and to the NYSDEC Central Office Oil Spill Technology Section. The report shall include all of the data collected, and the results of all of the tests performed. It shall contain an evaluation of the data and results, and recommendations as to whether the planned bioremediation can be successfully applied at the site. The recommendations are expected to fit one of the following general conditions: (1) biological treatment is not feasible now and not expected to be feasible in the future; (2) biological treatment is feasible now; or (3) biological treatment is not feasible now, but is expected to be feasible in the future. If the opinion of the contractor is that the site is not suitable for bioremediation, (condition 1 or 3) then such a recommendation shall be stated in the report along with the supporting documentation. If the opinion of the contractor is to proceed with the planned bioremediation (condition 2), then the report shall state the estimated time period needed to achieve the project cleanup criteria defined in the Overall Performance Criteria Section.

> The NYSDEC will review the Feasibility Report and decide on how to proceed with the project, within [30] days of receiving the completed report. If the NYSDEC decides not to proceed beyond the Feasibility Phase, then the bioremediation project will end, with no further work or expenses incurred under this bid project. If the NYSDEC decides to proceed with the bioremediation project, then the contractor will be instructed to immediately begin the Design Phase.

2. Design Phase

The Design Phase shall consist of the submittal of a complete description of the design and function of the bioremediation system components including, but not limited to, the following:

a. identification of equipment associated with other remediation technologies already on-site, which may be used in the bioremediation project;
b. locations and specifications for any and all groundwater wells and infiltration galleries to be installed;
c. locations and specifications for all equipment to be installed;
d. identification of all materials to be used;
e. quantity of materials to be used for both initial construction and operation of the system;
f. operational parameters of the bioremediation system, such as flow rates, nutrient loadings, discharge rates, and anticipated maintenance shut-downs;
g. identification of locations for soil sampling in accordance with Appendix II;
h. identification of locations for groundwater sampling in accordance with Appendix III;

i. identification of laboratories to be utilized to satisfy all of the reporting requirements of the project;
j. identification of chemicals to be used as tracer compounds, or other methods to be used to evaluate the effectiveness of the nutrient addition and distribution;
k. site-specific health and safety plan;
l. demonstration of hydraulic control of the site by groundwater modeling or pump test data interpretation;
m. identification of right-of-way and adjacent properties to be utilized in the bioremediation system operation;
n. identification of state and local permits needed to construct and operate the bioremediation system;
o. identification of the monitoring frequency and parameters for determining system performance during the start-up period; and
p. any other information the contractor believes to be appropriate.

The Design Phase Report shall be submitted to the NYSDEC Regional Spill Project Manager and the NYSDEC Central Office Oil Spill Technology Section. NYSDEC will respond within [45] days of receiving the report with comments and/or approval. Once the system design is approved by the NYSDEC, the contractor will be expected to begin the Implementation Phase within [10] days. If prevailing circumstances such as weather conditions prohibit the commencement of the Implementation Phase, then an extension may be requested. The NYSDEC shall be the sole judge on whether an extension is warranted.

3. Implementation Phase

The Implementation Phase shall consist of all activities, equipment, and materials required for installation, start-up, and operation of the bioremediation system. Individuals responsible for each activity shall be identified, along with a description of their qualifications. The work for this phase shall include, but is not limited to, the following items:

a. obtaining any and all permits needed to construct and operate the bioremediation system (DEC permits shall be obtained through the Regional Spill Project Manager, non-DEC permits shall be obtained from the appropriate agencies directly);
b. procurement, delivery, installation and maintenance of all equipment, materials and supplies needed;
c. identification of start-up period whereby the groundwater pumping and recharging rates, nutrient addition concentrations and rates, and oxygen addition concentration and rate will be adjusted to their optimal values;
d. system performance monitoring during the start-up period;
e. training for operating personnel including relevant standard company procedures and OSHA training;
f. system performance monitoring after start-up period, including site visits to (1) check depth to water and depth to product on all groundwater wells at

least [twice] per week; (2) check overall operation of system equipment at least [twice] per week; and (3) any monitoring or sampling requirements which may be needed with regard to groundwater discharges, air stripper discharges, or soil venting system discharges;

g. sampling and laboratory analyses of soil in accordance with Appendix II;
h. sampling and laboratory analyses of groundwater in accordance with Appendix III;
i. monthly submittal to the NYSDEC Regional Spill Project Manager of all data collected during the previous month;
j. quarterly reports to the NYSDEC Regional Spill Project Manager and the NYSDEC Central Office Oil Spill Technology Unit, evaluating the progress of the bioremediation project; and
k. provide and pay for all utilities needed to operate the bioremediation system.

The Implementation Phase shall conclude when the monitoring and sampling data indicate, to the satisfaction of NYSDEC, that the groundwater and soil cleanup criteria, defined in the Overall Performance Criteria Section, have been achieved.

4. Closure Phase

The Closure Phase shall consist of all activities and procedures involved in monitoring and sampling the soil and groundwater to verify that the site continues to meet the groundwater standards, and that the site can be closed. This phase of the project shall include, but is not limited to, the following items:

a. removal of the biological treatment equipment and materials from the site, once this phase is initiated (equipment on-site prior to the initiation of biological treatment will be handled separately in accordance with previous contract conditions with previous contractor);
b. [monthly] monitoring of all of the groundwater wells for depth to water, and for the presence of floating product;
c. sampling and laboratory analyses of soil in accordance with Appendix II;
d. sampling and laboratory analyses of groundwater in accordance with Appendix III;
e. verification of the decline in bacterial populations approaching levels which were present prior to the bioremediation project;
f. [monthly] submittal of monitoring and analytical data to the NYSDEC Regional Spill Project Manager;
g. [quarterly] reports to the NYSDEC Regional Spill Project Manager and the Central Office Spill Technology Unit, evaluating the status of the closure phase; and
h. restoration of the site surface to its condition prior to the commencement of the biological treatment activities.

Once the Closure Phase reports indicate, to the satisfaction of the NYSDEC, that the groundwater and soil criteria are sustained, the contractor will be instructed to discontinue monitoring and sampling the site, and to proceed with the site restoration activities. Further site restoration will be arranged by NYSDEC with the previous contractor under the previous contract provisions. The contractor shall submit a final report to the Regional Spill Project Manager and the Central Office Spill Technology Unit, within [30] days of completing the Closure Phase. The final report shall include a synopsis of the bioremediation project, appropriate data and graphs illustrating the project progress, and general site history summary.

II. OVERALL PERFORMANCE CRITERIA

Throughout the life of the bioremediation project, the selected bidder shall be required to conduct all activities in accordance with industry-wide and company-established standard practices, procedures and codes, including, but not limited to, the following items:

1. The contractor shall coordinate the work on the project with the Regional Spill Project Manager.
2. The contractor shall be the permittee on all non-DEC permits needed for the project.
3. The contractor shall perform all water and soil sampling in accordance with NYSDEC and USEPA protocols and procedures.
4. The contractor shall install and operate all equipment in accordance with standard practices, procedures and codes.
5. The contractor shall be responsible for the proper handling, storage and disposal of any and all materials, equipment and supplies employed or generated by the project.
6. The contractor shall be responsible for the procurement, delivery, installation and maintenance of all materials and equipment needed for the project.
7. The contractor shall be responsible for meeting any and all security needs of the site.
8. The contractor shall be responsible for controlling the project activities so that activities, such as nutrient addition, do not contribute to the contravention of groundwater quality standards as measured in the perimeter monitoring wells at the site, (for example: Nitrate, as N, less than 10 ppm).
9. The contractor shall be responsible for the continual, efficient operation of the ground equipment associated with the bioremediation system components. If the system operation is interrupted for more than two consecutive days, then written

Appendix M

Anchor Chemical Industrial Chemical Survey

DIVISION OF PURE WATERS
BUREAU OF [illegible]

INDUSTRIAL CHEMICAL SURVEY

PART I

(A)

LEASE COMPLETE AND RETURN TO THE ABOVE ADDRESS, ATTENTION: INDUSTRIAL CHEMICAL SURVEY.

COMPANY NAME AI[illegible]OR CHEMICAL CO., INC.		SIC CODE (If known) 2842	OFFICE USE 28 773
NY MAILING ADDRESS 500 West John St.	CITY Hicksville	STATE New York	ZIP CODE 11801
LANT NAME (If different)	CONTACT NAME Alfred A. Jasser		TELEPHONE Area 516-433-080
PLANT ADDRESS (If different) Street	CITY	STATE	ZIP CODE

RINCIPAL BUSINESS OF PLANT
Blend and pack chemical specialties for Graphic Arts Industries

OTE: (If parent company, give name and addresses of all divisions, subsidiaries, etc. located in New York State. A separate questionnaire is to be completed and submitted for each.)

PART II
Discharge Information

ATER

1. Does your plant discharge liquid wastes to a municipally owned sanitary sewer system? ☐ Yes ☒
   Name of System
2. Is your facility permitted to discharge liquid wastes under a State (SPDES) or Federal (NPDES) permit? Permit Number ☐ Yes ☒
3. Do you discharge liquid wastes in any other manner? ☐ Yes ☒
   Explain

any of the above are "Yes":

a. Do you discharge process or chemical wastes — (i.e. water used in manufacturing including direct contact cooling water and scrubber water)? ☐ Yes ☑
b. Do you discharge non-contact cooling water? ☐ Yes ☑
c. Do you discharge collected storm drainage only? ☐ Yes ☒
d. Do you discharge sanitary wastes only? ☑ Yes ☐

AIR

1. Does your facility have sources of possible emissions to the atmosphere? ☒ Yes ☐
2. Enter Location and Facility Code as shown on your Air Pollution Control Application for Permits and Certification (If applicable) 2 8 2 4 0 0 0 1 7 2

SOLID & CONCENTRATED LIQUID WASTES

1. List Name and Address of Firm (Including yourself) removing wastes other than office and cafeteria refuse.

Name Master Sanitation Co.			
Address P.O. Box 625	Huntington	State NY	Zip Code 11743
Name			
Address	City	State	Zip Code

2. List Location(s) of Landfill(s) owned and used by your facility.

		Active	
1	NONE	☐	☐
2	NONE	☐	☐

PES

1. Does this facility:
   Manufacture Pesticides or Pesticide Product Ingredients? ☐ Yes ☑
   Produce Pesticides or Pesticide Product Ingredients? ☐ Yes ☑
   Formulate Pesticides? ☐ Yes ☑
   Repackage Pesticides? ☐ Yes ☑
2. EPA Establishment Number ☐☐☐☐☐☐ – ☐☐ – ☐☐☐

302280

PART III

SUBSTANCES OF CONCERN
(Refer to attached TABLE I)

Complete all information for those substances your facility has used, produced, stored, distributed or otherwise disposed of since January 1, 1971. Do include chemicals used only in analytical laboratory work. Enter the name and code from Table I. If facility uses a substance in any of the Classes A which is not specified in the list, enter it as code class plus 99, e.g. B99 with name, usage, etc.

NAME OF SUBSTANCE	CODE	AVERAGE ANNUAL USAGE	AMOUNT NOW ON HAND	(✓) GAL.	LB.	PURPOSE OF USE (State whether produced, reacted, ble packaged, distributed, no longer used,
Methylene chloride	A02	50,000	1,500	x		blended with other mat
						and packaged
1,1,1 trichlorethane	A07	13,000	180	x		blended and packaged u
						other materials
ethylbenzene	D06	13,000	760	x		blended and packaged u
						other materials
petroleum tars (Napthalene	E02	270	50		x	blended and packaged u
Anthracene)						other materials
dyes and organic pigments	F24	40	20		x	blended with other mat

6/79- Ron Jasser relocated in Conn.
(203) 644-2577 - He is the on
one who can give information
on Chemicals

e use chemicals of unknown composition, list trade name or other identification, name of supplier and complete information.

NAME OF SUBSTANCE	AVERAGE ANNUAL USAGE	AMOUNT NOW ON HAND	(✓) GAL.	LB.	SUPPLIER	PURPOSE OF USE (State whether produced, rea blended, packaged, distribu no longer used, etc.)

I hereby affirm under penalty of perjury that information provided on this form is true to the best of my knowledge and belief. False statements made her are punishable as a Class A misdemeanor pursuant to Section 210.45 of the Penal Law.

NATURE (Owner, Partner, or Officer) Alfred Jasser

DATE February 10,1977

E (Printed or Typed) ALFRED A. JASSER

TITLE

all information for those substances your facili... s used, produced, stored, distributed or otherwis... sed of since January 1, 1971. On no... chemicals used only in analytical laboratory work.

Of Substance/Trade Name lier and Address	CODE	AVERAGE ANNUAL USAGE	AMOUNT NOW ON HAND	(✓) GAL.	LB.	PURPOSE OF USE (State whether produced, marketed, blended, packaged, distributed, no longer used, etc.)
HYLENE CHLORIDE	402	36,000	1500	✓		BLENDED WITH OTHER MATERI
1-TRICHLORETHANE	407	19,000	160	✓		PACKAGED BLENDED PACKAGED WITH OTHER MATERIALS
HYBENZENE	806	13,000	760	✓		BLENDED & PACKAGED WITH OTHER MATERIALS
ULEUM TAR	602	240	50	✓		HEATED & PACKAGED WITH OTHER MATERIALS
ORGANIC PIGMENTS	724	40	20		✓	BLENDED WITH OTHER MAT
ADDITION TO THE	ABOVE	SUBSTANCES		NOT		INCLUDED ABOVE
TROLEUM NAPHTHA DISTILLATE		50,000 gal		✓		
YLENE GLYCOL MONOETHYLETHER		20,000 gal		✓		
YLENE, GLYCOLS		2,000 gal		✓		
IONIC ~~ETHOXYLATED~~ ALCOHOL ETHOXYLATED		10,000 gal		✓		

Final Discharge Point

☐ Sewers
☒ Cesspools
☐ Sumps or basins
☐ Drums
☐ Landfills
☐ Other

Recommended Action

☐ Immediate abatement
☑ Sample
☒ SPDES Appication
☐ Reinspection
☐ Referred to ________
☐ No Action

STATIONARY COMBUSTION AND INCINERATION

A. Heating System	☐ None	☒ Boiler	☐ Space Heaters
B. Fuel	☐ Electric	☒ Gas	☐ Oil
C. Incinerator	☐ Yes	☐ No	

hereby affirm under penalty of perjury that information provided on this form is true to the best of my knowledge and belief. False statements ma... re punishable as a Class A misdemeanor pursuant to Section 210.45 of the Penal Law.

e (Owner, Partner, or Officer) Alfred A Jasser

DATE 2/9/77

nted or Typed). Alfred A. Jesser

TITLE Pres.

ctors Name

Received 4/26/95 [illegible]

ANSON
ENVIRONMENTAL LTD.

33 Gerard Street • Suite 100
Huntington, New York 11743
516•351•3555
Fax 516•351•3615

REMEDIAL INVESTIGATION REPORT

SUPPLEMENT

Anchor Chemical Site
Hicksville, New York

April 1995

Prepared for:
K.B. Co.
375 North Broadway
Jericho, New York 11753

Prepared by:
Anson Environmental Ltd.
33 Gerard Street
Suite 100
Huntington, New York 11743

ANS N
ENVIRONMENTAL LTD.

33 Gerard Street • Suite 100
Huntington, New York 11743
516•351•3555
Fax 516•351•3615

Supplementary Soil and Groundwater Sampling
Remedial Investigation Report
Anchor Chemical Site
500 West John Street
Hicksville, New York

April 10, 1995

Due to its concern over the presence of 2-butoxyethanol and 1,4-dioxane in earlier sampling events, EPA required additional soil and groundwater analysis. This report discusses the results of that additional sampling.

Soil Sampling

On February 7, 1995, two vertical soil borings were installed in the vicinity of tank 14 outside the building using hollow-stem augers. Soil samples were collected using a two-inch diameter split spoon samplers and were collected at five foot intervals from the ground surface to thirty five feet below grade.

The soil samples were collected at the following distances below grade in both vertical borings.

5 to 7 feet
10 to 12 feet
15 to 17 feet
20 to 22 feet
25 to 27 feet
30 to 32 feet
35 to 37 feet

As agreed upon with EPA, soil samples were selected for laboratory analysis based on head space readings, soil color and appearance. Head space readings did not exceed background levels and none of the soil samples appeared to be discolored. Therefore, the two deepest soil samples, 30 to 32 feet and 35 to 37 feet, were selected from both borings for laboratory analysis.

Boring #1 was installed 5.8 feet to the east of the outside wall and 52.65 feet south from the northeastern corner of the building. Boring #2 was installed 5.8 feet from the eastern wall and 39.05 feet south of the northeastern corner of the building. The boring locations are marked on the site diagram.

On February 8th, an angle boring was installed such that a soil sample was collected from under tank 14 at a depth of approximately 17 feet, as prescribed by the USEPA. This boring was installed 24.4 feet east of the building, beginning on Nassau County Park property-the grass easement along the entrance road to Cantiague Park.

The boring logs for the two vertical borings are attached in the appendix.

Data Analysis and Results

The soil and groundwater samples were analyzed for Target Compound List volatile and semi-volatile organic compounds in accordance with CLP protocol. The analysis for 2-butoxyethanol was done as part of the analysis for semi-volatile compounds. The protocol for 1,4-dioxane was implemented as a supplement to the basic CLP analysis.

The data were submitted to Environmental Standards Inc. of Valley Forge, PA for validation. The validated data results are discussed in the following sections.

The validated data sheets and validator's commentary are attached in the appendix.

Soil Sample Analyses

There were no volatile or semi-volatile organic compounds detected in the soil samples. The phthlates were considered "non-detect" by the validator as the compounds were detected in the blanks.

The 1,4-dioxane was not detected by the laboratory utilizing the agreed upon protocol. The 2-butoxyethanol was not detected in any soil sample.

The former contents of Tank 14, butylcellusolve, was also not detected.

Groundwater Sample Analyses

Monitoring wells, MW 2 and MW 3, could not be sampled as planned as the water table had dropped at the time of sampling due to the lack of precipitation. Therefore, there was insufficient volume to conduct the sampling. With the concurrence of the USEPA, MW 4, a downgradient well on-site, was sampled as was MW 5S, as was prescribed in the original scope of work.

There were no volatile or semi-volatile organic compounds detected in the sample collected from MW 4. The compounds 1,4-dioxane and 2-butoxyethanol were not detected.

In MW 5S, 29 µg/L of 1,1,1-trichloroethane was detected and an estimated 3 µg/L of 1,1-dichloroethane. The ARAR for the each of the two compounds is 5 µg/L.

The compounds, 1,4-dioxane and 2-butoxyethanol, were not detected.

Conclusions

The soil samples did not reveal the presence of contamination near Tank 14. The downgradient well, MW 4, did not indicate the presence of contamination. Although 29 µg/L of 1,1,1-trichloroethane was detected in MW 5S, no additional sampling is recommended since there is no current source of 1,1,1-trichloroethane on site. The conditions of the Upper Glacial Aquifer in this region have been documented to include generalized contamination by chlorinated solvents, such as 1,1,1-trichloroethane and 1,1-dichloroethane.

In addition, no further action is warranted with respect to the 1,1-dichloroethane since the level present was below the ARAR of 5 µg/L.

APPENDIX

BORING LOGS

SUBSURFACE LOG

PR CT: Anchor Chemical

DRILL TYPE: Hollow Stem Auger

DATE: 2 / 7 / 95

BOTTOM OF BORING (BOB): 37'

BORING NO.: B #1

SAMPLER TYPE: Split Spoon

RECORDED BY: F. Tooher

WEATHER: sunny, cool

DRILLER: S. Taylor

SAMPLE NO.	DEPTH FROM-TO FEET	RECOV. (ft)	OVM READING (s/s)	OVM READING (headspace)	SAMPLE DESCRIPTION
1	5 - 7		0 ppm	.8 ppm	tan sand, gravel, pebbles
2	10 - 12		0 ppm	.8 ppm	tan sand, gravel, some pebbles
3	15 - 17		0 ppm	.2 ppm	tan-orange sand, gravel and some pebbles
4	20 - 22		0 ppm	.5 ppm	tan - orange coarse sand, gravel, some pebbles and cobbles
5	25 -27		0 ppm	.8 ppm	tan- brown fine sand, some gravel, pebbles
6	30 - 32		0 ppm	.8 ppm	tan- brown fine sand some gravel, pebbles
7	35 -37		0 ppm	.8 ppm	tan- brown fine sand some gravel, pebbles

Complete boring at 35 - 37

SUBSURFACE LOG

OJECT: Anchor Chemical

DRILL TYPE: Hollow Stem Auger

TE: 2 / 7 / 95

BOTTOM OF BORING (BOB): 37'

RING NO.: B #2

SAMPLER TYPE: Split Spoon

CORDED BY: F. Tooher

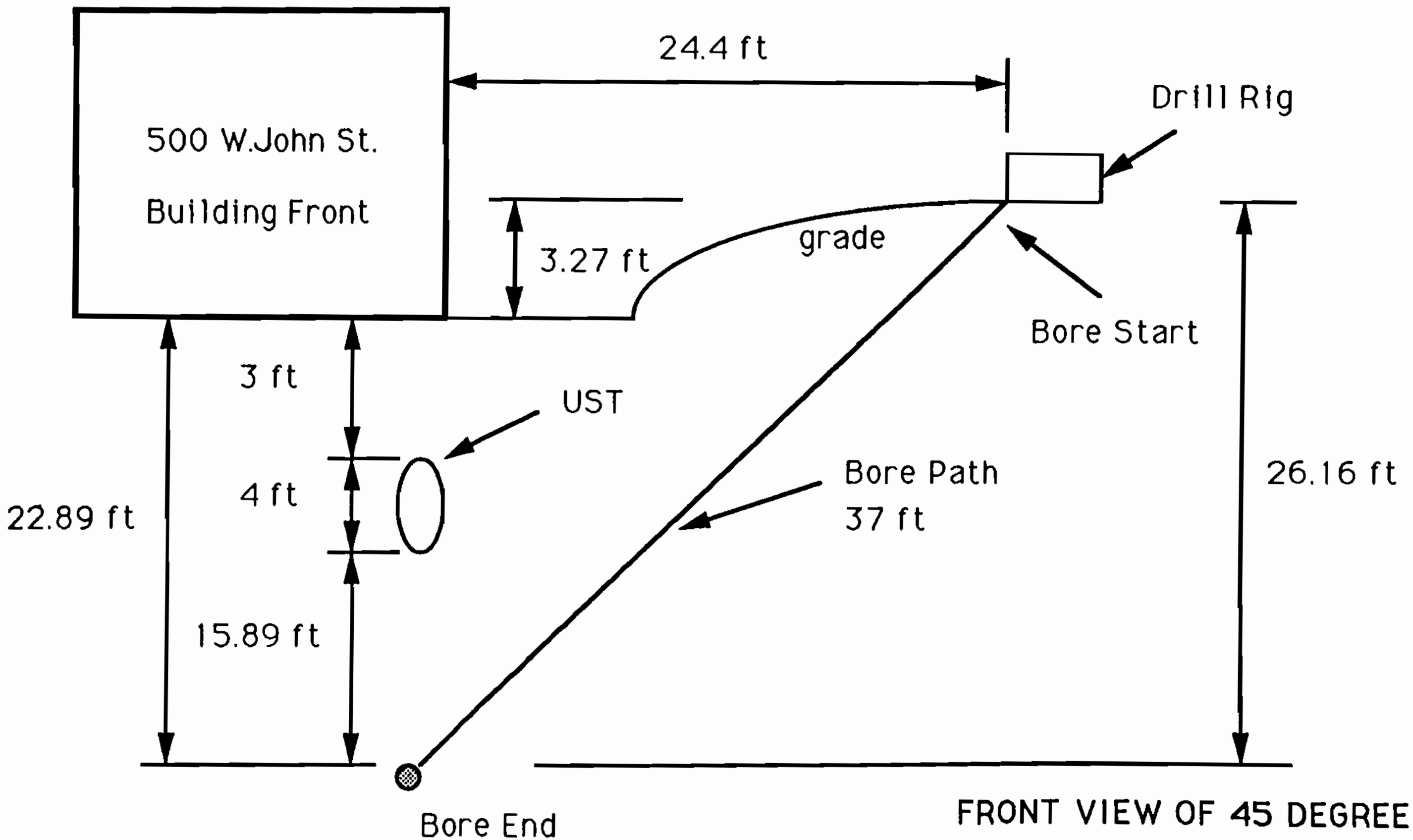
WEATHER: sunny, cool

ILLER: S. Taylor

MPLE).	DEPTH FROM-TO FEET	RECOV. (ft)	OVM READING (s/s)	OVM READING (headspace)	SAMPLE DESCRIPTION
	5 - 7		0 ppm	1-3 ppm	tan sand, gravel, pebbles
	10 - 12		0 ppm	.8 ppm	tan sand, gravel, some pebbles
	15 - 17		0 ppm	.5 ppm	tan-orange sand, gravel,some pebbles some cobbles
	20 - 22		0 ppm	.5 ppm	tan - orange coarse sand, gravel, some pebbles and cobbles
	25 -27		0 ppm	.8 ppm	tan- brown fine sand, some gravel, pebbles
	30 - 32		0 ppm	.5 ppm	tan- brown fine sand, some gravel, pebbles
	35 -37		0 ppm	1.0 ppm	tan- brown fine sand some gravel, pebbles

lete boring at 35 - 37

SITE DIAGRAMS



FRONT VIEW OF 45 DEGREE
SLANT BORING

500 WEST JOHN STREET

ANSON ENVIRONMENTAL LTD.
ENGINEERING SKETCH

BORING DATE: 2/8/95 Scale: none

VALIDATED LABORATORY RESULTS

VOLATILE ORGANIC ANALYSIS						
Sample Number Laboratory Sample Number	T502120-	Boring 1 (30-32) 01	Boring 1 (35-37) 02	Boring 2 (30-32) 03	Boring 2 (35-37) 04	Boring 3 05
Remarks						
Units		µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/Kg
VOLATILE COMPOUNDS	Quantitation Limit					
Chloromethane	10	UJ	UJ	UJ	UJ	UJ
Bromomethane	10	UJ	UJ	UJ	UJ	UJ
Vinyl Chloride	10	UJ	UJ	UJ	UJ	UJ
Chloroethane	10					
Methylene Chloride	10				10 U	
Acetone	10					
Carbon Disulfide	10					
1,1-Dichloroethene	10					
1,1-Dichloroethane	10					
Total 1,2-Dichloroethene	10					
Chloroform	10					
1,2-Dichloroethane	10					
2-Butanone	10					
1,1,1-Trichloroethane	10					
Carbon Tetrachloride	10					
Bromodichloromethane	10					
1,1,2,2-Tetrachloroethane	10					
1,2-Dichloropropane	10					
trans-1,3-Dichloropropene	10					
Trichloroethene	10					
Dibromochloromethane	10					
1,1,2-Trichloroethane	10					
Benzene	10					
cis-1,3-Dichloropropene	10					
Bromoform	10					
2-Hexanone	10	UJ	UJ	UJ	UJ	UJ
4-Methyl-2-Pentanone	10	UJ	UJ	UJ	UJ	UJ

VOLATILE ORGANIC ANALYSIS						
Sample Number		Boring 1 (30-32)	Boring 1 (35-37)	Boring 2 (30-32)	Boring 2 (35-37)	Boring 3
Laboratory Sample Number	T502120-	01	02	03	04	05
Remarks						
Units		µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/Kg
VOLATILE COMPOUNDS	Quantitation Limit					
Tetrachloroethene	10					
Toluene	10					
Chlorobenzene	10					
Ethylbenzene	10					
Styrene	10					
Total Xylenes	10					
1,4-Dioxane	1000	R	R	R	R	R
Quantitation Limit Multiplier		1.00	1.00	1.00	1.00	1.00
Date of Sample Collection		2/7/95	2/7/95	2/7/95	2/7/95	2/7/95
Date Sample Received by Laboratory		2/8/95	2/8/95	2/8/95	2/8/95	2/8/95
Date of Sample Analysis		2/16/95	2/16/95	2/16/95	2/16/95	2/16/95
Instrument Used for Analysis		MSD/B	MSD/B	MSD/B	MSD/B	MSD/B

VOLATILE ORGANIC ANALYSIS					
Sample Number		MW-5S	Trip Blank	Field Blank	MW-4
Laboratory Sample Number	T502120-	06	09	10	11
Remarks					
Units		µg/L	µg/L	µg/L	µg/L
VOLATILE COMPOUNDS	Quantitation Limit				
Chloromethane	10				
Bromomethane	10				
Vinyl Chloride	10				
Chloroethane	10				
Methylene Chloride	10				
Acetone	10				
Carbon Disulfide	10				
1,1-Dichloroethene	10				
1,1-Dichloroethane	10	3 J			
Total 1,2-Dichloroethene	10				
Chloroform	10				
1,2-Dichloroethane	10				
2-Butanone	10				
1,1,1-Trichloroethane	10	29			
Carbon Tetrachloride	10				
Bromodichloromethane	10				
1,1,2,2-Tetrachloroethane	10				
1,2-Dichloropropane	10				
trans-1,3-Dichloropropene	10				
Trichloroethene	10				
Dibromochloromethane	10				
1,1,2-Trichloroethane	10				
Benzene	10				
cis-1,3-Dichloropropene	10				
Bromoform	10				
2-Hexanone	10	UJ	UJ	UJ	UJ
4-Methyl-2-Pentanone	10				

VOLATILE ORGANIC ANALYSIS					
Sample Number		MW-5S	Trip Blank	Field Blank	MW-4
Laboratory Sample Number	T502120-	06	09	10	11
Remarks					
Units		μg/L	μg/L	μg/L	μg/L
VOLATILE COMPOUNDS	Quantitation Limit				
Tetrachloroethene	10				
Toluene	10				
Chlorobenzene	10				
Ethylbenzene	10				
Styrene	10				
Total Xylenes	10				
1,4-Dioxane	1000	R	R	R	R
Quantitation Limit Multiplier		1.00	1.00	1.00	1.00
Date of Sample Collection		2/8/95	2/8/95	2/8/95	2/8/95
Date Sample Received by Laboratory		2/8/95	2/8/95	2/8/95	2/8/95
Date of Sample Analysis		2/15/95	2/15/95	2/15/95	2/15/95
Instrument Used for Analysis		MSD/C	MSD/C	MSD/C	MSD/C

TBLES01.XLS

EXTRACTABLE ORGANIC ANALYSIS						
Sample Number Laboratory Sample Number		T502120-	Boring 1 (30-32) 01	Boring 1 (35-37) 02	Boring 2 (30-32) 03	Boring 2 (35-37) 04
Remarks						
Units			µg/Kg	µg/Kg	µg/Kg	µg/Kg
SEMIVOLATILE COMPOUNDS	Quantitation Limit (Aq)	Quantitation Limit (Sol)				
Phenol	10	330				R
bis(2-Chloroethyl)ether	10	330				R
2-Chlorophenol	10	330				R
1,3-Dichlorobenzene	10	330				R
1,4-Dichlorobenzene	10	330				R
1,2-Dichlorobenzene	10	330				R
2-Methylphenol	10	330				R
2,2'-oxybis(1-Chloropropane)	10	330				R
4-Methylphenol	10	330				R
N-Nitroso-di-n-Propylamine	10	330				R
Hexachloroethane	10	330				R
Nitrobenzene	10	330				R
Isophorone	10	330				R
2-Nitrophenol	10	330				R
2,4-Dimethylphenol	10	330				R
bis(2-Chloroethoxy)methane	10	330				R
2,4-Dichlorophenol	10	330				R
1,2,4-Trichlorobenzene	10	330				R
Naphthalene	10	330				R
4-Chloroaniline	10	330				R
Hexachlorobutadiene	10	330				R
4-Chloro-3-Methylphenol	10	330				R
2-Methylnaphthalene	10	330				R
Hexachlorocyclopentadiene	10	330				R
2,4,6-Trichlorophenol	10	330				R
2,4,5-Trichlorophenol	25	800				R
2-Chloronaphthalene	10	330				R

EXTRACTABLE ORGANIC ANALYSIS						
Sample Number Laboratory Sample Number		T502120-	Boring 1 (30-32) 01	Boring 1 (35-37) 02	Boring 2 (30-32) 03	Boring 2 (35-37) 04
Remarks						
Units			µg/Kg	µg/Kg	µg/Kg	µg/Kg
SEMIVOLATILE COMPOUNDS	Quantitation Limit (Aq)	Quantitation Limit (Sol)				
2-Nitroaniline	25	800				R
Dimethylphthalate	10	330				R
Acenaphthylene	10	330				R
2,6-Dinitrotoluene	10	330				R
3-Nitroaniline	25	800				R
Acenaphthene	10	330				R
2,4-Dinitrophenol	25	800				R
4-Nitrophenol	25	800				R
Dibenzofuran	10	330				R
2,4-Dinitrotoluene	10	330				R
Diethylphthalate	10	330				R
4-Chlorophenyl-phenylether	10	330				R
Fluorene	10	330				R
4-Nitroanaline	25	800				R
4,6-Dinitro-2-Methylphenol	25	800				R
N-Nitrosodiphenylamine	10	330				R
4-Bromophenyl-phenylether	10	330				R
Hexachlorobenzene	10	330				R
Pentachlorophenol	25	800				R
Phenanthrene	10	330				R
Anthracene	10	330				R
Carbazole	10	330				R
Di-n-Butylphthalate	10	330	340 U	340 U	340 U	R
Fluoranthene	10	330				R
Pyrene	10	330				R
Butylbenzylphthalate	10	330				R
3,3'-Dichlorobenzidine	10	330				R

EXTRACTABLE ORGANIC ANALYSIS						
Sample Number Laboratory Sample Number		T502120-	Boring 1 (30-32) 01	Boring 1 (35-37) 02	Boring 2 (30-32) 03	Boring 2 (35-37) 04
Remarks						
Units			µg/Kg	µg/Kg	µg/Kg	µg/Kg
SEMIVOLATILE COMPOUNDS	Quantitation Limit (Aq)	Quantitation Limit (Sol)				
Benzo(a)anthracene	10	330				R
bis(2-Ethylhexyl)phthalate	10	330	340 U	340 U	340 U	340 U
Chrysene	10	330				R
Di-n-Octylphthalate	10	330				R
Benzo(b)fluoranthene	10	330				R
Benzo(k)fluoranthene	10	330				R
Benzo(a)pyrene	10	330				R
Indeno(1,2,3-cd)pyrene	10	330				R
Dibenz(a,h)anthracene	10	330				R
Benzo(g,h,i)perylene	10	330				R
Quantitation Limit Multiplier			1.02	1.03	1.03	1.03
Date of Sample Collection			2/7/95	2/7/95	2/7/95	2/7/95
Date Sample Received by Laboratory			2/8/95	2/8/95	2/8/95	2/8/95
Date of Sample Extraction			2/9/95	2/9/95	2/9/95	2/9/95
Date of Sample Analysis			3/6/95	3/6/95	3/6/95	3/6/95
Instrument Used for Analysis			MSD/J	MSD/J	MSD/J	MSD/J

EXTRACTABLE ORGANIC ANALYSIS							
Sample Number			Boring 2 (35-37)	Boring 3	MW-5S	Field Blank	MW-4
Laboratory Sample Number		T502120-	04	05	06	10	11
Remarks			Rextraction/Reanalysis				
Units			µg/Kg	µg/Kg	µg/L	µg/L	µg/L
SEMIVOLATILE COMPOUNDS	Quantitation Limit (Aq)	Quantitation Limit (Sol)					
Phenol	10	330	R				
bis(2-Chloroethyl)ether	10	330	R				
2-Chlorophenol	10	330	R				
1,3-Dichlorobenzene	10	330	R				
1,4-Dichlorobenzene	10	330	R				
1,2-Dichlorobenzene	10	330	R				
2-Methylphenol	10	330	R				
2,2'-oxybis(1-Chloropropane)	10	330	R				
4-Methylphenol	10	330	R				
N-Nitroso-di-n-Propylamine	10	330	R				
Hexachloroethane	10	330	R				
Nitrobenzene	10	330	R		UJ	UJ	UJ
Isophorone	10	330	R				
2-Nitrophenol	10	330	R				
2,4-Dimethylphenol	10	330	R				
bis(2-Chloroethoxy)methane	10	330	R				
2,4-Dichlorophenol	10	330	R		UJ	UJ	UJ
1,2,4-Trichlorobenzene	10	330	R				
Naphthalene	10	330	R				
4-Chloroaniline	10	330	R				
Hexachlorobutadiene	10	330	R		UJ	UJ	UJ
4-Chloro-3-Methylphenol	10	330	R				
2-Methylnaphthalene	10	330	R				
Hexachlorocyclopentadiene	10	330	R				
2,4,6-Trichlorophenol	10	330	R				
2,4,5-Trichlorophenol	25	800	R				
2-Chloronaphthalene	10	330	R				

EXTRACTABLE ORGANIC ANALYSIS							
Sample Number			Boring 2 (35-37)	Boring 3	MW-5S	Field Blank	MW-4
Laboratory Sample Number		T502120-	04	05	06	10	11
Remarks			Rextraction/Reanalysis				
Units			µg/Kg	µg/Kg	µg/L	µg/L	µg/L
SEMIVOLATILE COMPOUNDS	Quantitation Limit (Aq)	Quantitation Limit (Sol)					
2-Nitroaniline	25	800	R				
Dimethylphthalate	10	330	R				
Acenaphthylene	10	330	R				
2,6-Dinitrotoluene	10	330	R				
3-Nitroaniline	25	800	R				
Acenaphthene	10	330	R				
2,4-Dinitrophenol	25	800	R				
4-Nitrophenol	25	800	R				
Dibenzofuran	10	330	R				
2,4-Dinitrotoluene	10	330	R				
Diethylphthalate	10	330	R		10 U	1 J	
4-Chlorophenyl-phenylether	10	330	R				
Fluorene	10	330	R				
4-Nitroanaline	25	800	R				
4,6-Dinitro-2-Methylphenol	25	800	R				
N-Nitrosodiphenylamine	10	330	R				
4-Bromophenyl-phenylether	10	330	R				
Hexachlorobenzene	10	330	R				
Pentachlorophenol	25	800	R		UJ	UJ	UJ
Phenanthrene	10	330	R				
Anthracene	10	330	R				
Carbazole	10	330	R				
Di-n-Butylphthalate	10	330	340 U	350 U	10 U		
Fluoranthene	10	330	R				
Pyrene	10	330	R				
Butylbenzylphthalate	10	330	R				
3,3'-Dichlorobenzidine	10	330	R				

EXTRACTABLE ORGANIC ANALYSIS							
Sample Number			Boring 2 (35-37)	Boring 3	MW-5S	Field Blank	MW-4
Laboratory Sample Number		T502120-	04	05	06	10	11
Remarks			Rextraction/Reanalysis				
Units			µg/Kg	µg/Kg	µg/L	µg/L	µg/L
SEMIVOLATILE COMPOUNDS	Quantitation Limit (Aq)	Quantitation Limit (Sol)					
Benzo(a)anthracene	10	330	R				
bis(2-Ethylhexyl)phthalate	10	330	R	350 U	13 U	1 J	10 U
Chrysene	10	330	R				
Di-n-Octylphthalate	10	330	R				
Benzo(b)fluoranthene	10	330	R				
Benzo(k)fluoranthene	10	330	R				
Benzo(a)pyrene	10	330	R				
Indeno(1,2,3-cd)pyrene	10	330	R				
Dibenz(a,h)anthracene	10	330	R				
Benzo(g,h,i)perylene	10	330	R				
Quantitation Limit Multiplier			1.03	1.04	1.00	1.00	1.00
Date of Sample Collection			2/7/95	2/7/95	2/8/95	2/8/95	2/8/95
Date Sample Received by Laboratory			2/8/95	2/8/95	2/8/95	2/8/95	2/8/95
Date of Sample Extraction			3/7/95	2/9/95	2/10/95	2/10/95	2/10/95
Date of Sample Analysis			3/7/95	3/6/95	3/7/95	3/7/95	3/7/95
Instrument Used for Analysis			MSD/J	MSD/J	MSD/J	MSD/J	MSD/J

TENTATIVELY IDENTIFIED COMPOUNDS						
Sample Number	Boring 1 (30-32)	Boring 1 (35-37)	Boring 2 (30-32)	Boring 2 (35-37)	Boring 3	MW-5S
Laboratory Sample Number T502120-	01	02	03	04	05	06
Remarks						
Units	µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/L
COMPOUNDS						
VOLATILE COMPONENTS						
Blank Contamination (Number of Peaks)	41(2) R	35(2) R	51(2) R	78(2) R	23(2) R	14(2) R
Laboratory Artifacts			6 R	5 R	5 R	
SEMIVOLATILE COMPONENTS						
Blank Contamination	1900 R	1500 R	1700 R	290 R	1700 R	
Unknowns (Number of Peaks)						270(17) J
Saturated Hydrocarbon and Phthalate					160 J	
Saturated Hydrocarbon						5 J
Unknown Phthalate						18(3) J

TENTATIVELY IDENTIFIED COMPOUNDS				
Sample Number	Trip Blank	Field Blank	MW-4	Boring 2 (35-37)
Laboratory Sample Number T502120-	9	10	11	04
Remarks				Rextraction/Reanalysis
Units	µg/L	µg/L	µg/L	µg/Kg
COMPOUNDS				
VOLATILE COMPONENTS	-			NA
Blank Contamination (Number of Peaks)		16(2) R	10(2) R	
Laboratory Artifacts				
SEMIVOLATILE COMPONENTS				
Blank Contamination	NA	-	-	1600 R
Unknowns (Number of Peaks)				900(3) J
Saturated Hydrocarbon and Phthalate				
Saturated Hydrocarbon				
Unknown Phthalate				

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VOLATILE ORGANIC ANALYSIS						
Sample Number		Boring 1 (30-32)	Boring 1 (35-37)	Boring 2 (30-32)	Boring 2 (35-37)	Boring 3
Laboratory Sample Number	L5-02-044-	01	02	03	04	05
Remarks						
Units		µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/Kg
VOLATILE COMPOUNDS	Quantitation Limit					
2-Butoxyethanol	1000					
Quantitation Limit Multiplier		1.00	1.00	1.00	1.00	1.00
Date of Sample Collection		2/7/95	2/7/95	2/7/95	2/7/95	2/7/95
Date Sample Received by Laboratory		2/10/95	2/10/95	2/10/95	2/10/95	2/10/95
Date of Sample Analysis		2/15/95	2/15/95	2/15/95	2/15/95	2/15/95

VOLATILE ORGANIC ANALYSIS				
Sample Number		MW-5S	Field Blank	MW-4
Laboratory Sample Number	L5-02-044-	06	09	10
Remarks				
Units		µg/L	µg/L	µg/L
VOLATILE COMPOUNDS	Quantitation Limit			
2-Butoxyethanol	1000			
Quantitation Limit Multiplier		1.00	1.00	1.00
Date of Sample Collection		2/8/95	2/8/95	2/8/95
Date Sample Received by Laboratory		2/10/95	2/10/95	2/10/95
Date of Sample Analysis		2/15/95	2/15/95	2/15/95

Section 1 Quality Assurance Review

A. Organic Data

The organic analyses of seven soil samples (including the matrix spike and matrix spike duplicate samples) and six aqueous samples (including one trip blank and one field blank) were performed by Laboratory Resources, Inc., of Teterboro, New Jersey, and Bethlehem, Pennsylvania. These samples were collectively analyzed by CLP protocols for the Target Compound List (TCL) volatile organics and TCL semivolatile organics (at the Teterboro facility). In addition, library searches were performed for 30 extraneous chromatographic peaks for the volatile and semivolatile fractions combined. The samples were also analyzed for 1,4-dioxane using SW-846 Method 8240 (at the Teterboro facility) and for 2-butoxyethanol by SW-846 Method 8015 (at the Bethlehem facility). The analytical results are presented in Section 2.

The findings offered in this report are based upon a review of holding times, blank analysis results, matrix spike and surrogate recoveries, GC/MS tuning, internal standard areas, target compound matching quality, calibrations, analytical sequence, quantitation of positive results and overall system performance. Sample-specific quantitation limits can be obtained by multiplying the general quantitation limits by the quantitation limit multipliers presented on the data summary tables in Section 2. The sole purpose of the quantitation limit multipliers is to reproduce the laboratory-rounded, sample-specific quantitation limits.

The analytical data were examined to determine data usability in accordance with the U.S. EPA Region II validation checklist SOP No. HW-6 (Revision #9) "CLP Organics Data Review and Preliminary Review." The analytical requirements and required deliverables specified in the CLP Statement of Work were met for this data set with the following exceptions and comments. It should be noted that the following items are contractual in nature and do not necessarily affect data usability. Usability is addressed in a subsequent section.

Correctable Deficiencies

1. The laboratory did not label the surrogate compound recovery forms, method blank summary forms, or the internal standard summary forms with the Anson Environmental Ltd. sample numbers in the data package for the analysis of 1,4-dioxane. The data reviewer has entered these sample numbers on the quality control summary forms for the analysis.

2. The laboratory did not assign unique sample designations on all forms and raw data for two semivolatile method blanks (both SBLK01). All semivolatile method blanks should be designated using SBLK## (SOW, Document OLM01.8, B-28).

3. The laboratory did not supply any retention time information for the analysis of 2-butoxyethanol in the project samples. The data reviewer has examined the chromatograms for the initial calibration standards and matrix spike/matrix spike

duplicate analyses and has determined that the compound displayed a retention time of 17.9 minutes; this appeared to be very stable throughout the analytical sequence. The laboratory did not report any positive results for the compound in the samples, and no significant peaks were observed in the region around 17.9 minutes on the sample chromatograms.

4. The laboratory did not report what sample weights/volumes were used in the analysis of the samples for 2-butoxyethanol. The data reviewer has assumed that the laboratory consistently used a 5-gram aliquot of solid sample and a 5-mL volume for all aqueous samples and standards for the purge as suggested in the methods.

5. The GC column used for the GC/MS analysis of the project samples was able to separate the two isomers of 1,2-dichloroethene, and the laboratory used the later-eluting peak (eluting at 9.49 minutes on instrument MSD/C and at 8.07 minutes on instrument MSD/B) for the calculation of the relative response factors for total-1,2-dichloroethene in the initial and continuing calibrations performed for the volatile organics analysis. However, the laboratory should have summed the areas of the two peaks for the calculation of the relative response factors for total-1,2-dichloroethene in the initial and continuing calibrations (OLM01.8, D-28/VOA). This issue has no impact on data quality because no positive results were reported for total-1,2-dichloroethene in the samples.

6. The laboratory reported the absolute value of the percent differences on the continuing calibration summary forms for the volatile and semivolatile organics analyses. The laboratory should not be using the absolute value operator in the equation for the percent differences as the CLP protocol states that negative percent differences are possible (OLM01.8, D-30/VOA and D-48/SV).

7. The "B" qualifier flag (indicating the presence of the compound in a method blank) appears for methylene chloride on the Form I's for the matrix spike and matrix spike duplicate analyses of sample Boring 3. However, since methylene chloride was not detected in these analyses (as the quantitation limits were also flagged "U"), the "B" flag should not have been entered on the forms.

8. The laboratory often labelled the quality control summary forms and raw data for sample MW-5S in the semivolatile and volatile organics data packages with "MW-5S2." In many cases, the laboratory caught the error and corrected the mistakes. However, the correction was not made in all instances in which the error appeared.

9. Several inconsistencies were noted between the results reported on the matrix spike recovery summary forms (Form IIIs) and the associated Form I's for the matrix spike/matrix spike duplicate analyses performed in the volatile and semivolatile organics analyses. The table below summarizes these inconsistencies. It should be noted that in all cases, the Form IIIs reflect the results as calculated by the data reviewer from the raw data for the analyses. Data usability was not affected by this issue.

Sample	Fraction	Compound	Result From Form I	Result From Form III
Boring 3MS	volatile	toluene	57 μg/Kg	56 μg/Kg
MW-5SMSD	volatile	trichloroethene	43 μg/L	42 μg/L
MW-5SMS	semivolatile	pyrene	32 μg/L	31 μg/L
MW-5SMSD	semivolatile	4-nitrophenol	60 μg/L	59 μg/L

10. The laboratory reported a relative response factor of 1.5170 for total-1,2-dichloroethene on the continuing calibration check summary form (Form VII VOA) for the check performed on 2/15/95 at 11:17. The raw data indicates that the true relative response factor was 1.6238; in addition, the percent difference should have been reported as 5.02% instead of 1.88% as reported on the Form VII. (See Correctable Deficiency #5 above for the procedure used to calculate the relative response factors for the compound.)

11. The area counts reported for internal standard compound bromochloromethane on the internal standard area and retention time summary (Form VIII VOA) for the solid samples analysis do not match those observed in the raw data for the volatile organics analysis of the solid samples. The data reviewer has transcribed the area counts for the internal standard from the raw data to the summary form. This issue does not impact data usability because the correct internal standard areas are within the specified quality control limit.

12. A time of analysis of 12:02 was reported on the initial and continuing calibration summary forms for the time the first standard was analyzed for the initial calibration of instrument MSD/J for the semivolatile organics analysis. However, the raw data shows that the true time of analysis for the first standard of the initial instrument calibration was 12:17. The data reviewer has altered the associated quality control summary forms and has provided these in the Support Documentation section of this report.

13. The laboratory reported lab file IDs of >8083 for the 20 ppb standard and >8079 for the 10 ppb standard on the Form VI for the CLP volatile organics initial calibration performed on 2/13/95. The raw data shows that the laboratory should have reported the file IDs as >8083 for the 10 ppb standard and >8079 for the 20 ppb standard.

14. The laboratory reported that low recoveries were obtained for all surrogate compounds in the semivolatile organics analysis of sample Boring 2 (35-37). The laboratory reextracted and reanalyzed the sample and acceptable recoveries were obtained for the surrogate compounds. The laboratory provided raw data and results for both sets of analyses. However, per CPL protocol, the laboratory was required to submit only the raw data and summary forms for the analysis which displayed acceptable surrogate recoveries. It should be noted that the reextraction of the sample was performed outside holding time requirements. For completeness, both sets of analytical results for the sample appear on the data summary tables.

15. The laboratory did not label the data package quality control summary forms with the sample identifications as they appear on the Chain-of-Custody Records. This may be due to the fact that the sample identifications were rather long and could not fit into the fields provided for the sample identification on the summary forms (which have a capacity of about ten characters). The data reviewer has referred to all samples by the identifications as they appear on the Chain-of-Custody Records.

16. The laboratory used a quantitation limit of 830 µg/Kg as the basis for the sample-specific quantitation limits for 2,4,5-trichlorophenol, 2-nitroaniline, 3-nitroaniline, 2,4-dinitrophenol, 4-nitrophenol, 4-nitroaniline, 4,6-dinitro-2-methylphenol and pentachlorophenol in the semivolatile organics analysis of the project samples. Per CLP protocol, the laboratory should have used 800 µg/Kg as the basis for the sample-specific quantitation limits for these compounds. The correct quantitation limits appear on the data summary tables in this report.

Noncorrectable Deficiencies

1. According to the raw data and quality control summary forms, instrument MSD/C (which was used for the aqueous samples) did not detect 1,4-dioxane in the low concentration initial calibration standard (0.50 mg/L). Therefore, the laboratory did not use the required five standards for the initial calibration of instrument MSD/C for the analysis of 1,4-dioxane in the aqueous samples (SW-846, 8015A-3). It should be noted that 1,4-dioxane was not detected in any aqueous sample, and it is the opinion of the data reviewer that the analysis was capable of determining the presence of the compound down to the reporting limit of 1.5 mg/L for the aqueous samples. (See Comment #2 below.)

2. The laboratory used initial and continuing calibration standards for the semivolatile organics analysis that contained the non-target compound 3-methylphenol as well as target compound 4-methylphenol, which coeluted on the instrument used for analysis (MSD/J). Therefore, the laboratory calculated the relative response factor for 4-methylphenol based on the concentration of both compounds combined. This deviation from the protocol was noted in the case narrative submitted with the data package. Because there were no positive results for 4-methylphenol in the project samples, data usability was not affected by this issue.

Comments

1. Based on the raw data provided, it appeared that the laboratory did not spike the aqueous matrix spike/matrix spike duplicate sample with 1,4-dioxane in the Method 8240 analysis. The laboratory confirmed that the CLP spiking solution was used for the Method 8240 analysis, and this solution does not contain 1,4-dioxane. In addition, the laboratory did

not spike a solid sample with the spiking solution. Therefore, it is the opinion of the data reviewer that the laboratory did not perform a matrix spike/matrix spike duplicate analysis for the analytical method used for the determination of 1,4-dioxane in the project samples.

2. The laboratory supplied the analytical results for the method detection limit (MDL) study for the analysis for 1,4-dioxane. However, it is the opinion of the data reviewer that the laboratory did not perform the MDL study according to good laboratory practice. The laboratory should have analyzed the low-spike samples seven times over three non-consecutive days. In addition, since the samples are analyzed over three nonconsecutive days, at least three different continuing calibrations should be used for quantitation of the analytical results. For instrument MSD/C, all seven analyses were performed within six hours of each other, and the quantitation was based on one continuing calibration. For instrument MSD/B, the laboratory performed the analysis of the seven low-spike samples on three consecutive days, and apparently the quantitation was based on one continuing calibration standard (despite the fact that the analysis covered three days). The data reviewer has examined the raw data from the initial calibration standards and has determined that the laboratory could detect 1,4-dioxane in the solid samples at the reported quantitation limit (1 mg/Kg). The instrument used for the aqueous samples did not detect 1,4-dioxane in the 0.5 mg/L initial calibration standard but did detect the compound in the 1.5 mg/L initial calibration standard. The ability to observe the compound in the 0.5 mg/L standard for the solid sample analysis but not the aqueous sample analysis may be due to the heated purge used in the volatile organics analysis of solid samples. Without other supporting raw data or quality control summaries, the quantitation limit for 1,4-dioxane in the aqueous samples should be considered to be 1.5 mg/L (the concentration of the compound in the lowest calibration standard in which the compound was detected).

3. It should be noted that the EPA Region II data validation checklist used for the review of the laboratory data was expressly developed for use with the CLP protocols and not for analyses performed using SW-846 methods. The data reviewer has used the checklist as guidance for the review of the data from the 1,4-dioxane and 2-butoxyethanol analyses; however, it should be remembered that some requirements stipulated in the checklist are not applicable to the SW-846 analyses and were not considered during the data validation. Notations in the checklist indicate which applications were considered inappropriate for the analyses.

4. SW-846 Method 8240 requires that the mass ion m/e=173 be less than or equal to 2.0% of the mass ion m/e=95 in the bromofluorobenzene mass tuning and calibration. The laboratory used a more restrictive 1.0% criterion. All mass tuning and calibration runs met the requirements of both the method and the laboratory.

5. The laboratory did not report the relative response factors or percent differences for the calibration check compounds (CCCs) or the system performance check compounds (SPCCs) on the initial and continuing calibration summary forms for the analysis of 1,4-dioxane in the project samples. However, the analysis for this compound was performed in conjunction with the volatile organics analysis of the samples by CLP protocols. The initial and continuing calibration summary forms in the volatile organics data package were used by the data reviewer to assess instrument stability and performance.

6. It should be noted that the concentration levels that are reported on the initial calibration summary forms for the 1,4-dioxane analysis (20 ppb, 50 ppb, 100 ppb, 150 ppb and 200 ppb) are for the surrogate compounds only. The laboratory used concentrations of 500 ppb, 1500 ppb, 2500 ppb, 4000 ppb and 5000 ppb for 1,4-dioxane in the initial calibration for the Method 8240 analysis.

7. The laboratory did not spike all samples and standards with a surrogate compound for the analysis of the project samples for 2-butoxyethanol as recommended in the method (SW-846, 8015A-4). Therefore, sample-specific purge efficiencies and matrix effects could not be assessed by the data reviewer.

8. The laboratory did not include the raw data and chromatogram for the analysis of the Procedure Blank and the chromatogram for sample Boring 2 (30-32) in the sample data package for 2-butoxyethanol submitted for validation. Upon notification, the laboratory provided the requested information.

9. The data reviewer noted that compounds benzo(b)fluoranthene and benzo(k)fluoranthene coeluted in the low-level initial calibration standard (and the laboratory reported the same relative response factors for the compounds on the initial calibration summary form) but the compounds were resolved in the higher concentration standards. Because no positive results were reported for the compounds in the semivolatile organics analysis of the project samples, this occurrence should have no effect on data quality.

With regard to data usability, the principal areas of concern are blank contamination, exceeded holding times, very low surrogate recoveries, very low relative response factors and high percent differences in the continuing calibrations. Based upon the data packages reviewed, the following organic data qualifiers are offered. It should be noted that the following organic data usability issues represent an interpretation of the quality control results obtained for the project samples. Quite often, data qualifications address issues relating to the sample matrix problems. Similarly, the validation guidelines routinely specify areas of the data that require qualification, yet the methods used for analysis do not require any corrective action by the laboratory. Accordingly, the following data usability issues should not necessarily be construed as an indication of laboratory performance.

Organic Data Qualifiers

- Due to the presence of methylene chloride in the associated field blank and the presence of common phthalate esters in the method and field blanks, the positive results for methylene chloride in sample Boring 2 (35-37) and for di-*n*-butylphthalate, diethylphthalate, and *bis*(2-ethylhexyl)phthalate in all samples except the field blank should be considered "not-detected" and have been flagged "U" on the data table. In addition, all results reported at concentrations below the contract required quantitation limits (CRQLs) have been raised to the CRQL (corrected for percent solids for the soil samples) and assigned the appropriate "U" qualifier code.

- The analysis for 1,4-dioxane in all samples should be considered unreliable and the "not-detected" results for the compound in the project samples have been flagged "R" on the data summary tables. Very low relative response factors (<0.050) were reported for the compound in the initial and continuing calibrations performed for the volatile organics analysis. According to U.S. EPA Region II guidelines, the analysis is unreliable. However, it should be noted that the compound has historically displayed poor responses when analyzed by SW-846 Method 8240. The calibrations and method detection limit study performed for the analysis for the compound show that the analysis was capable of detecting the compound at the reported quantitation limits (1 mg/Kg) for solid samples. Therefore, although the validation guidelines require that the analysis for 1,4-dioxane in the project samples be considered unreliable, it is the opinion of the data reviewer that the analysis was capable of determining the presence or absence of the compound in the solid project samples at the reported quantitation limits and at a quantitation limit of 1.5 mg/L for the aqueous samples (See Noncorrectable Deficiency #1 and Comment #2).

- The analyses for all compounds in the semivolatile organics analysis of the initial and reextraction/reanalysis of sample Boring 2 (35-37) should be considered unreliable and the "not-detected" results for the semivolatile compounds in the sample have been flagged "R" on the data summary tables (unless previously flagged "U"). The initial semivolatile organics analysis of the sample displayed very low recoveries (<10%) for all surrogate compounds. Per CLP protocol, the laboratory reextracted and reanalyzed the sample. Acceptable recoveries were obtained in the reanalysis of the sample; however, the sample was reextracted greater than 28 days from sample collection. According to Region II guidelines, both analyses of the sample should be considered unreliable.

- The actual detection limits for chloromethane, bromomethane, vinyl chloride, 2-hexanone and 4-methyl-2-pentanone in all solid samples may be higher than reported and have been flagged "UJ" on the data tables. High percent differences (>25%) in the direction of a decrease in instrument sensitivity were observed between the response factors for these compounds in the associated continuing calibrations compared to the average response factors calculated from the associated initial multipoint calibration.

- The actual detection limits for 2-hexanone, nitrobenzene, 2,4-dichlorophenol, hexachlorobutadiene and pentachlorophenol in all aqueous samples should be considered estimated and have been flagged "UJ" on the data tables. High percent differences (>25%) in the direction of an increase in instrument sensitivity were observed between the response factors for these compounds in the associated continuing calibrations compared to the average response factors calculated from the associated initial multipoint calibrations. It should be noted that although qualification was stipulated by the Region II checklist, the percent differences were in the direction of an increase in instrument sensitivity; therefore, the actual detection limits may be acceptable as reported.

- Per CLP reporting conventions, all results below the quantitation limits should be considered estimated and have been flagged "J" on the data tables.

- Tentatively Identified Compounds (TICs) have been examined and the results appear on the data tables as part of Section 2. The majority of the TICs appear to be blank contamination and laboratory artifacts. Sample MW-5S displayed a significant number of TICs; the majority of these were classified as unknowns due to poor compound mass spectrum matches obtained in the library searches. All TICs identified as laboratory artifacts and blank contamination have been flagged "R" on the data summary tables and should be considered unreliable. All other TIC results have been flagged "J" on the data summary tables and should be considered estimated because the quantitation of the positive results for these compounds assume a relative response factors of 1.0 (per CLP methodology).

A complete support documentation of this organic quality assurance review is presented in Section 3 of this report. The Region II organics analysis data validation SOP checklist is presented in Section 4 of this report.

B. Conclusions

This quality assurance review has identified several aspects of the analytical data that have required qualification. Overall, the majority of the data appears to represent good qualitative and quantitative analyses, although some analyses were qualified due to blank contamination, exceeded holding times, surrogate compound recoveries and calibration issues. To confidently use any of the analytical results from the data sets examined, the data users should understand the qualifications and limitations stated in this report.

Reported prepared by:

Donald J. Lancaster
Senior Quality Assurance Chemist II

Report reviewed by:

Ruth L. Forman
Senior Quality Assurance Chemist II

Report reviewed and approved by:

Rock J. Vitale, CPC
Director of Chemistry

ENVIRONMENTAL STANDARDS, INC.
1140 Valley Forge Road
P.O. Box 911
Valley Forge, PA 19482

(610) 935-5577

Date: 4/6/95

SECTION 2

ANALYTICAL RESULTS

Organic Qualifiers

-	Compound was not detected.
U	This compound should be considered "not-detected" since it was detected in a blank at a similar level.
J	Quantitation is approximate due to limitations identified during the quality assurance review (data validation).
R	Unreliable result - Compound may or may not be present in this sample.
UJ	This compound was not detected, but the quantitation limit is probably higher due to a low bias identified during the quality assurance review.

It should be noted that the results for all solid samples are reported on a "dry-weight" basis.

JUN 0 2 1995

K.B. Company
c/o Jerry Speigel Associates
375 North Broadway
Gericho, New York 11753

Dean Anson
Anson Environmental
33 Gerard Street
Huntington, NY 11743

Attention: Arthur D. Sanders,
President

Richard G. Leland, Esq.
Rosenman and Colin
575 Madison Avenue
New York, New York 10022-2585

Re: Anchor Chemical Superfund Site; Final Risk Assessment

Dear Sirs:

Attached is the final Risk Assessment report for the Anchor Chemical Superfund Site. The assessment is based on groundwater, soil and sediment analytical data for the samples taken in April and November 1992. As you know, the samples were collected as part of the remedial investigation study (RI) of the site. Risks were calculated for human exposure due to ingestion of groundwater, soil and sediments and inhalation of contaminants, which may volatilize from groundwater during showering.

The additional groundwater and soil samples, which were collected during February 1995 to complete the RI, were not included in the final Risk Assessment since the data showed minimal contamination.

Any questions on this matter, should be directed to Mr. Tom Taccone at (212) 637-4281.

Sincerely yours,

Carole Petersen, Chief
NY/Caribbean Superfund Branch II

Attachment

cc: G. Ferreira, ERRD-PSB
J. Greco, NYSDEC
S. Boone, CDM
J. O'Brien, Esq.

bcc: J. Doyle, ORC

FINAL RISK ASSESSMENT
ANCHOR CHEMICAL SITE
HICKSVILLE, NEW YORK

Prepared for

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

Work Assignment No.:	C02125
EPA Region:	II
EPA Site/Facility I.D. No.:	NYD001485226
Contract No.:	68-W9-0003 (TES-6)
TRC Document No.:	L93-667
TRC Project No.:	1-635-354-0-2PG1-0
TRC Project Manager:	Susan Stoloff
Telephone No.:	(508) 970-5757, ext. 5271
Subcontractor No.:	N/A
Subcontractor Project Manager:	N/A
Telephone No.:	N/A
EPA Work Assignment Manager:	Tom Taccone
Telephone No.:	(212) 264-6321
Date Prepared:	April 1, 1994

TRC ENVIRONMENTAL CORPORATION
291 Broadway, Suite 1206
New York, NY 10007
(212) 349-4616

TRC

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1.0 INTRODUCTION

1.1 Overview

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

As part of the RI oversight effort at the Anchor Chemical site in Hicksville, New York, TRC Environmental Corporation (TRC, formerly Alliance Technologies Corporation) conducted a baseline risk assessment, which includes both a human health and an ecological risk assessment. This effort was conducted under EPA Contract No. 68-W9-0003 (TES-6), Work Assignment C02125. The risk assessment focuses on chemical contamination associated with the former Anchor Chemical property as well as impacted adjacent residential/commercial properties. The human health risk assessment presented in this report is primarily a quantitative analysis based on RI field sampling and analysis results. The ecological risk assessment is qualitative; it is based on previously published information and data collected during the RI.

The risk assessment evaluates actual or potential exposures to site chemical contaminants under current and future land use scenarios. The RI Report for the Anchor Chemical site, prepared for K.B. Company by Anson Environmental Ltd. (Anson, 1993), has been used to determine likely receptors and exposure pathways for current and future land use and demographic scenarios. Receptors evaluated in the human health risk assessment include the following:

- Future Residents; and
- Future Excavation Workers.

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

The ecological risk assessment is composed of four parts:

- Problem Formulation,
- Exposure Assessment,
- Ecological Effects Assessment, and
- Risk Characterization.

Due to the nature and extent of contamination at this site, the opportunities for impacts to ecological receptors are minimal. As a result, the Anchor Chemical site ecological risk assessment is comprised only of two steps, Problem Formulation and Risk Characterization. Problem Formulation describes ecological characteristics including habitats and species at the site and in the vicinity. Risk Characterization evaluates potential risks to biota based on available information.

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

- *Superfund Exposure Assessment Manual.* April, 1988. Office of Remedial Response (EPA, 1988a).
- *Risk Assessment Guidance for Superfund (RAGS). Volume I - Human Health Evaluation Manual (Part A),* December 1989, Interim Final (EPA, 1989a).
- *Risk Assessment Guidance for Superfund (RAGS). Volume II - Environmental Evaluation Manual*, March, 1989, Interim Final. Office of Emergency and Remedial Response (EPA, 1989b).
- *Ecological Assessment of Hazardous Waste Sites: A Field and Laboratory Reference.* March 1989. U.S. EPA Environmental Research Laboratory, Corvalis, OR (EPA, 1989e).
- *Guidance for Data Useability in Risk Assessment*, April 1992, Interim Final. Office of Emergency and Remedial Response (EPA, 1992f).
- *Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors".* March 1991. Office of Solid Waste and Emergency Response (EPA, 1991a).
- *Ecological Assessment of Superfund Sites: An Overview.* December 1991. U.S. EPA Office of Emergency and Remedial Response, ECO Update Vol. 1(2) (EPA, 1991b).
- *Dermal Exposure Assessment: Principles and Applications.* Interim Report. Office of Research and Development. January 1992 (EPA, 1992a).
- *Guideline for Predictive Baseline Emissions Estimation Procedures for Superfund Sites.* January 1992 Interim Final (EPA 1992k).
- *Supplemental Guidance to RAGS: Calculating the Concentration Term.* May 1992. Office of Solid Waste and Emergency Response (EPA 1992j).

The report is organized into the following sections:

- Section 1 - Introduction
- Section 2 - Data Evaluation
- Section 3 - Contaminant Fate and Transport

- Section 4 - Human Health Risk Assessment
- Section 5 - Ecological Risk Assessment
- Section 6 - Summary and Conclusions
- Section 7 - References

Appendices provide supporting information for relevant sections of the text.

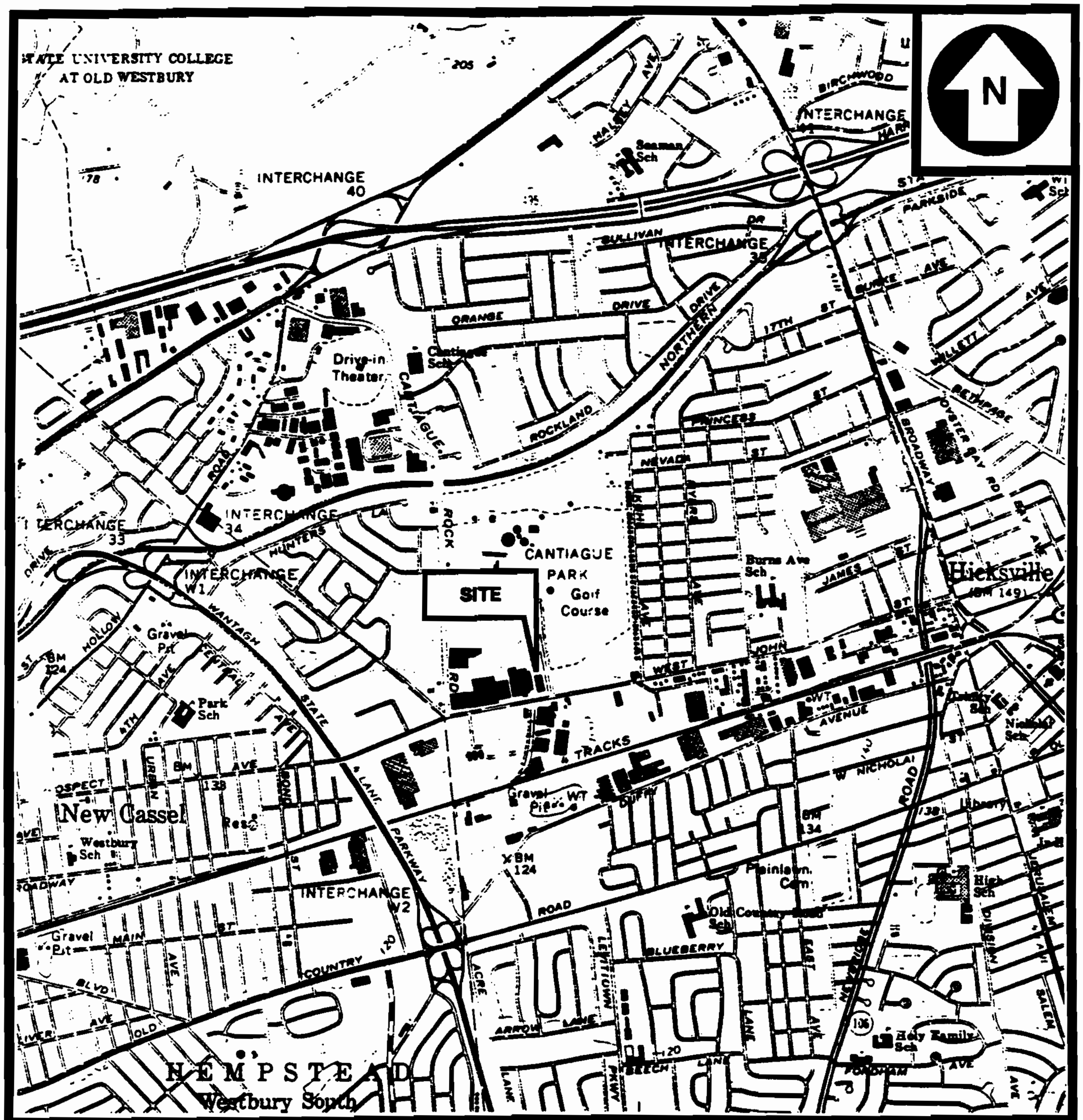
The following sections, 1.2 Site Description, 1.3 Site History, and 1.4 Summary of Site Investigations, summarize applicable information provided in the RI Report (Anson, 1993). Section 1.5, Summary of Site Contamination, is a brief description of ground water, soil, and sediment contamination at the site.

1.2 Site Description

The study area for this risk assessment includes the former Anchor Chemical property, which is located at 500 West John Street in the Village of Hicksville, Town of Oyster Bay, Nassau County, New York (see Figure 1-1). The area surrounding the site is predominantly industrialized but also recreational in nature. Prior to the initial development of this land in 1964, the site was used for agricultural purposes.

The site's total area is 1.5 acres and includes one two-story building, 25,850 square feet in size. It is bordered to the west by commercial property, to the south by West John Street, and to the northeast by Cantiague Park, a 125-acre recreational facility. In addition, a ground water recharge basin is situated in close proximity to the east of the site. Public water supply wells are located within three miles of the site; however, there are no known private residential drinking water wells within the area.

Currently, seventeen inactive storage tanks are located two feet below the concrete floor in the northeast corner of the building. These tanks previously stored chemicals used in manufacturing processes at the site. Nine on-site drywells and one drain



BASE MAP IS A PORTION OF THE FOLLOWING USGS 7.5' SERIES QUADRANGLE:
HICKSVILLE, NY, 1967; PHOTOREVISED 1979

QUADRANGLE LOCATION

LOCATION MAP

ANCHOR CHEMICAL PROPERTY
HICKSVILLE, NEW YORK

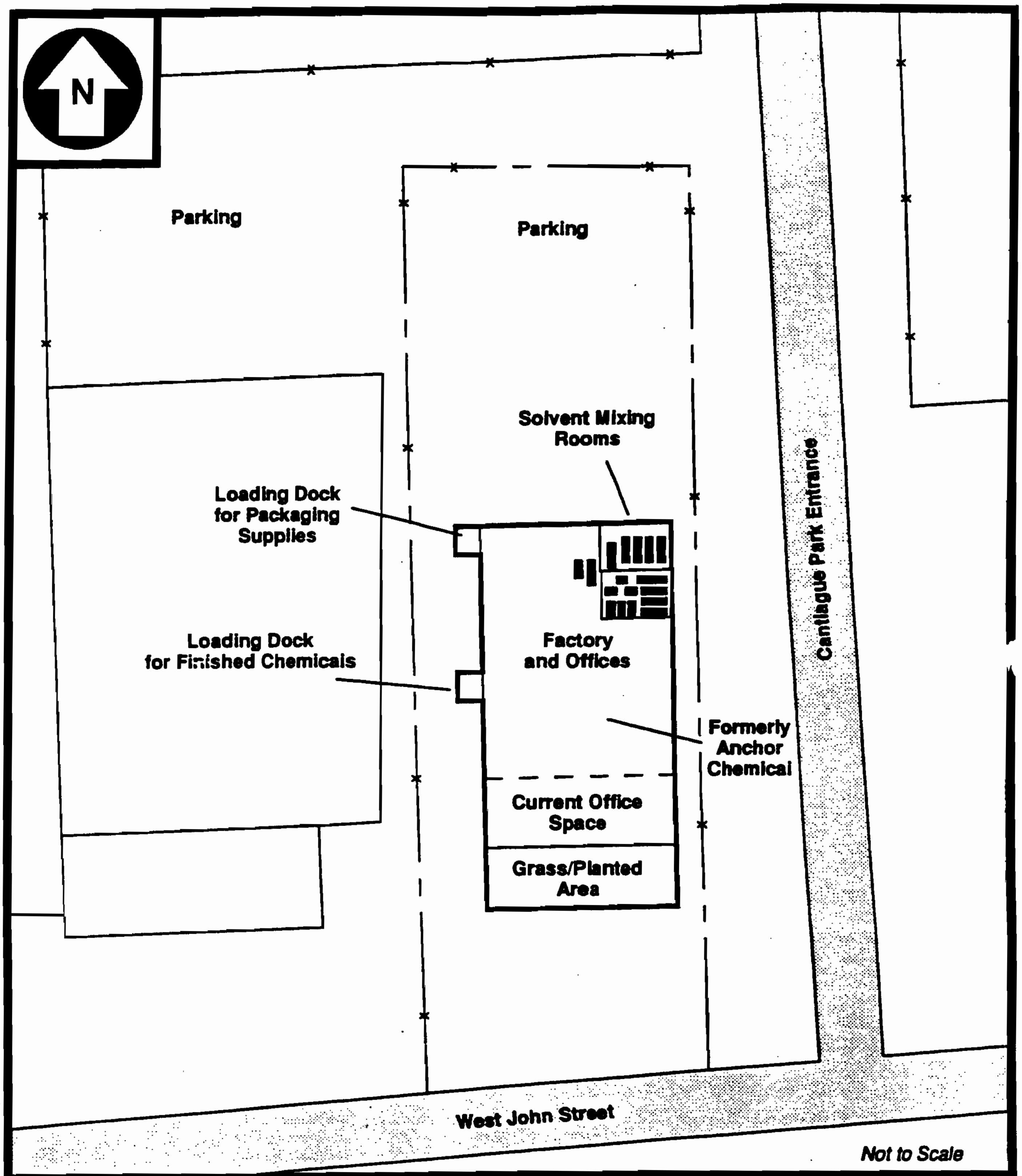
collect surface runoff from the pavement. These drywells are not connected to a sewer system, but instead drain directly into the soils.

The topography of the Anchor Chemical area is basically flat, urban land, with at least 85 percent of the surface covered with asphalt or concrete. The surrounding areas are comprised mostly of parking lots, shopping centers, or industrial facilities, and are nearly level or gently sloping. There are no wetlands in the area of the site. Ground water generally flows to the southwest from the site.

1.3 Site History

On September 30, 1964, the K.B. Company (Jerry Spiegel) purchased the site. At that time, as is previously noted, the site was undeveloped and had been used for agricultural purposes only. From 1964 to 1978, Anchor Chemical Company leased the site's newly constructed building and began manufacturing, blending, and storing chemicals for the graphic arts industry. The company maintained two solvent mixing rooms and several container and drum storage areas. Seventeen underground storage tanks (USTs) with capacities ranging from 500 to 4,000 gallons were installed under the mixing rooms in 1964. In addition, seven aboveground storage tanks, ranging in size from 550 to 1,500 gallons, occupied the blending rooms and were reported to contain chemical products (see Figure 1-2). These tanks are no longer on-site; it is believed they were removed in 1985.

In 1978, Anchor Chemical Company changed its name to Anchor/Lith Kem-Ko but continued to manufacture, blend, and store chemicals. From 1985 to 1988, Emery Worldwide Freight, a shipping company, leased the building from K.B. Company, and from 1988 to 1992, J.D. Brauner manufactured furniture on the site. Currently, Distributors of America, a company which distributes marketing flyers, occupies the facility, which is still owned by K.B. Company.



☐ Building
—✕— Fence
— - — Approx. Property Boundary
☐ Paved Roads
▬ Underground Storage Tanks

SITE SKETCH

ANCHOR CHEMICAL SITE
HICKSVILLE, NEW YORK

TRC

Figure 1-2.

1.4 Summary of Site Investigations

In 1977, the Nassau County Department of Health (NCDH) discovered 1,1,1-trichloroethane, trichloroethylene, and tetrachloroethylene in samples of liquid obtained from a drywell located north of the building in a parking lot. In response, Anchor/Lith Kem-Ko submitted a spill prevention plan to NCDH and sealed all lines leading to the drywell, including those originating from the floor drains located in the mixing rooms.

In May of 1981, the Nassau County Fire Marshall notified Anchor/Lith Kem-Ko that its USTs had neither been tested nor registered. In a subsequent inspection of 14 of the USTs, five failed air over product tests and were thus decommissioned. During the testing of the three remaining tanks in 1982 and 1983, one tank failed the hydrostatic test and was found to be leaking. In 1982, NCDH requested that Anchor/Lith Kem-Ko investigate the possibility of ground water and soil contamination at the site. In January 1983, the site was added to the National Priorities List (NPL). Anchor/Lith Kem-Ko contracted Lockwood, Kessler & Bartlett (LKB) to install monitoring wells and analyze samples.

LKB's results revealed concentrations of methylene chloride and 1,1,1-trichloroethane in the soils, and tetrachloroethylene, dichloroethane, methylene chloride, and trichloroethylene in the ground water at levels which exceeded both Federal and State standards (LKB, 1985). Subsequent sampling from the same three existing monitoring wells between October 1987 and early 1991 indicated that the concentrations of these volatile organic compounds had significantly decreased from 30,000 ppb in 1982 to 9 ppb in 1991 (Roux, 1991). In 1991, K.B. Company retained Anson Environmental Ltd. and Blasland Bouck Engineers, P.C. to implement the RI.

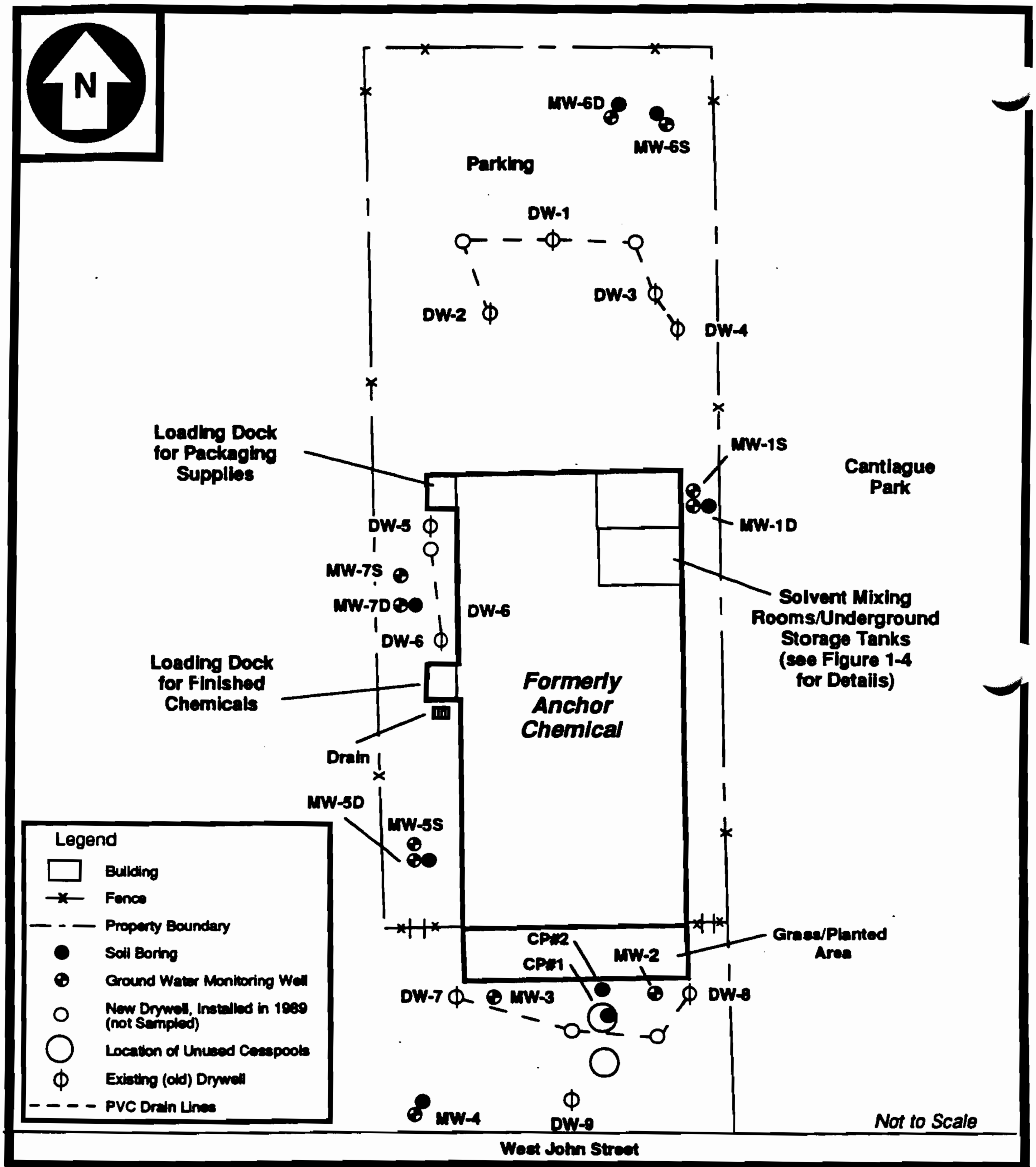
1.5 Summary of Site Contamination

Contamination is discussed separately for each medium evaluated at the Anchor Chemical site. The discussion makes note, where appropriate, of chemicals that tend to contribute most to risk based on carcinogenic potency or toxicity. Media evaluated include ground water, sediments, and soils. Appendix A presents chemical contamination summary statistics. Figure 1-3 shows approximate on-site sampling locations and Figure 1-4 shows locations of indoor soil borings.

1.5.1 Ground Water Contamination

Chemical Contamination

Ground water samples were collected from 11 sampling locations at the Anchor Chemical site. One pesticide, heptachlor epoxide, was detected at a maximum concentration of 0.08, which is below its Maximum Contaminant Level (MCL). However, one base/neutral and extractable compound (BNA) and two metals were detected at concentrations which exceeded their MCLs. Chrysene, a polyaromatic hydrocarbon (PAH) with an MCL of 0.2 µg/L was detected at 1.00 µg/L; chromium was reported at a maximum concentration of 1,440 µg/L which exceeds its MCL by over 14 orders of magnitude; and lead, whose action level is 15 µg/L, was detected at concentrations ranging from 10.2 to 240 µg/L. The highest detected concentrations for both metals were detected at well MW-2. The five Volatile Organic Compounds (VOCs), eight BNAs, and one pesticide were detected relatively infrequently (detection rates ranged from one to 13 out of 22), whereas of the 18 inorganics detected, 13 were reported 17 times or more.



APPROXIMATE GROUND WATER, SOIL, AND SEDIMENT SAMPLING LOCATIONS

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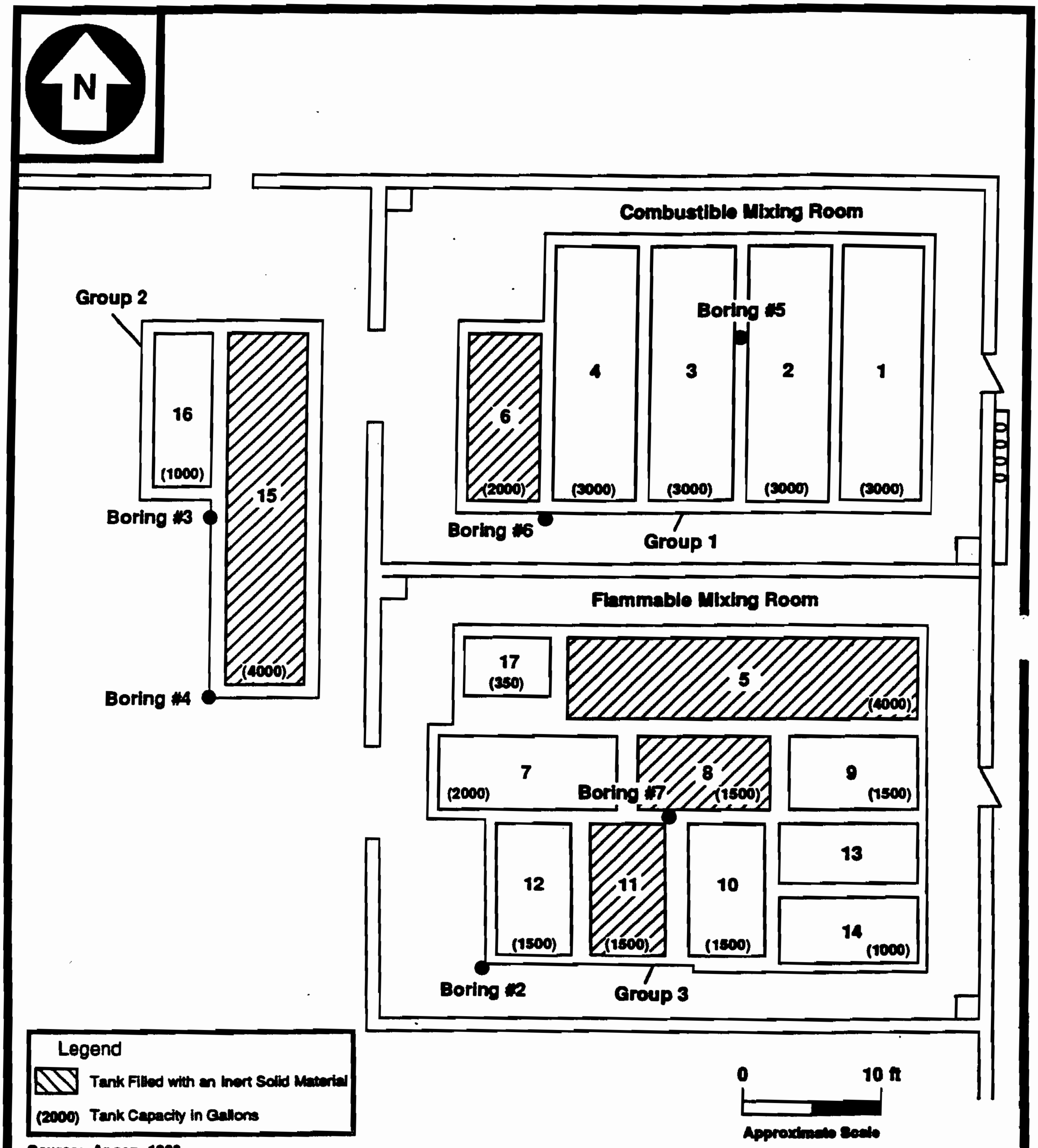
ANCHOR CHEMICAL SITE
HICKSVILLE, NEW YORK

TRC

Figure 1-3.

M568/2/02



Source: Anson, 1993

LOCATION OF INDOOR BORINGS AND UNDERGROUND STORAGE TANKS

ANCHOR CHEMICAL SITE
HICKSVILLE, NEW YORK

MS69/2/03

1.5.2 Soil Contamination

For the purposes of assessing risks associated with soil exposures at the Anchor Chemical site, soils data have been separated into subsurface and deep subsurface depth profiles. Boring samples for soils collected at depths below the surface but no deeper than 15 feet were included in the subsurface soil grouping, while soils from depths greater than 15 feet were included in the deep subsurface grouping. No surface soil samples were collected because most of the site is covered with pavement or the building.

Subsurface Soil Chemical Contamination

BNAs, two pesticides, and inorganics were detected at the six on-site subsurface sample locations. Of the 14 metals detected, six of the reported maximum concentrations were from boring location IB-1 at depths of 10 to 12 feet. Arsenic concentrations ranged from 0.55 to 1.50 mg/kg and lead concentrations ranged from 2.0 to 13.6 mg/kg. The detected BNAs included 4-chloroaniline and bis(2-ethylhexyl) phthalate which were detected at maximum concentrations of 78 and 190 µg/kg, respectively. The two pesticides detected, dieldrin and methoxychlor, were reported at maximum concentrations of 7.50 µg/kg and 14.00 µg/kg, respectively, in sample CP#2 collected from one of the unused cesspools.

Deep Subsurface Soil Chemical Contamination

One VOC (2-butanone), one BNA (bis(2-ethylhexyl) phthalate), and one pesticide (heptachlor) were detected at depths of 15 feet or greater. However, several inorganics were reported more than nine times from the 11 sampling locations. Heptachlor was detected only once at a maximum concentration of 0.20 µg/kg. Lead was detected 13 times in the 17 samples analyzed, but at relatively low concentrations ranging from 0.62 to 2.10 mg/kg. Maximum concentrations of metals were detected at boring IB-3.

1.5.3 Sediment Contamination

For the purposes of assessing risks associated with sediment exposures at the Anchor Chemical site, data have been separated into sediment and deep sediment depth profiles. Boring samples for sediments at depths less than 15 feet were included in the first grouping, while sediments from depths greater than 15 feet were included in the latter grouping.

Sediment Contamination (Less than 15 feet)

A total of 46 analytes were detected at 10 drywell sediment locations on the Anchor Chemical site. All of the VOCs, BNAs, and pesticides had relatively low frequencies of detection (an average detection rate of three out of ten), whereas metals were reported more often (an average detection rate of eight out of ten). Arsenic, a known human carcinogen, was detected in nine out of 10 samples analyzed, at concentrations ranging from 0.79 to 3.7 mg/kg. Twelve other metals were found in every sediment sample evaluated. Of the six detected VOCs, total xylenes were reported at a maximum concentration of 67,000 µg/kg, while carbon disulfide concentrations ranged between 5 and 21 µg/kg. The majority of the BNA compounds were either PAH, or phthalates. Of the seven pesticides, alpha-BHC was found in one of the nine samples analyzed at a concentration of 183 µg/kg, and beta-BHC was detected in one sample at a concentration of 8.20 µg/kg. Four metals, aluminum, iron, lead, and vanadium were detected at boring DW-8 at maximum concentrations ranging from 81.00 (vanadium) to 22,700 mg/kg (iron).

Deep Sediment Contamination

Nine deep sediment samples were collected from different depths at four drywells at the Anchor Chemical site. All VOCs, BNAs, and pesticides/PCBs were detected only once and all in a sample collected from location DW-2, while the frequency of detection for the inorganics ranged from one to nine out of approximately nine samples. Metals detected most frequently (nine times) included aluminum, iron, lead, manganese, vanadium, and zinc. The VOC total xylenes had the highest reported maximum concentration at 82,000 µg/kg, with ethylbenzene and toluene following at 4,800 µg/kg, and 2,300 µg/kg, respectively. Of the four pesticide/PCBs detected, three were found at relatively low concentrations (1.10, 1.30, and 2 µg/kg), while aroclor-1254 was detected at 230 µg/kg. Arsenic and cyanide were reported at concentrations ranging from 0.87 to 2.30 mg/kg and 1.60 to 6.20 mg/kg, respectively.

2.0 DATA EVALUATION

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

2.1 Data Sources

The environmental data used in this risk assessment were generated from samples taken during the Remedial Investigation (RI). All hardcopy data provided to TRC from EPA and presented in the RI report were manually entered into dbase computer format via LABDATA, TRC's proprietary data entry program. TRC resolved questions about the data by contacting EPA and Anson Environmental directly (TRC, 1993a). A 100 percent quality control check against the data presented in Volumes 2 and 3 of the RI report was performed to ensure the integrity and accuracy of the data base.

2.2 Data Review

Table 2-1 presents a list of all samples and analytical parameters evaluated for the risk assessment.

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

- detection limits,
- data validation qualifiers,
- sample duplicates, and
- sample dilutions and re-analyses.

The validated RI data were provided in data tables that contained sample specific detection limits for analytes not detected. Validation qualifiers were treated according

TABLE 2-1. SAMPLES INCLUDED IN THE ANCHOR CHEMICAL RISK ASSESSMENT

Medium/Area	Sample Number	Volatile Organics	BNAs	Pesticide/ PCBs	Inorganics
Ground Water	MW-1D-II	X	X	X	X
Ground Water	MW-1D	X	X	X	X
Ground Water	MW-1S-II	X	X	X	X
Ground Water	MW-1S	X	X	X	X
Ground Water	MW-2-II	X	X	X	X
Ground Water	MW-2	X	X	X	X
Ground Water	MW-3-II	X	X	X	X
Ground Water	MW-3	X	X	X	X
Ground Water	MW-4-II	X		X	X
Ground Water	MW-4-II/DP	X	X	X	
Ground Water	MW-4-II/RE		X		
Ground Water	MW-4	X	X	X	X
Ground Water	MW-4/DP	X	X	X	X
Ground Water	MW-5D-II	X	X	X	X
Ground Water	MW-5D	X	X	X	X
Ground Water	MW-5S-II	X	X	X	X
Ground Water	MW-5S	X	X	X	X
Ground Water	MW-6D-II	X	X	X	X
Ground Water	MW-6D	X	X	X	X
Ground Water	MW-6S-II	X	X	X	X
Ground Water	MW-6S	X	X	X	X
Ground Water	MW-7D-II	X	X	X	X
Ground Water	MW-7D	X	X	X	X
Ground Water	MW-7S-II	X		X	X
Ground Water	MW-7S-II/RE		X		
Ground Water	MW-7S	X	X	X	X
Subsurface Soils	IB-1(10-12')	X	X	X	X
Subsurface Soils	IB-2(10-12')	X	X	X	X
Subsurface Soils	IB-2(5-7')	X			
Subsurface Soils	IB-4(10-12')	X	X	X	X
Subsurface Soils	MW-5D(5-7')	X	X	X	X
Subsurface Soils	CP#1 (14-16')	X	X	X	X
Subsurface Soils	CP#2 (14-16')	X	X	X	X
Deep Subsurface Soils	IB-1(15-17')	X	X	X	X
Deep Subsurface Soils	IB-2(15-17')	X	X	X	X
Deep Subsurface Soils	IB-3(25-27')	X	X	X	X
Deep Subsurface Soils	IB-3(30-32')	X			
Deep Subsurface Soils	IB-3(30-34')		X	X	X
Deep Subsurface Soils	IB-3(35-37')	X			
Deep Subsurface Soils	IB-3(35-39')		X	X	X
Deep Subsurface Soils	IB-4(15-17')	X	X	X	X
Deep Subsurface Soils	IB-5(15-17')	X	X	X	X
Deep Subsurface Soils	IB-5(35-37')	X	X	X	X
Deep Subsurface Soils	IB-6(30-32')	X	X	X	X
Deep Subsurface Soils	IB-6(40-42')	X	X	X	X
Deep Subsurface Soils	MW-1D(120-122)	X	X	X	X

TABLE 2-1. (CONTINUED)

Medium/Area	Sample Number	Volatile Organics	BNAs	Pesticide/ PCBs	Inorganics
Deep Subsurface Soils	MW-1D(59-61')	X			
Deep Subsurface Soils	MW-1D(59-63')		X	X	X
Deep Subsurface Soils	MW-4(60-62')	X	X	X	X
Deep Subsurface Soils	MW-4(75-77')	X	X	X	X
Deep Subsurface Soils	MW-5D(115-117)	X	X	X	X
Deep Subsurface Soils	MW-5D(60-62')	X	X	X	X
Deep Subsurface Soils	MW-6D(60-62')	X	X	X	X
Deep Subsurface Soils	MW-6S(74-76')	X	X	X	X
Deep Subsurface Soils	MW-7D(114-116)	X	X	X	X
Deep Subsurface Soils	MW-7D(56-58')	X	X	X	X
Sediments	DRAIN	X	X	X	X
Sediments	DW-1 (Drywell Surface)	X	X		X
Sediments	DW-1/DP (Drywell Surface)				X
Sediments	DW-2 (Drywell Surface)	X		X	X
Sediments	DW-2/RE (Drywell Surface)		X		
Sediments	DW-3 (Drywell Surface)	X		X	X
Sediments	DW-3/RE (Drywell Surface)		X		
Sediments	DW-4 (Drywell Surface)	X	X	X	X
Sediments	DW-5 (Drywell Surface)	X		X	X
Sediments	DW-5/RE (Drywell Surface)		X		
Sediments	DW-6 (Drywell Surface)	X	X	X	X
Sediments	DW-7 (Drywell Surface)	X	X	X	X
Sediments	DW-8 (Drywell Surface)	X		X	X
Sediments	DW-8/RE (Drywell Surface)		X		
Sediments	DW-9 (Drywell Surface)	X	X	X	X
Deep Sediments	DW-1(25-27')	X	X	X	X
Deep Sediments	DW-1(30-32')	X	X	X	X
Deep Sediments	DW-2(15-17')	X	X	X	X
Deep Sediments	DW-2(25-27')	X	X	X	X
Deep Sediments	DW-6(30-32')	X	X	X	X
Deep Sediments	DW-6(35-37')	X	X	X	X
Deep Sediments	DW-7(40-42')	X	X	X	X
Deep Sediments	DW-7(45-47')	X	X	X	X
Deep Sediments	DW-7(45-47')/DP	X	X	X	X
Deep Sediments	DW-7(55-57')	X	X	X	X

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

In most cases, the results of duplicate samples were averaged. The resulting value was the arithmetic mean of positive results or the arithmetic mean of the reported detection limits if both samples were non-detects. Conservatively, if one duplicate sample was a positive result and the other a negative result, the positive result was used. Duplicates of the following RI samples were included in the analytical data:

- DW-7 (45-47'),
- MW-4 (all round I results and organics results for round II), and
- DW-1 (inorganics only).

The RI data included several samples that required dilution or re-analyses during analysis. The decision of whether to use the original or the diluted/re-analyzed result was made on a case-by-case basis. Generally, TRC used the analysis that contained less rejected or estimated data. If the quality of both analyses was equal, TRC used the results of the first analysis. Except for duplicates, only one result per sample was presented in the RI Report.

The initial mercury and cyanide results for drywell sediment samples DW-1 through DW-9 were rejected due to exceeded laboratory holding times. Therefore, these locations were re-sampled and re-analyzed for mercury and cyanide. Conversations with Anson Environmental indicated that the second set of results, and not the initial results, should be incorporated into the risk assessment data base (TRC, 1993b).

2.3 Statistical Analyses

Summary statistics for all chemicals detected in each medium evaluated in the public health risk assessment are presented in Appendix A, and are divided into the following subgroups:

- Ground Water
- Subsurface Soils
- Deep Subsurface Soils
- Sediments
- Deep Sediments
- Subsurface Soils and Sediments
- Deep Subsurface Soils and Deep Sediments

The data groupings are determined by the exposure scenarios developed in Section 4.0, Exposure Assessment, of this report. Tables in Appendix A list frequency of detection, number of samples analyzed (excluding rejected data points), the lowest and highest detected concentrations, the location of the highest concentration, the arithmetic mean concentration, the upper 95-percent confidence limit of the mean (95% UCL), and the lowest and highest observed detection limits for non-detects. All data were analyzed using SAS™, a widely-used statistical software package (SAS Institute Inc., 1988).

As agreed with EPA Region II and in accordance with current Superfund risk assessment guidance (EPA, 1989a), risk calculations for the "reasonable maximum exposure" scenario were based on either the 95% UCL of the mean concentration or the maximum concentration; the lower of these two values was used in human health risk calculations. The 95% UCL was obtained using the method developed by Land (1975) as described by Gilbert (1987). This method is preferred for log normally distributed data, which are typical of environmental sampling programs, and is one of the methods currently recommended by EPA.

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

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

where:

exp = inverse natural log function;

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

s^2 = variance of the mean;

s = standard deviation;

H = H statistic (Land, 1975); and

n = sample size (number of samples analyzed).

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

3.0 CONTAMINANT FATE AND TRANSPORT

3.1 Introduction

This section describes the fate and transport of chemical contaminants detected at the Anchor Chemical site. The discussion integrates the geology, hydrology, and nature and extent of contamination (summarized in Section 1.0) with physical and chemical characteristics of the contaminants detected. The evaluation presented here is qualitative and focuses on the contaminants that are of primary concern from a human health risk perspective. Groups of chemicals (e.g., VOCs) are evaluated together when physical and chemical characteristics are similar. The discussion provides a separate analysis for the following chemical classes: VOCs, BNAs, pesticides, and inorganics. Table 3-1 presents a summary of the physical and chemical properties of the organic COCs based on values obtained from the literature.

3.2 Potential Routes of Migration

Contamination at the site will be transported from potential source areas to uncontaminated areas by the movement of contaminated media via natural processes. In the case of the Anchor Chemical site, chemical contamination source areas include underground storage tanks which were located beneath the building and former drywells. Chemical contamination has been detected in subsurface soils, ground water, and drywell sediments. In general, contaminant movement will occur as ground water moves away from areas of contamination. Site-specific features will influence this movement.

The features which influence the transport and fate of the detected contaminants are medium specific. Ground water transport will depend on the nature of the geologic materials as well as the direction and velocity of ground water flow. The locations of

TABLE 3-1. PHYSICAL AND CHEMICAL PROPERTIES OF ORGANIC COMPOUNDS

Detected Compounds	Chem. Class	Water Solubility (mg/L)	Koc Organic Carbon Partition Coeff. (mL/g)	Log Kow Octanol Water Partition Coeff.	Fish Bioconc. Factor (L/Kg)	Vapor Pressure (mm Hg)	Henry's Law Constant (atm*m3/mol)	Soil Half-Life (days)	Surface Water Half-Life (days)	Ref
Benzene	VOC	1750	83	2.12	5.2	9.52e+01	5.59e-03		1-6	A
Benzo(a)pyrene	PAH	0.0012	6e+06	6.06		5.60e-09	1.55e-06	420-480	0.4	A
Benzo(b)fluoranthene	PAH	0.014	550000	6.06		5.00e-07	1.19e-05		1-2	A
Benzo(k)fluoranthene	PAH	0.0043	550000	6.06		5.10e-07	3.94e-05			A
Bis(2-ethylhexyl)phthalate	BN	0.4		8.73		2.00e-07	2.57e-07			E
BHC (alpha-)	Pest/Herb	1.63	3800	3.90		2.50e-05	5.87e-06			H
BHC (beta-)	Pest/Herb	0.28	3800	3.90		2.80e-07	4.47e-07			H
Butylbenzyl phthalate	BN	42.2		4.8						H
Carbon disulfide	VOC	2940	54	2	0	3.60e+02	1.23e-02			A
Chlordane (gamma-)	Pest/Herb	0.56	140000	3.32		1.00e-05	9.63e-06			H
Chloroaniline (4-)	BN					1.50e-02				G
DDE (4,4'-)	Pest/Herb	0.04	4e+06	7	51000	6.50e-06	6.80e-05			H
Dichloroethane (1,1-)	VOC	5500	30	1.79		1.82e+02	4.31e-03		1-5	A
Dieldrin	Pest/Herb	0.195	1700	3.5	4760	1.78e-07	4.58e-07			A
Diethylphthalate	BN	896		3.22						E
Di-n-butyl phthalate	BN	13	170000	5.6		1.00e-05	2.82e-07			A
Di-n-octyl phthalate	BN	3		9.2		2.00e-01				E
Ethylbenzene	VOC	152	1100	3.15	37.5	7.00e+00	6.43e-03		1.5-7.5	A
Endrin	Pest/Herb	0.024		5.34		2.00e-07	4.17e-06			H
Fluoranthene	PAH	0.206	38000	4.9	1150	5.00e-06	6.46e-06		1-2	A

TABLE 3-1. (CONTINUED)

Detected Compounds	Chem. Class	Water Solubility (mg/L)	Koc Organic Carbon Partition Coeff. (mL/g)	Log Kow Octanol Water Partition Coeff.	Fish Bioconc. Factor (L/Kg)	Vapor Pressure (mm Hg)	Henry's Law Constant (atm*m3/mol)	Soil Half-Life (days)	Surface Water Half-Life (days)	Ref
Indeno(1,2,3-cd)pyrene	PAH	0.00053	2e+06	6.5		1.00e-10	6.86e-08		0.02-2.08	A
Methoxychlor	Pest/Herb	0.003	80000	4.68						H
Methylnaphthalene (2-)	PAH	25.596		3.86			4.81e-04			C
Naphthalene	PAH	30.6		3.35		2.30e-01	4.48e-04			C
Phenanthrene	PAH	1	14000	4.46	2630	6.80e-04	1.59e-04		0.38-2.00	A
Pyrene	PAH	0.132	38000	4.88		2.50e-06	5.04e-06			A
Toluene	VOC	535	300	2.73	10.7	2.81e+01	6.37e-03		0.2	A
Trichloroethane (1,1,1-)	VOC	1500	152	2.5	5.6	1.23e+02	1.44e-02		0.1-7.0	A
Xylene (total)	VOC	198	240	3.26		1.00e+01	7.04e-03		1.5-9.0	A

LEGEND:

VOC	- Volatile Organic Compound
A	- Acid Extractable Organic Compound
BN	- Base/Neutral Extractable Organic Compound
PAH	- Polycyclic Aromatic Hydrocarbon
P/PCB	- Pesticide/Polychlorinated Biphenyl
*	- Estimated Value

REFERENCES:

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

ground water recharge and discharge will also influence the anticipated exposure routes. Surface transport and air emissions of contaminants are considered to be negligible because the entire site is paved or covered by a building.

3.2.1 Ground Water Transport

The hydrogeologic environment in the immediate site vicinity is defined by three overburden water-bearing units (aquifers), the Upper Glacial, Magothy and Lloyd Aquifers. All three aquifers are in hydraulic communication. The Upper Glacial Aquifer is the uppermost unconfined aquifer and is separated from the Magothy Aquifer by discontinuous confining units. In the vicinity of the site, the Magothy and Lloyd aquifers are separated by the Raritan Clay formation, which is approximately 175 feet thick (Anson, 1993). However, to the north of the site, the Lloyd and upper glacial aquifers are in direct communication.

The Upper Glacial aquifer is comprised of mainly sand and gravel deposits with some cobbles in an unstratified mixture. However, the stratigraphy of underlying Magothy aquifer in the Hicksville area is poorly defined (Anson, 1993). The predominant direction of ground water flow in the overburden is expected to be in a horizontal direction, generally flowing towards the south west; however, some vertical recharge of the underlying aquifers is known to occur (Anson, 1993).

The Upper Glacial Aquifer is of concern because it is the direct receptor of near surface contamination. However, the majority of public drinking water wells draw water from the Magothy Aquifers.

Historically, VOC contamination has been documented in monitoring wells located on the site. The most probable source of these contaminants are the documented leaking underground storage tanks. Ground water contamination most likely resulted as contents of the leaking USTs were leached through the underlying soils from precipitation entering the soil via the nine site drywells. These ground water contaminants would be transported in the south-westerly direction of ground water flow.

3.3 Contaminant Persistence and Migration

Organic and inorganic contaminants were identified in ground water, subsurface soils, and drywell sediments. Intermedia transfer of contaminants may occur by a variety of processes. The general processes associated with each medium and contaminant type are discussed below. This discussion is focused on those contaminants which are defined as contaminants of concern (COCs) in Section 4.1, Selection of Contaminants of Concern. This discussion is focused to emphasize the transport characteristics of those contaminants which contribute most to potential site risks.

3.3.1 Ground Water

Volatile Organics

The only volatile contaminant detected more than once in on-site ground water was 1,1,1-trichloroethane at concentrations of 2 to 8 µg/L. This compound was not present in subsurface soils although it was detected at high concentrations (3,300 µg/kg) in one drywell sediment sample. Monitoring well results from the early 1980s detected 1,1,1-trichloroethane in ground water at concentrations as high as 24,000 µg/L (Anson, 1993).

The moderate to high water solubilities of this VOC indicates that it could be transported in the south-westerly direction of ground water flow. The trace concentrations of ground water contaminants are predicted to diminish during horizontal transport from the site as a result of dispersion and dilution within the water column and by adsorption to geologic materials.

Base-Neutral/Acid Extractable Organics

Five BNAs were selected as ground water COCs, however, only one compound, bis(2-ethylhexyl)phthalate (BEHP), had concentrations above the quantitation limit. Bis(2-ethylhexyl)phthalate was also detected in overlying soils, although leaching is unlikely since it has limited water solubility and high affinity for sorption to soils. The relatively low ground water concentration of BEHP will most likely diminish as a result of dispersion and dilution during horizontal transport.

Pesticides/Herbicides/PCBs

No pesticides or PCBs were selected as ground water COCs at the Anchor Chemical site.

Inorganics

The migration of inorganics in ground water is a complex process, dependent on whether the inorganic exists as a dissolved species, suspended particulate, or colloid. The form of the detected inorganics is presently unknown because dissolved species were not characterized in the RI. Dissolved inorganics are generally transported in the direction of ground water flow although concentrations are lessened by dispersion, dilution, and adsorption to geologic materials. Facilitated transport of inorganics by colloids in ground water has been shown to increase the mobility of some inorganics to a dramatic extent.

3.3.2 Subsurface Soils

Volatile Organics

No volatile organics were detected in subsurface soils from a depth of two to 15 feet and only 2-butanone was detected at relatively low concentrations in the greater than 15 feet depth profile. This indicates that solvents that may have leaked from the USTs prior to their decommissioning in 1983 have either completely migrated away from the source area, or have been completely degraded. Both scenarios are possible given the high permeability of the sandy overburden and rapid degradation rates of volatile organic compounds.

Base-Neutral/Acid Extractable Organics

Only two BNAs were detected in subsurface soils: 4-chloroaniline and bis(2-ethylhexylphthalate). Both were reported at concentrations below the contract required detection limits. It is not likely that bis(2-ethylhexylphthalate) will migrate vertically or horizontally since transport is limited as a result of low water solubility and a high octanol-water partition coefficient. It is possible, however, that 4-chloroaniline could migrate slightly due to its higher vapor pressure.

Pesticides/PCBs

No PCBs and only two pesticides--dieldrin and methoxychlor reported at maximum concentrations of 7.5 µg/kg and 14 µg/kg, respectively--were detected in subsurface soils. Only one pesticide, heptachlor, detected in one out of 20 samples analyzed, was reported in deep subsurface soils. The concentration detected was less than 0.3 µg/kg. The low concentrations reported for these pesticides are unlikely to result in significant transfer to other media (e.g., ground water).

Inorganics

Inorganic contaminants can be leached by rainfall infiltrating through drywells and subsurface soils into the ground water. Ion exchange and desorption are the two primary mechanisms which can result in the movement of inorganics from soil to ground water. However, this transport process cannot be predicted because the cation-exchange capacity (CEC) of soils is unknown. Subsurface soils may act as a source of contamination to ground water; however, the extent of this process cannot be evaluated at this time because the pH and eH (oxidation/reduction potential) of the soils and ground water are unknown. These measurements provide information on the acidity and oxidation state of the different media and can be used to predict the equilibrium state of the inorganics in the soil and ground water.

3.3.3 Sediments

Volatile Organics

The six VOCs detected in the drywell sediments include carbon disulfide, 1,1-dichloroethane, 1,1,1-trichloroethane, ethylbenzene, toluene and total xylenes. Of these compounds, 1,1-dichloroethane, 1,1,1-trichloroethane, ethylbenzene, toluene and total xylenes were detected in drywell DW-2 at fairly high concentrations. The chlorinated solvents (1,1-dichloroethane and 1,1,1-trichloroethane) have potential to migrate to ground water given their high water solubilities and low affinities for soils. However, ground water samples taken from DW-2 in the 15 to 17 foot interval do not indicate the presence of these solvents.

The volatile aromatics detected in DW-2 (ethylbenzene, toluene and total xylenes) are also likely to migrate due to high solubilities and vapor pressures, however, they would be less likely to migrate than the chlorinated solvents. There is evidence that these volatile aromatics have migrated, since sediments taken from the 15 to 17 foot interval contain these compounds at similar concentrations.

Base-Neutral/Acid Extractable Organics

Fourteen semivolatile compounds were detected in the sediment samples collected at depths of less than 15 feet. Of these, ten are classified as PAHs and three are phthalates. The transport behavior of PAHs can generally be described as being similar to that of either naphthalene or benzo(a)pyrene. The migration of phthalates is also similar to that of benzo(a)pyrene. In the Anchor sediments, naphthalene and 2-methylnaphthalene would be expected to migrate further than the other PAHs or phthalates due to their higher water solubilities and lower adsorptive tendencies. Results from drywell sediments in the 15 to 17 foot interval verify this premise since only the more mobile PAHs and phthalates are present.

Pesticides/PCBs

Seven pesticides were detected in the sediments. These compounds exhibit transport characteristics that are similar to benzo(a)pyrene and leaching would be expected to be minimal.

Inorganics

Inorganics in sediments will be subject to the same processes as described for subsurface soils. Concentrations of metals, most notably lead, diminished with depth suggesting that the detected metals are not mobile.

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4.0 HUMAN HEALTH RISK ASSESSMENT

4.1 Selection of Contaminants of Concern

A variety of Target Compound/Analyte List chemicals were detected at the Anchor Chemical site in all media evaluated in the risk assessment. The concentration and frequency of detection for all these chemicals are summarized in Appendix A. Contaminants of concern (COCs), a subset of the total list of contaminants detected, represent those contaminants likely to contribute most to the overall human health risk at the site. COCs in each medium were selected based on toxicity and frequency of detection. The elimination of chemicals from the list of COCs is discussed in the following paragraphs.

Due to their low toxicity, the common human nutrients calcium, magnesium, potassium, and sodium were eliminated from further consideration as COCs for all media. The following chemicals were eliminated because they were detected infrequently (<5% detection), and at concentrations below or near the quantitation limits:

- Ground Water: 1,1-dichloroethane, 1,2-dichloroethane (total), acetone, chloromethane, 4-methylphenol, chrysene, fluoranthene, heptachlor epoxide, and cadmium.

Data on background concentrations of inorganics in site soils and ground water presented in the RI report was determined to be inappropriate for use in the COC selection process. Sample analysis results from well MW-1S, identified as being upgradient and therefore representative of background conditions in the RI report, could not be used because they also showed high levels of contamination during pre-remedial sampling events. Data presented in the RI report for off-site soils and ground water could not be used to select inorganic COCs because these data are from investigations not known to be approved by EPA. Finally, soil background inorganic data from an EPA soil treatment publication (EPA, 1984) presented in the RI report and background inorganic data for the Eastern U.S. Region could also not be used because the data is not specific to the Anchor Chemical site or representative of the vicinity soil type. Literature values are generally unreliable since wide variations in inorganic concentrations can occur over small areas; however, this data is presented in Table 4-1 for informational purposes.

Ground water and soil samples collected from locations MW-6S and MW-6D may be considered to be representative of site background conditions due to their apparent upgradient position. However, it is important to note that ground water and soil samples collected from these locations showed detectable concentrations of one or more phthalate compounds (see Appendix A Summary Statistics for MW-6S and MW-6D/Ground Water, and MW-6S and MW-6D/Deep Subsurface Soils) indicating the soils may have been disturbed by anthropogenic activity. As recommended by the EPA, TRC compared the reported maximum concentrations of inorganics in these background samples to the reported maximum concentrations in all site ground water, soil, and sediment samples. This comparison determined that concentrations of inorganics detected in site media exceeded those reported in the "background" samples from locations MW-6S and MW-6D. No detected contaminants were therefore eliminated from further evaluation in the risk assessment based on a comparison to "background" conditions.

Nontarget analytes, or tentatively identified compounds (TICs), also were not included as COCs because the assigned identity of TICs is highly uncertain and the reported concentrations are only estimations, are highly questionable, and may be orders of magnitude higher or lower than actual concentrations (EPA, 1989a).

Table 4-2 lists all COCs and the media in which the COCs were detected.

TABLE 4-1. BACKGROUND CONCENTRATIONS OF INORGANICS IN SOILS OF THE EASTERN U.S. (mg/kg)

Analyte	Conc. Range For Soils In the Eastern U.S.[1]	EPA, 1984[2]
Aluminum	7000 - 100000	--
Antimony	<1 - 8.8	2 - 10
Arsenic	<0.1 - 73	1 - 50
Barium	10 - 1500	--
Beryllium	<1 - 7	0.1 - 40
Cadmium	NA	.01 - 7
Calcium	100 - 280000	700 - 36,000
Chromium	1 - 10000	1 - 1,000
Cobalt	<0.3 - 70	1 - 40
Copper	<1 - 7000	2 - 100
Iron	100 - >100000	5,000 - 50,000
Lead	<10 - 300	2 - 200
Magnesium	50 - 50000	1,200 - 15,000
Manganese	<2 - 7000	200 - 10,000
Mercury	0.01 - 3.4	.01 - 3
Nickel	<5 - 700	5 - 500
Potassium	50 - 37000	1,700 - 33,000
Selenium	<0.1 - 3.9	.1 - 2.0
Silver	NA	.01 - 5
Sodium	<500 - 50000	--
Thallium	2.2 - 23	--
Vanadium	<7 - 300	20 - 500
Zinc	<5 - 2900	10 - 300

LEGEND:

1 - Results from Shacklette and Boerngen, 1984.

2 - Results from Review of In-Place Treatment Technologies for Contaminated Surface Soils, Volume 2-EPA-540/2-84-0036.

TABLE 4-2. ANCHOR CHEMICAL SITE: CONTAMINANTS OF CONCERN

	Ground Water	Subsurface Soils	Sediments
VOCs			
1,1-dichloroethane			X
1,1,1-trichloroethane	X		X
carbon disulfide			X
ethylbenzene			X
toluene			X
total xylenes			X
BNAs			
2-methylnaphthalene			X
4-chloroaniline		X	
benzoic acid			X
benzo(a)pyrene			X
benzo(b)fluoranthene			X
benzo(k)fluoranthene			X
bis(2-ethylhexyl)phthalate	X	X	X
butylbenzylphthalate	X		X
chrysene			X
diethylphthalate	X		
di-n-butylphthalate			X
di-n-octylphthalate	X		
fluoranthene			X
indeno(1,2,3-cd)pyrene			X
naphthalene			X
phenanthrene			X
pyrene	X		X

TABLE 4-2. ANCHOR CHEMICAL SITE: CONTAMINANTS OF CONCERN (CONTINUED)

	Ground Water	**Subsurface Soils**	**Sediments**
PESTICIDE/PCBs			
4,4'-DDE			X
alpha-BHC			X
beta-BHC			X
dieldrin		X	X
endrin			X
gamma-chlordane			X
methoxychlor		X	X
INORGANICS			
aluminum	X	X	X
antimony			X
arsenic	X	X	X
barium	X	X	X
beryllium		X	
cadmium		X	
chromium	X	X	X
cobalt	X	X	X
copper	X	X	X
iron	X	X	X
lead	X	X	X
manganese	X	X	X
mercury	X		X
nickel	X		X
thallium			X
vanadium	X	X	X
zinc	X	X	X

X - Contaminant of Concern

4.2 Exposure Assessment

4.2.1 Introduction

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

- Characterization of Exposure Setting (Section 4.2.2)
    - description of the physical setting
    - identification of potentially exposed populations
- Identification of Exposure Pathways (Section 4.2.3)
    - identification of media of concern
    - identification of actual and potential exposure routes
- Development of Exposure Scenarios (Section 4.2.4)
    - present and future scenarios
    - exposure parameters
- Quantification of Exposure (Section 4.2.5)
    - estimation of exposure point concentrations
    - estimation of exposure doses

The physical characteristics of the site were examined in order to assess adequately the pathways by which human receptors may become exposed to site contaminants. Exposure scenarios were then developed with consideration of demographics, land use, and human behavior patterns. Estimates of exposure doses were calculated for each actual and potential exposure pathway and receptor population, considering both present and future use of the site. In accordance with current EPA guidance, the reasonable maximum exposure (RME) was assessed. Values for intake variables (e.g., consumption rates) were selected so that the combination of all values used to calculate exposure doses will result in conservative but reasonable estimates. As such, not all intake variables represent maximum values. Compounding maximum values for all inputs would result in unrealistically high exposure estimates.

4.2.2 Characterization of Exposure Setting

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

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

Receptors under Present Land Use: Under present land use there are no on-site receptors. The entire site, except for a small landscaped area, is either occupied by a building or paved. All contaminated media are below the ground surface. Therefore, due to the site's physical characteristics, current exposure to contaminated soils is not likely.

The immediate area surrounding the site is zoned for light industry. To the north and east of the site is a park and golf course, and the closest residential area is a quarter mile from the site.

Current exposure to ground water beneath the site is not known to be occurring. However, because the aquifer is a sole source aquifer that serves all of Long Island, the water quality must meet Federal and New York State drinking water requirements.

Receptors under Future Land Use: As stated above, the site is zoned for light industry. Although it is unlikely, residential use could be permitted in the future.

Likely receptors under the future scenario include utility workers (telephone, water, electricity, sewer) who could become exposed to subsurface contaminants during excavation activities.

Other receptors under future land use conditions involve residents who install private domestic wells in locations impacted by site ground water contaminants.

4.2.3 Identification of Exposure Pathways

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

- a source and a mechanism of chemical release into the environment;
- a transport medium by which the released chemical may reach a receptor (e.g., ground water);
- a point of potential contact between the human receptor and the contaminated medium (e.g., individual ingests contaminated ground water); and
- an exposure route (e.g., ingestion).

The sources and mechanisms of chemical releases and transport media are discussed in Sections 1.0 and 3.0 of this report. Points of potential contact and exposure routes are discussed in the following subsections.

4.2.3.1 Media and Exposure Routes of Concern

Potential exposures to site-related COCs were evaluated for the following media:

- ground water,
- subsurface soils,
- sediments, and
- air.

Because the site is predominantly paved or covered by a building, surface soil is not a potential exposure media and, as a result, no "surface" soil samples were collected.

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

Ground Water

As stated above, due to the existence of a public water supply, site ground water is not currently used as a drinking water source. However, site ground water is classified as potable, and it is possible that in the future an area resident may choose to install a private well that could potentially be affected by site contamination. Therefore, exposure of future residents to ground water contaminants is quantitatively evaluated. Employees may also be exposed under future conditions; however, exposure is expected to be of shorter duration and, therefore, less significant. Employee exposure is not evaluated quantitatively.

Future residents may be exposed to site ground water contaminants via ingestion and dermal contact during bathing, showering, or cooking. Residents may also be exposed to volatilized contaminants inhaled during showering. Exposure via ingestion and via inhalation during showering is evaluated quantitatively. Exposure of future residents to ground water contaminants via dermal contact may also occur, but is expected to be less significant than the combined exposure via ingestion and inhalation.

Inhalation of contaminants volatilizing from ground water and migrating into basements is also a potential exposure pathway. However, because depth to ground water in areas of residential development is expected to be greater than 15 feet, below the depth of home foundations, this exposure pathway is considered unlikely and less significant than exposure via ingestion and via inhalation during showering where warm temperatures and aeration serve to increase the rate of volatilization from water. In addition, the on-site depth to ground water is 60 feet which precludes exposure via this pathway to on-site receptors.

Subsurface Soils and Sediments

If the site undergoes further development in the future, workers may be exposed to subsurface soils and drywell sediments from depths of less than 15 feet during excavation activities. Exposure of excavation workers to subsurface soils and sediments by incidental ingestion is evaluated. Per EPA (EPA, 1992a) exposure via dermal contact is evaluated quantitatively only for cadmium since PCBs and dioxins were not detected in these media. Exposure by future excavation workers via the inhalation of contaminants in surface and subsurface soils and sediments is expected to be minimal due to the short duration of exposure.

4.2.3.2 Summary of Exposure Pathways Considered

Table 4-3 summarizes the exposure pathways considered in the risk assessment for both present and future land use, and provides the rationale for their inclusion or exclusion. The following pathways were retained for the purposes of developing exposure scenarios used for quantitative evaluation:

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

TABLE 4-3. ANCHOR CHEMICAL SITE: SUMMARY OF EXPOSURE PATHWAYS

		TIME-FRAME EVALUATED		DEGREE OF ASSESSMENT			
Pathway	**Receptor**	**Present**	**Future**	**Quant.**	**Qual.**	**Rationale for Selection or Exclusion**	**Data Grouping**
Ground Water							
Ingestion of Ground Water	Resident	No	Yes	X		Ground water is domestic and commercial water supply for Nassau County. Current exposure is not known to be occurring.	All ground water samples.
Inhalation of Ground Water Contaminants during Showers	Resident	No	Yes	X		Expected to be less significant than exposure via ingestion.	Same as above.
Inhalation of Contaminants that Volatilize from Ground Water and Seep into Basements	Resident	No	No			Depth to ground water, 60 feet, precludes exposure via this pathway.	
Dermal Contact with Ground Water	Resident	No	Yes		X	Expected to be less significant than exposure via ingestion and inhalation during showering.	
Soils and Sediments							
Incidental Ingestion of Soils	Excavation Worker	No	Yes	X		Entire site is paved, so current exposure to surface soil contaminant is not occurring. Exposure to site soils may occur during excavations for future site development.	All soil samples, including drywell and drain samples, collected to a depth of no more than 15 feet.
Dermal Contact with Soils*	Excavation Worker	No	Yes	X		Entire site is paved, so current exposure to surface soil contaminant is not occurring. Exposure to site soils may occur during excavations for future site development.	All soil samples, including drywell and drain samples, collected to a depth of no more than 15 feet.
Inhalation of VOC Emissions and Particulates from Soils	Excavation Worker	No	Yes		X	Expected to be less significant than exposure via ingestion and dermal contact.	

*Cadmium, PCBs, and dioxin only (if present).

- dermal contact with subsurface soils and sediments; and
- inhalation of contaminants volatilized from ground water during showering.

4.2.4 *Exposure Scenarios*

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

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

The parameters used for each exposure scenario are summarized in Tables 4-4 through 4-7. The tables correspond to the pathways presented in Table 4-3. Note that parameters are summarized only for those pathways evaluated quantitatively. Note also that exposures from dermal contact with subsurface soils and sediments were quantitatively evaluated for cadmium only. EPA currently only recommends quantitatively evaluating dermal contact exposures for cadmium, PCBs, and dioxin (EPA, 1992a). This is due to a lack of relevant exposure data (e.g., absorption rates) to evaluate this pathway. Dioxins and PCBs were not detected in subsurface soils or sediments.

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

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

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

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

TABLE 4-5. EXPOSURE PATHWAY: INHALATION OF CONTAMINANTS VOLATILIZED FROM GROUND WATER WHEN RESIDENTS SHOWER FOR FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Residents	
Contaminant Concentration (mg/cu. m)					
Modeled value based on results associated with on-site wells					
Body Weight (kg)					
Adult	--	--	70	Per EPA Guidance	RAGS Suppl.
Exposure Time (hours/day)					
Adult	0.116 - 0.2	0.158	0.2	90th percentile value for showering	RAGS
Duration of Exposure (years)					
Adult	1 - 70	35	30	90th percentile for time at a single residence	RAGS Suppl.
Exposure Frequency (days/year)	1 - 365	182.5	350	Equivalent to ingestion frequency	RAGS Suppl.
Inhalation Rate (cu. m/hour)					
Adult	--	--	0.6	Value used is an hourly rate that is specific to showering activities	RAGS
Averaging Time (days)					
Adult					
noncarcinogens	--	--	10950	Values used are based on exposure duration for noncarcinogens and lifetime exposure for carcinogens	RAGS
carcinogens	--	--	25550		

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

RAGS Suppl.: U.S. EPA, *Risk Assessment Guidance for Superfund, Vol. I. Supplemental Guidance: Standard Default Exposure Factors*, Interim Final. Office of Emergency and Remedial Response. March 1991.

TABLE 4-6. EXPOSURE PATHWAY: INGESTION OF SITE SOILS FOR FUTURE SCENARIOS

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

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

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TABLE 4-7. EXPOSURE PATHWAY: DERMAL CONTACT WITH SITE SOILS FOR FUTURE SCENARIOS

VARIABLE	RANGE	MIDPOINT	VALUE USED	RATIONALE	REFERENCE
Receptor Population				Excavation Worker	
Body Weight (kg) Excavation Worker	---	---	70	Per EPA Guidance	RAGS Suppl.
Duration of Exposure (years) Excavation Worker	1 - 30	15	1	Per EPA Guidance	RAGS Suppl.
Exposure Frequency (days/year) Excavation Worker	1 - 365	182.5	65	Assume excavation occurs 5 days/week for 3 months	
Skin Surface Area Contacted (sq cm)					
Arms	--	--	2300	Values used are presented in RAGS	RAGS
Hands	--	--	820		
Total Area of These Limbs	--	--	3120		
Soil Skin Adherence Factor (mg/sq cm)	0.2 - 1.0	0.6	0.6	Values used is midpoint of range	DEA
Absorption Factor Cadmium	0.001-0.01	0.005	.01	Per EPA Guidance.	DEA
Averaging Time (days)					
noncarcinogens	--	--	91	Values used are based on exposure duration for noncarcinogens and lifetime for carcinogens	RAGS
carcinogens	--	--	25550		

RAGS Suppl.: *Risk Assessment Guidance for Superfund, Vol. I. Supplemental Guidance: Standard Default Exposure Factors, Interim Final.* Office of Emergency and Remedial Response. March 1991.
RAGS: *Risk Assessment Guidance for Superfund, Volume I,* EPA 540/1-89/002. Office of Emergency and Remedial Response. December 1989.
DEA: *Dermal Exposure Assessment: Principles and Applications,* Interim Report. EPA/600/8-91/911B. Office of Research and Development. January 1992.

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

Under a future exposure scenario, the excavation worker is assumed to be a 70 kg adult exposed to subsurface soils five days a week for three months . The duration of exposure is assumed to be one year as recommended by EPA (EPA, 1991a). These workers are assumed to ingest subsurface soils for five days per week for three months (totalling 65 days).

4.2.5 Quantification of Exposure

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

4.2.5.1 Estimation of Exposure Point Concentrations

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

The exposure point concentrations for soils are expressed in mass per unit weight (mg/kg) and for water in mass per unit volume (mg/L). To represent the reasonable maximum exposures (as defined by EPA, 1989a), the upper 95-percent confidence limit of the arithmetic mean of log transformed data concentration or the maximum concentration was used as the exposure point concentration. The methodology for determining these values was described in Section 2.0. In brief, the upper 95-percent confidence limit concentration was calculated and compared to the maximum concentration; the lower of the two values was used as the exposure concentration. When the upper 95-percent confidence limit was greater than the maximum, it usually indicated a small number of samples and widely distributed data. In these cases, it was believed that the upper 95-percent confidence limit did not adequately represent the available sampling data.

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

Shower Inhalation Model

To evaluate the inhalation of contaminants that volatilize during showering, airborne concentrations of VOCs and BNAs detected in ground water were estimated by the Foster and Chrostowski model (1987). This model uses the ground water exposure point concentrations discussed above to predict volatile emissions. Unlike other models which assume that a certain percentage of volatiles is released into air, this model is dynamic and considers many of the variables that influence the release of VOCs from water and accumulation in closed bathrooms. The model was validated with experimental data from an independent study (Andelman, 1985).

The rate of VOC and BNA emissions from household water and the subsequent inhaled dose per shower event is determined using the following equation:

$$\text{Dose} = \frac{Ir \ x \ S}{BW \ x \ Ar \ x \ 10^6}\left[D_s + \left(\exp\left(-Ar \ x \ D_t\right)\right)/Ar - \left(\exp\left(Ar\left(D_s - D_t\right)\right)/Ar\right)\right]$$

where:

Dose = inhaled dose per shower event (mg/kg-shower)
Ir = inhalation rate (m^3/min)
S = generation rate of volatile compounds ($\mu g/m^3$-min)
BW = body weight (kg)
Ar = air exchange rate (min^{-1})
D_s = shower duration (min)
D_t = total duration in bathroom (min)

This model accounts for inhalation that occurs during and after a shower, while the receptor remains in the bathroom. Application of the model to this site assumes an inhalation rate of 0.6 m^3/hour (EPA, 1989a), an air exchange rate of 1.5 changes per hour which corresponds to "tight homes", a shower duration of 12 minutes, a total duration of 20 minutes spent in the bathroom, and a body weight of 70 kg. The VOC generation rate was calculated from supplemental equations that determine mass transfer of contamination from water to air. These supplemental equations are presented in Appendix D.

4.2.5.2 Exposure Doses

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

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

Where:

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

Each of the exposure parameters from Tables 4-4 through 4-7 were applied to this general equation. The specific exposure dose calculations are presented in Appendix B.

To evaluate noncarcinogenic health effects, exposure point concentrations were calculated for subchronic (short-term), and chronic (long-term) exposure periods. Subchronic and chronic doses were calculated for exposures over a variety of exposure periods depending on the receptor population. These population-specific exposure periods are identified in Tables 4-4 through 4-7. When assessing carcinogenic health effects, only chronic doses were calculated. These were averaged over a 70-year lifetime.

4.3 Toxicity and Dose-Response Assessment

4.3.1 Introduction

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

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

- IRIS (EPA, 1993a);
- Health Effects Assessment Summary Tables (HEAST) (EPA, 1992b); and
- EPA's Environmental Criteria and Assessment Office (ECAO).

A summary of the relevant toxicity values for all COCs used in the risk assessment appears in Table 4-8. The table contains available oral slope factors and weight of evidence classifications for evaluating carcinogenic risks, and oral chronic reference doses (RfDs) and subchronic reference doses (RfDs) used to evaluate noncarcinogenic risks. Interim toxicity values obtained from ECAO (EPA, 1992e) are also included in this table for certain RfDs which were not available in IRIS or HEAST.

Sections 4.3.2 and 4.3.3 describe the dose-response information used to evaluate potential carcinogenic and noncarcinogenic effects of COCs at the Anchor Chemical site. Full toxicity profiles for all COCs are presented in Appendix C.

4.3.2 Carcinogenic Effects

4.3.2.1 General Method

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

TABLE 4-8. TOXICITY VALUES FOR CONTAMINANTS OF CONCERN AT THE ANCHOR CHEMICAL SITE

	CARCINOGENIC		CHRONIC	SUBCHRONIC
Chemical	**Weight of Evidence Classification**	**Oral Slope Factor (mg/kg/day)-1**	**Chronic Oral RfD (mg/kg/day)**	**Subchronic Oral RfD (mg/kg/day)**
Volatiles				
Carbon disulfide	--		1.00E-01 a	1.00E-01 b
1,1-Dichloroethane	C a		1.00E-01 b	1.00E+00 b
Ethylbenzene	D a		1.00E-01 a	1.00E+00 b
Toluene	D a		2.00E-01 a	2.00E+00 b
1,1,1-Trichloroethane	D a		9.00E-02 b	9.00E-01 b
Xylenes	D a		2.00E+00 a	4.00E+00 b
BNAs				
Benzoic acid	D a		4.00E+00 a	4.00E+00 b
Benzo(a)pyrene	B2 a	7.30E+00 a		
Benzo(b)fluoranthene	B2 a	7.30E-01 d		
Benzo(k)fluoranthene	B2 a	7.30E-01 d		
Benzylbutylphthalate	C a		2.00E-01 a	2.00E+00 b
Bis(2-ethylhexyl)phthalate	B2 a	1.40E-02 a	2.00E-02 a	2.00E-02 b
4-Chloroaniline	--		4.00E-03 a	4.00E-03 b
Chrysene	B2 a	7.30E-02 d		
Diethylphthalate	D a		8.00E-01 a	8.00E+00 b
Di-n-butyl phthalate	D a		1.00E-01 a	1.00E+00 b
Di-n-octyl phthalate	--		2.00E-02 b	2.00E-02 b
Fluoranthene	D a		4.00E-02 a	4.00E-01 b
Indeno(1,2,3-cd)pyrene	B2 a	7.30E-01 d		
2-Methylnaphthalene	--			
Naphthalene	D a			
Phenanthrene	D a			
Pyrene	D a		3.00E-02 a	3.00E-01 b
Pesticides				
alpha-BHC	B2 a	6.30E+00 a		
beta-BHC	C a	1.80E+00 a		
gamma-Chlordane (r)	B2 a	1.30E+00 a	6.00E-05 a	6.00E-05 b
4,4' DDE	B2 a	3.40E-01 a		
Dieldrin	B2 a	1.60E+01 a	5.00E-05 a	5.00E-05 b
Endrin	D a		3.00E-04 a	3.00E-04 b
Methoxyclor	D a		5.00E-03 a	5.00E-03 b
Inorganics				
Aluminum	D c		1.00E+00 c	1.00E+00 e
Antimony	--		4.00E-04 a	4.00E-04 b
Arsenic	A a	2.00E+00 f	3.00E-04 a	3.00E-04 b
Barium	--		7.00E-02 a	7.00E-02 b
Beryllium	B2 a	4.30E+00 a	5.00E-03 a	5.00E-03 b
Cadmium	B1 a		5.00E-04 a,g	5.00E-04 e
Chromium, total	--		8.76E-01 h	8.77E-01 h
Cobalt	--			
Copper	D a		5.50E-02 c	5.50E-02 e
Iron	D c		5.00E-01 c	5.00E-01 e
Lead	B2 a			
Manganese	D a		5.00E-03 a,i	1.00E-01 b
Mercury	D a		3.00E-04 b	3.00E-04 b
Nickel	A a		2.00E-02 a,j	2.00E-02 b
Thallium	--		7.00E-05 b,k	7.00E-04 b,k
Vanadium	--		7.00E-03 b	7.00E-03 b
Zinc	D a		3.00E-01 b	3.00E-01 b

a. From IRIS.
b. From HEAST.
c. Interim value from ECAO.
d. Oral slope factor for B(a)P used for PAHs classified as B2 carcinogens with the TEFs applied.
e. Chronic RfD used as Subchronic RfD if no Subchronic value is available per RAGS.
f. Arsenic oral slope factor derived from unit risk in IRIS.
g. Cadmium RfD is for water; 1.0E-03 mg/kg/day is RfD for food.
h. Value is weighted-average value of the Hex and Tri RfDs assuming 7 parts Tri to 1 part Hex.
i. Manganese RfD is for water; RfD for food is 1.4E-1 mg/kg/day.
j. Value is for nickel, soluble salts.
k. Value is for Thallic oxide.

A summary of the available data for carcinogenic effects for each COC is presented in Table 4-9. This table includes the COC, the type of cancer and species from which the oral slope factor was derived, the source of the slope factor, and the primary studies used in the derivation of the oral slope factors. Table 4-10 presents an overview of the EPA weight-of-evidence categories for human carcinogenicity. Table 4-11 presents the criteria used to assess human and animal data for each EPA weight-of-evidence category in Table 4-10.

4.3.2.2 Chemical-Specific Considerations

PAHs

A number of PAHs, several of which have been demonstrated to be carcinogenic, were detected in the media sampled at the Anchor Chemical site. All PAHs detected at the site which are class B2 carcinogens are listed in Table 4-12. The slope factor for benzo(a)pyrene (B(a)P) was applied to this group of compounds. In addition, toxic equivalency factors (TEFs), provided by EPA (EPA, 1992i), were applied to the B(a)P slope factor as follows:

Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.1
Chrysene	0.01
Indeno(1,2,3-cd)pyrene	0.1

The B(a)P slope factor of 7.30 $(mg/kg\text{-}day)^{-1}$ currently available on IRIS was used.

TABLE 4-9. POTENTIAL CARCINOGENIC EFFECTS OF THE COCS AT THE ANCHOR CHEMICAL SITE

Chemical	Type of Cancer		Species	Source of Data
Volatiles				
Carbon Disulfide	Oral:	NA		
	Inhal:	NA		
1,1-Dichloroethane	Oral:	NA		
	Inhal:	NA		
Ethylbenzene	Oral:	NA		IRIS (7/1/93)
	Inhal:	NA		
Toluene	Oral:	NA		
	Inhal:	NA		
1,1,1-Trichloroethane	Oral:	NA		
	Inhal:	NA		
Xylenes	Oral:	NA		
	Inhal:	NA		
Base Neutral/Acid Extractables (Semivolatiles)				
Benzo(a)pyrene	Oral:	stomach tumors	rodent	IRIS (7/1/93)
	Inhal:	respiratory tract tumors	hamster	IRIS (7/1/93)
Benzo(b)fluoranthene	Oral:	epidermoid carcinomas/pleomorphic sarcomas	rat	IRIS (7/1/93)
	Inhal:	lack of data in animals and humans		IRIS (7/1/93)
Benzo(k)fluoranthene	Oral:	adenomas/hepatomas	mice	IRIS (7/1/93)
	Inhal:	NA		
Benzoic Acid	Oral:	NA		
	Inhal:	NA		
Benzylbutylphthalate	Oral:	mononuclear cell leukemia	rat	IRIS (7/1/93)
	Inhal:	NA		

TABLE 4-9. (CONTINUED)

Chemical	Type of Cancer	Species	Source of Data
Bis(2-Ethylhexyl)phthalate	Oral: liver tumors Inhal: NA	rat	IRIS (7/1/93)
4-Chloroaniline	Oral: NA Inhal: NA		
Chrysene	Oral: carcinoma/malignant lymphomas Inhal: NA	mice	IRIS (7/1/93)
Diethylphthalate	Oral: NA Inhal: NA		
Di-n-butyl phthalate	Oral: NA Inhal: NA		
Di-n-octyl phthalate	Oral: NA Inhal: NA		
Fluoranthene	Oral: NA Inhal: NA		
Indeno(1,2,3-cd)pyrene	Oral: lung epidermoid carcinomas Inhal: NA	rat	IRIS (7/1/93)
2-Methylnaphthalene	Oral: NA Inhal: NA		
Naphthalene	Oral: NA Inhal: NA		
Phenanthrene	Oral: NA Inhal: NA		
Pyrene	Oral: NA Inhal: NA		

TABLE 4-9. (CONTINUED)

Chemical	Type of Cancer		Species	Source of Data
Pesticides/PCBs				
alpha-BHC	Oral:	liver tumors	mouse	IRIS (7/1/93)
	Inhal:	liver tumors	mouse	IRIS (7/1/93)
beta-BHC	Oral:	liver tumors	mouse	IRIS (7/1/93)
	Inhal:	liver tumors	mouse	IRIS (7/1/93)
gamma-Chlordane	Oral:	hepatocellular carcinomas	mouse	IRIS (7/1/93)
	Inhal:	hepatocellular carcinomas	mouse	IRIS (7/1/93)
4,4'-DDE	Oral:	liver/thyroid tumors	rodent	IRIS (7/1/93)
	Inhal:	NA		
Dieldrin	Oral:	hepatocarcinomas/pulmonary metastases	mouse	IRIS (7/1/93)
	Inhal:	hepatocarcinomas/pulmonary metastases	mouse	IRIS (7/1/93)
Endrin	Oral:	NA		
	Inhal:	NA		
Methoxychlor	Oral:	NA		
	Inhal:	NA		
Inorganics				
Aluminum	Oral:	NA		
	Inhal:	NA		
Antimony	Oral:	NA		
	Inhal:	NA		
Arsenic	Oral:	skin cancer	human	IRIS (2/1/93)
	Inhal:	lung cancer	human	IRIS (2/1/93)
Barium	Oral:	NA		
	Inhal:	NA		
Beryllium	Oral:	gross tumors	rat	IRIS (7/1/93)
	Inhal:	lung tumors	human	IRIS (7/1/93)

TABLE 4-9. (CONTINUED)

Chemical	Type of Cancer	Species	Source of Data
Cadmium	Oral: NA Inhal: lung tumors	rat	IRIS (7/1/93)
Chromium	Oral: NA Inhal: lung cancer	human	IRIS (7/1/93)
Cobalt	Oral: NA Inhal: NA		
Copper	Oral: NA Inhal: NA		
Iron	Oral: NA Inhal: NA		
Lead	Oral: bilateral renal carcinoma Inhal: NA	rat	IRIS (7/1/93
Manganese	Oral: NA Inhal: NA		
Mercury	Oral: NA Inhal: NA		
Nickel	Oral: NA Inhal: lung cancer	human	IRIS (7/1/93)
Thallium	Oral: NA Inhal: NA		
Vanadium	Oral: NA Inhal: NA		
Zinc	Oral: NA Inhal: NA		

NA Information Not Available on IRIS or HEAST. If IRIS referenced, information not available as stated in IRIS.
IRIS: Integrated Risk Information System. February 1, 1993.
HEAST: Health Effects Assessment Summary Tables, FY 1992 (including supplements).

TABLE 4-10. THE EPA WEIGHT-OF-EVIDENCE FOR HUMAN CARCINOGENICITY (EPA, 1986b)

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

TABLE 4-11. EPA CARCINOGENICITY WEIGHT-OF-EVIDENCE CRITERIA FOR HUMAN AND ANIMAL DATA (EPA, 1986b)

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

TABLE 4-11. EPA CARCINOGENICITY WEIGHT-OF-EVIDENCE CRITERIA FOR HUMAN AND ANIMAL DATA (EPA, 1986b) (CONTINUED)

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

TABLE 4-12. CARCINOGENICITY OF PAHs DETECTED AT THE ANCHOR CHEMICAL SITE

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

Benzo(a)pyrene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Chrysene
Indeno(1,2,3-cd)pyrene

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

Fluoranthene
2-Methylnaphthalene
Naphthalene
Phenanthrene
Pyrene

Source: EPA, 1993a.

4.3.3 Noncarcinogenic Effects

4.3.3.1 General Method

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

Chronic RfDs are derived from the following equation:

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

Where:

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

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

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

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

To evaluate short-term exposures, subchronic RfDs were used. A subchronic RfD is similar to a chronic RfD except that the duration of exposure is defined by EPA as being "substantially less-than-lifetime," from two weeks to seven years (EPA, 1989a). Subchronic RfDs are derived in the same manner as chronic values unless the uncertainty factor applied to derive a chronic RfD has been applied to adjust for the extrapolation from subchronic exposure to chronic exposure. In these cases, the uncertainty factor is not applied in deriving the subchronic RfD.

A summary of the available data for chronic noncarcinogenic effects is presented in Table 4-13. Included in the table is the COC, the confidence level of the study used to determine the chronic oral RfD, a description of the critical physiological effect and the species of animal used in the study, the uncertainty/modifying factor, the source of the study used to derive the chronic oral RfD, and the study itself. The confidence levels were provided by IRIS as either high, medium, or low.

Table 4-14 contains a summary of the available data for subchronic noncarcinogenic effects. Included in this table is the COC, a description of the critical physiological effect and the species of animal used in the study, the uncertainty factor, the source of the study used to derive the subchronic oral RfD and the study itself.

In cases where a subchronic oral RfD has not been developed by EPA, the chronic oral RfD was adopted as the subchronic oral RfD per EPA guidance (EPA, 1989a). If neither the chronic oral RfD nor the subchronic oral RfD was available for a given COC, long-term and short-term oral exposure pathways were not assessed quantitatively.

TABLE 4-13. POTENTIAL CHRONIC NONCARCINOGENIC EFFECTS OF THE COCS AT THE ANCHOR CHEMICAL SITE

Chemical	EPA Level of Confidence	Critical Effect		Species	Uncert. & Mod. Factors	Source of Data
Volatiles						
Carbon Disulfide	medium	Oral:	fetal toxicity	rabbit	UF: 100 MF: 1	IRIS (7/1/93)
	NA	Inhal:	fetal toxicity	rabbit	UF: 1000	HEAST (1992)
1,1-Dichloroethane	NA	Oral:	none observed	rat	UF: 1000	HEAST (1992)
	NA	Inhal:	NA			
Ethylbenzene	low	Oral:	liver and kidney toxicity	rat	UF: 1000 MF: 1	IRIS (7/1/93)
	low	Inhal:	developmental toxicity	rat	UF: 300 MF: 1	IRIS (7/1/83)
Toluene	NA	Oral:	changes in liver and kidney weight	rat	UF: 1000 MF: 1	IRIS (7/1/93)
	NA	Inhal:	CNS effects, eye and nose irritation	human	UF: 100	HEAST (1992)
1,1,1-Trichloroethane	NA	Oral:	hepatotoxicity	guinea pig	UF: 1000	HEAST (1992)
	NA	Inhal:	hepatotoxicity	guinea pig	UF: 1000	HEAST (1992)
Xylene	medium	Oral:	decreased body weight and mortality	rat	UF: 100 MF: 1	IRIS (7/1/93)
	NA	Inhal:	NA			
Base Neutral/Acid Extractables (Semivolatiles)						
Benzo(a)pyrene	NA	Oral:	NA			
	NA	Inhal:	NA			
Benzo(b)fluoranthene	NA	Oral:	NA			
	NA	Inhal:	NA			
Benzo(k)fluoranthene	NA	Oral:	NA			
	NA	Inhal:	NA			

TABLE 4-13. (CONTINUED)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
Benzoic Acid	medium NA	Oral: no adverse effects Inhal: NA	human	UF: 1 MF: 1	IRIS (7/1/93)
Benzylbutylphthalate	low NA	Oral: increased liver to body and liver to brain weight ratios Inhal: NA	mouse	UF: 1000 MF: 1	IRIS (7/1/93)
Bis(2-ethylhexyl)phthalate	medium NA	Oral: increased relative liver weight ratios Inhal: NA	mouse	UF: 1000 MF: 1	IRIS (7/1/93)
4-Chloroaniline	low NA	Oral: lesions of splenic capsule Inhal: NA	rat	UF: 3000 MF: 1	IRIS (7/1/93)
Chrysene	NA NA	Oral: NA Inhal: NA			
Di-n-butylphthalate	low NA	Oral: increased mortality Inhal: NA	rat	UF:1000 MF:1	IRIS (7/1/93)
Diethylphthalate	low NA	Oral: decreased growth rate, food consumption and altered organ weights Inhal: NA	rat	UF: 1000 MF: 1	HEAST (1992)
Di-n-octyl phthalate	NA NA	Oral: elevated kidney and liver weights; increased SGOT and SEPT Inhal: NA	rat	UF: 1000	HEAST (1992)
Fluoranthene	low NA	Oral: nephropathy, increased liver weights, hematological alternations Inhal: NA	mouse	UF: 300 MF: 1	IRIS (7/1/93)

TABLE 4-13. (CONTINUED)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
Indeno(1,2,3-cd)pyrene	NA NA	Oral: NA Inhal: NA			
2-Methylnapthalene	NA NA	Oral: NA Inhal: NA			
Naphthalene	NA NA	Oral: decreased body weight gain Inhal: NA	rat	UF: 1000	HEAST (1992)
Phenanthrene	NA NA	Oral: NA Inhal: NA			
Pyrene	low NA	Oral: kidney effects Inhal: NA	mouse	UF: 3000 MF: 1	IRIS (7/1/93)
Pesticides/PCBs					
alpha-BHC	NA NA	Oral: NA Inhal: NA			
beta-BHC	NA NA	Oral: NA Inhal: NA			
gamma-Chlordane	low NA	Oral: liver hypertrophy Inhal: NA	rat	UF: 1000 MF: 1	IRIS (7/1/93)
4,4′-DDE	NA NA	Oral: NA Inhal: NA			
Dieldrin	medium NA	Oral: liver lesions Inhal: NA	rat	UF: 300 MF: 1	IRIS (7/1/93)
Endrin	medium NA	Oral: liver lesions Inhal: NA	dog	UF: 100 MF: 1	IRIS (7/1/93)

TABLE 4-13. (CONTINUED)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
Methoxychlor	low	Oral: excessive litter loss	rabbit	UF: 1000 MF: 1	IRIS (7/1/93)
	NA	Inhal: NA			
Inorganics					
Aluminum	medium	Oral: decreased body weight gain and neurotoxicity	mouse	UF: 100	EPA 1992e
	NA	Inhal: NA			
Antimony	low	Oral: decreased longevity, blood glucose and cholesterol	rat	UF: 1000 MF: 1	IRIS (7/1/93)
	NA	Inhal: NA			
Arsenic	medium	Oral: hyperpigmentation, keratosis, and possible vascular complications	human	UF: 3 MF: 1	IRIS (7/1/93)
	NA	Inhal: NA			
Barium	medium	Oral: increased blood pressure	human	UF: 3 MF: 1	IRIS (7/1/93)
	NA	Inhal: NA			
Beryllium	low	Oral: no adverse effects	rat	UF: 100, MF: 1	IRIS (7/1/93)
	NA	Inhal: NA			
Cadmium	high	Oral: significant proteinuria	human	UF: 10 MF: 1	IRIS (7/1/93)
	NA	Inhal: NA			
Chromium	low	Oral: Chromium III: no adverse effects observed	rat	UF: 100 MF: 1	IRIS (7/1/93)
	low	Chromium VI: no effects reported	rat	UF: 500 MF: 1	IRIS (7/1/93)
	NA	Inhal: Chromium III: NA			
	NA	Chromium VI: nasal mucosa atrophy	human	UF: 300	HEAST (1992)

TABLE 4-13. (CONTINUED)

Chemical	EPA Level of Confidence	Critical Effect	Species	Uncert. & Mod. Factors	Source of Data
Cobalt	NA NA	Oral: NA Inhal: NA			
Copper	NA NA	Oral: local GI irritation Inhal: NA	human		EPA, 1992e
Iron	NA NA	Oral: NA Inhal: NA			
Lead	NA NA	Oral: NA Inhal: NA			
Manganese	medium medium	Oral: CNS effects Inhal: respiratory symptoms and psychomotor disturbances	human human	UF: 1 MF: 1 UF: 300 MF: 1	IRIS (7/1/93) IRIS (7/1/93)
Mercury	NA NA	Oral: kidney effects Inhal: nervous system effects	rat human	UF: 1000 UF: 30	HEAST (1992)
Nickel	medium NA	Oral: decreased body and organ weights Inhal: NA	rat	UF: 100 MF: 3	IRIS (7/1/93)
Thallium	NA NA	Oral: NA Inhal: NA			
Vanadium	NA NA	Oral: none observed Inhal: NA	rat	UF: 100	HEAST (1992)
Zinc	NA NA	Oral: anemia Inhal: NA	human	UF: 10	IRIS (7/1/93)

NA Information Not Available on IRIS or HEAST. If IRIS referenced, information not available as stated in IRIS.
IRIS: Integrated Risk Information System. July 1, 1993.
HEAST: Health Effects Assessment Summary Tables, FY 1992 (and supplements).

TABLE 4-14. POTENTIAL SUBCHRONIC NONCARCINOGENIC EFFECTS OF THE ANCHOR CHEMICAL SITE COCS

Chemical	Critical Effect	Species	Uncert. Factor
Volatiles			
Carbon Disulfide	fetal toxicity	rabbit	100
1,1-Dichloroethane	none observed	rat	100
Ethylbenzene	hepatoxicity and nephrotoxicity	rat	100
Toluene	changes in liver and kidney weights	rat	100
1,1,1-Trichloroethane	hepatotoxicity	guinea pig	100
Xylenes (total)	hyperactivity, decreased body weight and increased mortality	rat	100
Base Neutral/Acid Extractables (Semivolatiles)			
Benzo(a)pyrene	NA		
Benzo(b)fluoranthene	NA		
Benzo(k)fluoranthene	NA		
Benzoic Acid	none observed	human	1
Benzylbutylphthalate	effects on body weight gain, testes, liver, kidney	rat	100
Bis(2-ethylhexyl)phthalate	increased relative liver weight	guinea pig	1000
4-Chloroaniline	spleen lesions	rat	3000
Chrysene	NA		
Diethylphthalate	decreased body and organ weight	rat	100
Di-n-butylphthalate	NA		

TABLE 4-14. (CONTINUED)

Chemical	Critical Effect	Species	Uncert. Factor
Di-n-octylphthalate	elevated kidney and liver weights; increased SGOT and SGPT	rat	1000
Fluoranthene	nephropathy; liver weight changes; hematological changes	mouse	300
Indeno(1,2,3-cd)pyrene	NA		
2-Methylnaphthalene	NA		
Naphthalene	decreased body weight gain	rat	1000
Phenanthrene	NA		
Pyrene	renal effects	mouse	300
Pesticides/PCBs			
alpha-BHC	NA		
beta-BHC	NA		
gamma-Chlordane	liver necrosis	rat	1000
4,4'-DDE	NA		
Dieldrin	liver lesions	rat	100
Endrin	liver lesions	dog	100
Methoxychlor	loss of litters	rabbit	1000
Inorganics			
Aluminum	NA		
Antimony	increased mortality, altered blood chemistry	rat	1000

TABLE 4-14. (CONTINUED)

Chemical	Critical Effect	Species	Uncert. Factor
Arsenic	skin keratosis hyperpigmentation	human	3
Barium	increased blood pressure	human	
Beryllium	none observed	rat	100
Cadmium	NA		
Chromium, total	hepatotoxicity	rat	100
Cobalt	NA		
Copper	local GI irritation	human	NA
Iron	NA		
Lead	NA		
Manganese	CNS effects, respiratory effects	human	1-900
Mercury	neurotoxicity, kidney effects	human	30-1000
Nickel	decreased body and organ weight	rat	300
Thallium	NA		
Vanadium	none observed	rat	100
Zinc	anemia	human	10

Note: All subchronic information is from the *Health Effects Assessment Summary Tables*, FY 1992 (and supplements).
NA: Information Not Available

4.3.3.2 Chemical-Specific Considerations

PAHs

Several noncarcinogenic PAHs were detected at the site. RfD values do not exist for the noncarcinogenic PAH COCs benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, indeno(1,2,3-cd)pyrene, 2-methylnaphthalene, naphthalene and phenanthrene. The lack of toxicological data precludes the derivation of a systemic toxicity value. Per EPA, a surrogate RfD was not applied due to the uncertainties associated with assuming noncarcinogenic PAHs cause similar effects (EPA, 1991d).

Cadmium

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

Manganese

Two chronic oral RfDs are available to evaluate manganese exposures: 5×10^{-3} mg/kg/day for water consumption and 1.4×10^{-1} mg/kg/day for food consumption. In this risk assessment, the food consumption RfD was used for soil and sediment exposures and the water consumption RfD for water exposures.

4.4 Risk Characterization

4.4.1 Introduction

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

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

4.4.2 General Methodology

Risk is a function of chemical toxicity and the route and duration of exposure. EPA's cancer slope factors, RfDs, and health advisories, discussed in Section 4.3, were used as indicators of toxicity in the risk characterization. The chemical- and pathway-specific doses calculated in accordance with the methods outlined in Section 4.2 are used to represent exposure.

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

4.4.2.1 Carcinogenic Risk

The incremental carcinogenic (CA) risk associated with exposure to contaminants detected at the Anchor Chemical site was calculated according to the following equation:

$$\text{Incremental CA Risk} = \text{Slope Factor} \times \text{Dose}$$

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

The slope factor represents the carcinogenic potency of a chemical (Table 4-8). The dose, or intake, represents the amount of contaminant to which a receptor is exposed, as described in Section 4.2. When evaluating carcinogenic risks, the dose is the estimated daily intake of each contaminant during the specified period of exposure, and averaged over a 70-year lifetime.

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

EPA has not identified a single value that represents a significant incremental cancer risk. However, the NCP acceptable risk range for Superfund sites has been set at approximately 10^{-6} to 10^{-4} per environmental medium (NCP, 1990). In other words, the goal of the NCP is to reduce the cancer risk associated with site contaminants in a given medium to within or below a range of 1 in 10,000 to 1 in 1,000,000 (EPA, 1992j). Risks approximately equal to 10^{-4} (e.g., up to approximately 4×10^{-4}) may be considered acceptable under the NCP.

4.4.2.2 Noncarcinogenic Effects

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

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

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

HQs were calculated for each contaminant for which health criteria are currently available (Table 4-8). The HQs for each contaminant were summed to produce a rough estimate of the exposure pathway-specific risk, the Hazard Index (HI). Where appropriate, Hazard Indices for all soil media exposure pathways were summed to provide a total medium-specific risk. In estimating total noncarcinogenic risk, potential responses were conservatively assumed to be additive. However, all COCs do not have the same or similar toxic endpoints and responses may not be additive.

4.4.3 Risk Summary

An overall summary of Anchor Chemical site carcinogenic and noncarcinogenic risks is presented in Tables 4-15 and 4-16, respectively. These tables include cumulative cancer risk values and HIs for each exposure pathway and receptor population, and for present and future land use scenarios. Medium-specific cancer risk, for which the NCP acceptable risk range of 10^{-6} to 10^{-4} applies, are derived by adding all pathways for a given medium. Chemical-specific risk values are presented in Appendix B.

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

The greatest carcinogenic risk value at the Anchor Chemical site is associated with the ingestion of ground water under the future residential scenario. This scenario produced a total risk value of 10^{-5}, which does not exceed the upper limit of the NCP's acceptable risk range. Currently, ground water at the site is not used for consumption, so present scenarios were not assessed. Exposures to subsurface soils and sediments produced a total risk value of 3×10^{-7}. These values fall within or below the acceptable risk range established by the NCP.

The ingestion of ground water by future on-site and/or adjacent residents produced a noncarcinogenic HI which is slightly above unity. All other exposure pathways produced noncarcinogenic HIs at or below 4×10^{-1}.

It should be noted that information regarding the toxicity of 2-methylnaphthalene, naphthalene, phenanthrene, and cobalt is insufficient for the calculation of carcinogenic and noncarcinogenic toxicity values. As a result, the toxic effects of

TABLE 4-15. SUMMARY OF CARCINOGENIC RISK ESTIMATED FOR THE ANCHOR CHEMICAL SITE

Scenario	Receptor	Present/Future	Total Risk[a]
Ground Water			
Ingestion	Onsite and/or Adjacent Resident	F	8 x 10^{-5}*
Subsurface Soil and Sediments			
Ingestion	Excavation Worker	F	3 x 10^{-7}

*Exceeds 10^{-6} risk

[a] Chemical-specific risk assessment summary information is presented in the risk spreadsheets in Appendix B.

TABLE 4-16. SUMMARY OF NONCARCINOGENIC HAZARD INDICES (HI) ESTIMATED FOR THE ANCHOR CHEMICAL SITE

Scenario	**Receptor**	**Present/Future**	**Chronic HI**[a]
Ground Water			
Ingestion	Onsite and/or Adjacent Resident	F	$3 \times 10^{+0}$*
Inhalation	Onsite and/or Adjacent Resident	F	2×10^{-4}
			$3 \times 10^{+0}$*
Subsurface Soil and Sediments			
Ingestion	Excavation Worker	F	4×10^{-1}[b]
Dermal Contact	Excavation Worker	F	4×10^{-4}[b]
			4×10^{-1}

[a] Chemical-specific risk assessment summary information is presented in the risk spreadsheets in Appendix B.
[b] HI is based on Subchronic Protective Body Dose.

*Exceeds unity.

their presence could not be quantitatively evaluated. In addition, a toxicity value for lead is not currently available. Lead was evaluated in relation to its MCL and OSWER soil cleanup level.

4.4.3.1 Ground Water

Carcinogenic Risks

The carcinogenic risk value associated with a future resident ingesting ground water was estimated to be 8 x 10^{-5}. This value is attributable to the presence of arsenic and bis(2-ethylhexyl)phthalate. Arsenic was detected in three of the 22 ground water samples analyzed at concentrations that were less than the contract required detection limit (CRDL) of 10 µg/L. Bis(2-ethylhexyl)phthalate (BEHP) was detected in 13 of the 22 ground water samples analyzed. The highest concentration (160 µg/L) was detected during the second round of sampling in monitoring well MW-7S. BEHP also was detected at a concentration of 100 µg/L in the reanalysis of this sample, but was only detected at 17 µg/L in this well during the first sampling round. A review of the data validation documents provided in the RI revealed that the elevated concentrations of BEHP are most likely not the result of laboratory contamination.

The total carcinogenic risk for the ground water contaminant inhalation scenario could not be calculated due to a lack of appropriate toxicity values. Risk from this exposure route, however, is expected to be less significant than exposure through ingestion because few volatile organic compounds were detected.

Noncarcinogenic Risks

The chronic HI for the ingestion of ground water by future residents is slightly above unity (HI=3). This HI resulted mostly from the combined presence of four metals:

aluminum (HQ=0.8), arsenic (HQ=0.3), iron (HQ=0.8) and manganese (HQ=0.8). No chemical-specific hazard quotient exceeded unity.

The future resident ground water shower inhalation scenario resulted in an HI which was well below unity (2 x 10^{-4}). This risk value was due only to the presence of 1,1,1-trichloroethane (TCA), the only volatile COC. All of the other ground water COCs detected in site ground water were semivolatile organics which are not expected to volatilize nearly as much as TCA during showering and were therefore not quantitatively evaluated. However, as unity was not reached in the noncancer HI for inhaled volatiles while showering, the HI for semivolatiles would also be expected to be below unity.

Other Potential Risks

Lead was detected in 18 of the 19 ground water samples analyzed at concentrations exceeding the MCL/action level of 0.015 mg/L for drinking water sources. Site concentrations ranged from 0.0102 to 0.240 mg/L and were detected during both RI sampling events. Therefore, lead concentrations may pose added risk to potential receptors.

Cobalt, which was detected in three out of 22 samples analyzed, could not be assessed due to a lack of toxicity data. However, the calculated dose estimates for cobalt (see Appendix C) do not exceed 0.06 mg/kg/day, a value reported as typical of dietary intake (EPA, 1992c). Detected cobalt concentrations, therefore, are not expected to pose added risk to potential receptors.

Exposure to ground water contaminants via dermal contact was not quantitatively evaluated and may contribute additional risk to future receptors. However, this risk is not expected to be more than the total risk attributable to exposure via inhalation and ingestion.

4.4.3.2 Combined Subsurface Soils and Sediments

Carcinogenic Risks

A total carcinogenic risk estimate of 3 x 10^{-7} was estimated for the ingestion of subsurface soils and sediments by an excavation worker. This value is below the NCP's acceptable risk range, so future exposures via this pathway are expected to be negligible.

Noncarcinogenic Risks

The HI for excavation workers ingesting subsurface soils and sediments under the future scenario is estimated to be 4 x 10^{-1}. This is below unity, so exposure is not likely to present significant noncarcinogenic risks.

Under the dermal contact scenario for future excavation workers at the Anchor Chemical site, the HI was also less than unity (4 x 10^{-4}). This value was based only on detected concentrations of cadmium.

Other Potential Risks

The maximum concentration of lead detected in the subsurface soil and sediment grouping was 1,620 mg/kg, which is above EPA's target cleanup level (500 to 1,000 mg/kg; EPA, 1989d). Four of the nine drywell sediment samples had lead concentrations of above 500 mg/kg, while none of the subsurface soil samples contained significant levels of lead.

Exposure via inhalation of volatile or particulate emissions from subsurface soils and sediments was not quantitatively evaluated but may pose additional risks. However, this exposure scenario is expected to be minimal due to the short duration of exposure and also is not expected to be more significant than exposure via ingestion.

4.5 Discussion of Uncertainties

4.5.1 Introduction

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

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

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

4.5.2 General Methodological Uncertainties

4.5.2.1 Site Characterization

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

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

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

4.5.2.2 Toxicological Information

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

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

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

EPA and other regulatory agencies attempt to account for these sources of uncertainty by including uncertainty factors in the determination of health criteria such as RfDs.

In addition, the level of confidence in RfDs for noncarcinogenic effects and the weight of evidence for carcinogenic effects are specified for each contaminant. These qualifiers have been discussed in the dose-response section of this study.

4.5.2.3 Exposure Assumptions

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

skin surface area for dermal contact; and absorption rates via the skin and digestive tract.

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

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

4.5.2.4 Dermal Contact Pathway

EPA (EPA, 1992a) has recommended that dermal contact risks should only be evaluated quantitatively for three types of contaminants in soil media: cadmium, PCBs, and dioxins. This guidance is based on the high level of uncertainty in data needed to evaluate this pathway (e.g., chemical-specific dermal absorption factors). This approach leads to an underestimation of total risk for specific soil media.

4.5.2.5 Risk Characterization

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

4.5.3 Site-Specific Uncertainties

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

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

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

The ability of this risk assessment to estimate risks associated with the Anchor Chemical site is very much dependent on the representativeness of the analytical data presented in the RI. If the extent of contamination has not been adequately characterized in the RI and contaminated areas have not been sampled, then it is probable that uncertainty has been increased. Under this situation, actual risk estimates may be higher than those presented in this risk assessment.

There is inherent variability in environmental sampling results given the spatial distribution of contamination and compositions of the matrix sampled. In addition, due to the limited number of samples collected and absence of off-site samples, the extent of contamination at the Anchor Chemical site may not be completely delineated. Only ten sediment and six subsurface soil samples were collected from the 2-15 feet depth interval evaluated in this risk assessment. The ground water was characterized with only seven well locations, four of which were paired shallow and deep wells. A total of over 23 ground water samples were evaluated in the risk assessment.

The Anchor RI report notes a gradual decrease in ground water VOC contamination in three wells sampled from 1982 to 1991. This decrease may be due to the degradation or migration of the organic compounds. If migration is occurring, then higher concentrations of contaminants may be present in off-site ground waters. However, this possibility can not be evaluated in this risk assessment since all of the RI monitoring wells and soil borings were placed within the boundaries of the 1.5 acre site. No wells were placed on adjacent properties down-gradient of the site to accurately assess the extent of contamination.

COCs were selected using a conservative methodology. Only non-toxic inorganics, or chemicals detected at a frequency of less than five percent in a media grouping, were eliminated from further consideration as COCs. Due to this methodology, it is unlikely that significant contributors to risk were excluded.

Certain COCs lacked health criteria and therefore had to be evaluated qualitatively. For example, the evidence for lead carcinogenicity is under review and no slope factor is available. Lead was evaluated in relation to the target soil clean-up levels recommended by OSWER, and in relation to the MCL/action level for drinking water. Cobalt, 2-methylnaphthalene, naphthalene, and phenanthrene also were selected as COCs, but could not be evaluated due to a lack of appropriate health criteria.

Future land use and future use of public drinking water supplies are difficult to define. For this risk assessment, local zoning officials and site documents including the RI report (Anson, 1993) were consulted for information on these issues. Risk scenarios are based on land use and water supply estimates that would result in "reasonable maximum" exposures. The ground water ingestion scenario may overestimate risk because it assumes that 1) private wells will be installed on or adjacent to the site and will draw water from contaminated areas, or that 2) contaminant concentrations detected in the on-site monitoring wells will reach private residential wells.

Many other scenarios are possible at the Anchor Chemical site under present or future conditions. However, the risk estimates generated using the conservative exposure scenarios quantitatively evaluated in this risk assessment are expected to be protective of other potential receptors not evaluated. In addition, exposure routes evaluated are expected to be more significant than those not evaluated. For example, future excavation worker exposure to subsurface soils is expected to be more significant via ingestion than through inhalation at the Anchor Chemical site. The ingestion rate of 480 mg/day used to quantify exposure via ingestion is extremely conservative, and risk estimates based on this exposure route are expected to be more significant than those for other exposure routes such as inhalation or dermal contact.

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

Finally, one of the purposes of this risk assessment is to characterize the incremental risk associated with the Anchor Chemical site (i.e., risk over and above that attributable to anthropogenic or natural chemicals in the vicinity of the site). This determination relies partially on collection of background samples to estimate concentrations of chemicals (especially inorganics) in areas not influenced by the site. If site background conditions are not adequately characterized, then risk estimates for the site may not accurately reflect risk over and above that attributable to naturally occurring inorganics.

4.5.4 Analysis of Alternative Exposure Parameters

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

An estimate of central tendency risk can be obtained by substituting average or median (50th percentile) values for upper bound values. This is most useful for the only exposure pathway producing carcinogenic or noncarcinogenic risk estimates which are near the upper limits of NCP/EPA acceptance (i.e., ground water ingestion). Table 4-17 lists the exposure parameters used to calculate the RME ground water ingestion risks (from Section 4.2) as well as central tendency exposure parameters obtained from EPA guidance. Note that for some parameters, the RME risk calculation already uses an average value.

Table 4-18 indicates that values for three parameters decrease when estimating central tendency risk for ground water ingestion:

- exposure duration;

TABLE 4-17. COMPARISON OF GROUND WATER EXPOSURE PARAMETERS FOR RME VERSUS CENTRAL TENDENCY RISK

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

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

TABLE 4-18. COMPARISON OF RME RISK AND CENTRAL TENDENCY RISK FOR GROUND WATER INGESTION		
	Ground Water	
	RME Risk[a]	**Central Tendency Risk[b]**
Carcinogenic Risk	8×10^{-5}	2×10^{-5}
Driver Chemical:		
Bis(2-ethylhexyl)phthalate	8×10^{-6}	2×10^{-6}
Arsenic	7×10^{-5}	1×10^{-5}
Noncarcinogenic HI	$3 \times 10^{+0}$	$2 \times 10^{+0}$
Driver Chemical:		
Aluminum	8×10^{-1}	6×10^{-1}
Arsenic	3×10^{-1}	2×10^{-1}
Iron	8×10^{-1}	6×10^{-1}
Manganese	8×10^{-1}	6×10^{-1}

[a] From Appendix B.
[b] See text for derivation of central tendency risk estimates.

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

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

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

Central tendency risk values are compared with RME risk values in Table 4-18.

Central tendency risk for other exposure pathways were not determined since RME risk estimates were near or below the lower range of acceptable risk considered under the NCP and by EPA.

5.0 ECOLOGICAL RISK ASSESSMENT

5.1 Introduction

5.1.1 Background

This ecological risk assessment describes the important site characteristics that are present at and in the vicinity of the Anchor Chemical site. An evaluation of the risks associated with the exposure of potential ecological receptors to site contaminants is also provided. The objective of this ecological risk assessment is to evaluate whether contaminants from the Anchor Chemical site may pose adverse impacts to biota inhabiting the site or adjacent habitats.

5.1.2 General Methodology

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

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

- *Ecological Assessment of Hazardous Waste Sites: A Field and Laboratory Reference.* U.S. EPA Environmental Research Laboratory, Corvalis, OR, March 1989. (EPA, 1989e)

- *Ecological Assessment of Superfund Sites: An Overview.* Office of Emergency and Remedial Response, ECO Update, Vol. 1(4), December 1991. (EPA, 1991b)

This ecological risk assessment is typically composed of two parts:

- Problem Formulation, and
- Risk Characterization.

5.2 Problem Formulation

Problem Formulation is comprised of two primary components: Habitat and Species Characterization and Hazard Identification. Habitat and Species Characterization describes habitats present on and in the vicinity of the Anchor Chemical site and identifies potential receptor species. Hazard Identification identifies ecological exposure pathways and, if present, selects contaminants of ecological concern.

5.2.1 Habitat and Species Characterization

The characterization of habitats on the site and immediate vicinity is based on information provided in the Remedial Investigation Report (Anson, 1993). A site visit involving the collection of additional site-specific information by a TRC ecologist was not within the scope of this risk assessment.

The vicinity of the Anchor Chemical site is primarily industrial, but also includes a large recreational area (Cantiague Park) (Anson, 1993). The Anchor Chemical site itself consists of approximately 1.5 acres. Except for a narrow strip of lawn and plantings, the site is entirely covered by an existing building or asphalt (Anson, 1993). Although a receptor species survey was not conducted by Anson as part of the RI Report, there are no significant natural habitats present on the site that could potentially support indigenous wildlife receptor species. However, the Anchor Chemical site may provide habitat for various non-native species adapted for highly urbanized areas (e.g. rats, starlings, and pigeons).

Aquatic habitats or wetlands are not present within the vicinity of the site (Anson, 1993). Although areas of ecological significance are not known to be located in the vicinity of the site, potential habitats within the vicinity of the site include cemeteries, school grounds, and the adjacent Cantiague Park. The 125-acre

Cantiague Park includes a golf course and is likely to provide habitat for a variety of wildlife species. However, because of the extensive development and lack of suitable vegetated habitats at the Anchor Chemical site, potential receptor species which may inhabit the adjacent Cantiague Park (e.g., various songbirds, small mammals, herptiles) are not expected to frequent the site.

5.2.2 Hazard Identification

5.2.2.1 Exposure Pathways

Contaminants are present within the subsurface soils and subsurface sediments (dry wells) of the Anchor Chemical site and were also detected in the ground water underlying the site (Anson, 1993). Surface waters and surface soil contamination are not present at the site. For the characterization of ecological risk at the Anchor Chemical site, potential receptors are not expected to be exposed to contaminated subsurface soils or subsurface sediments. In addition, the risk of inhaling site contaminants, through either fugitive dust emissions or volatilization, is assumed to be negligible.

Contaminants present within the underlying ground water which may be discharged to nearby surface waters represent the primary ecological exposure pathway of concern at the Anchor Chemical site. Ground water is present beneath the site at a depth of approximately 60 feet and is reported to flow in a southwesterly direction (Anson, 1993). Due to the depth of the underlying ground water table and the apparent absence of surface waterbodies (which may receive ground water discharge) located southwest of the Anchor Chemical site (other than the Atlantic Ocean), it appears unlikely that ecological receptor species would be exposed to ground water contaminants originating from the site.

5.2.2.2 Contaminants of Concern

Due to the absence of complete exposure pathways that may affect ecological receptors, contaminants of concern (COCs) for the ecological risk assessment were not selected for the Anchor Chemical site. Wildlife receptor species are not expected to be significantly exposed to contaminants originating from the site.

5.3 Risk Characterization

Due to the absence of complete exposure pathways, it is unlikely that potential ecological receptor species would be exposed to contaminants originating from the Anchor Chemical site. Therefore, risk to ecological resources is not expected to be significant from contaminants present at the site.

7.0 REFERENCES

Andelman, J.B. 1985. "Inhalation Exposure in the Home to Volatile Organic Contaminants in Drinking Water. *The Science of the Total Environment.* 47:443-460.

Anson Environmental Ltd (Anson). 1993. Remedial Investigation Report, Anchor Chemical Site, Hicksville, New York. April 15, 1993.

EPA. 1979. *Water Related Environmental Fate of 129 Priority Pollutants, Volumes I and II.* Monitoring and Data Support Division. EPA 440/4-79-029a.

EPA. 1981. *Treatability Manual, Volume I - Treatability Data.* Office of Research and Development. EPA 600/2-82-001a.

EPA. 1984. Review of In-Place Treatment Technologies for Contaminated Surfaces. Volume 2-EPA-540/2-84-0036.

EPA. 1985. *Water Quality Assessment: Screening Procedures for Toxic and Conventional Pollutants in Surface and Ground Water-Part I (revised 1985).* Environmental Research Laboratory, Athens, Georgia. 600/6-85/002a.

EPA. 1986a. *Superfund Public Health Evaluation Manual.* Office of Emergency and Remedial Response. EPA 54/0/1-86/060.

EPA. 1986b. Guidelines for Carcinogenic Risk Assessment, *Federal Register,* Vol. 51, No. 185. September 24, 1986.

EPA. 1986c. Quality Criteria for Water. EPA 440/5-86-001.

EPA. 1988a. *Superfund Exposure Assessment Manual.* EPA 540/1-88/001, OSWER Directive 9285.5-1.

EPA. 1989a. *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A),* Interim Final. EPA 540/1-89/003.

EPA. 1989b. *Risk Assessment Guidance for Superfund, Volume II, Environmental Evaluation Manual,* Interim Final. EPA 540/1-89/001.

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

EPA. 1989d. Interim Guidance on Establishing Soil Lead Cleanup Levels at Superfund Sites. OSWER Directive, 9355-4-02, September 7, 1987.

EPA. 1989e. *Ecological Assessment of Hazardous Waste Site: A Field and Laboratory Reference*. EPA 600/3-89/013.

EPA. 1990b. *Basics of Pump and Treat Ground Water Remediation Technology*. EPA/600/8-90/003.

EPA. 1991a. Human Health Evaluation Manual Supplemental Guidance: "Standard Default Exposure Factors." OSWER Directive 9285.6-03. March 25, 1991.

EPA. 1991b. *Ecological Assessment of Superfund Sites: An Overview*. U.S. EPA Office of Emergency and Remedial Response, ECO Update Vol. 1(2), December 1991.

EPA. 1992a. *Dermal Exposure Assessment: Principles and Applications*. Interim Report. EPA/600/8-91/911B. Office of Research and Development. January 1992.

EPA. 1992b. *Health Effects Assessment Summary Tables*, FY-1992. OSWER (OS-230). OERR 9200. 6-303 (90-4). NTTS No. PB90-921104.

EPA. 1992c. Memorandum from Kenneth A. Poirer, Director, Superfund Health Risk Technical Support Center, to Dorothy Allen, U.S. EPA, Region II. Risk Assessment issue paper for cobalt (Tronic Plating Co. Site/Farmingdale, NY). March 2, 1992.

EPA. 1992e. Memorandum from Joan S. Dollarhide, Superfund Health Risk Technical Support Center, to Jeffrey Gratz, U.S. EPA Region II. Systemic toxicity and carcinogenicity information for multiple chemicals (Maywood Chemical Co./Maywood, New Jersey) September 3, 1992.

EPA. 1992f. Guidance for Data Useability in Risk Assessment. Office of Emergency and Remedial Response. EPA 540/G-90-008.

EPA. 1992i. Letter from Mike Walters, U.S. EPA Region II WAM, to TRC (formerly Alliance). May 4, 1992.

EPA. 1992j. *Supplemental Guidance to RAGS: Calculating the Concentration Term*. Office of Solid Waste and Emergency Response. May 1992.

EPA. 1992k. Guideline for Predictive Baseline Emissions Estimation Procedures for Superfund Sites.

EPA. 1993a. *Integrated Risk Information System* (IRIS). On-line Data Base. February 1, 1993.

EPA. 1993b. Letter from Joan S. Dollarhide, Superfund Health Risk Technical Support Center, to Mark Maddaloni, U.S. EPA Region I (Re: Oral and Inhalation Toxicity Information for Multiple Chemicals) January 29, 1993.

Foster, S.A. and P.C. Chrostowski. 1987. "Inhalation Exposures to Volatile Organic Contaminants in the Shower." Presented at the 80th Annual Meeting of the Air Pollution Control Association, New York. June 1987.

Gilbert, R.O. 1987. Statistical Methods for Environmental Pollution Monitoring. Van Nostrand Reinhold, New York, New York, 320 pp.

Hornbeck, R.W. 1975. Numerical Methods. Quantum Publishers, Inc., New York, New York, 310 pp.

Land, C.E. 1975. Tables of Confidence Limits for Linear Functions of the Normal Mean and Variance. In: Selected Tables in Mathematical Statistics, Vol. III. American Mathematical Society, Providence, Rhode Island, pp. 385-419.

LKB. 1985. Engineering Investigation of the Premises of Anchor/Lith Kem-Ko, Hicksville, Nassau County, New York.

MacKay, D. and W.Y. Shiu. 1981. "Critical Review of Henry's Law Constants for Chemicals of Environmental Interest." *Journal of Physical and Chemical Reference Data,* 19(4): 1175-1199.

Miller, N.M. and S.P. Wasik. 1985 "Relationship Between Octanol-Water Partition Coefficent and Aqueous Solubility." Environmental Science Technology. 19:552-529.

NCP. 1990. 40 CFR Part 300, National Oil and Hazardous Substances Pollution Contingency Plan; Final Rule. *Federal Register,* Vol 55, No. 46, p. 8666.

Roux. 1991. Project Operations Plan, Remedial Investigation, Roux Associates, Inc. April 1991.

SAS Institute Inc. 1988. SAS Language Guide for Personal Computers, Release 6.03 Edition. SAS Institute Inc., Cary, North Carolina.

Shacklette, H.T. and J.G. Boerngen. 1984. *Element Concentrations in Soils and Other Surficial Materials of Conterminous United States.* U.S. Geological Survey Professional Paper 1270 United States Government Printing Office, Washington.

TRC. 1993a. Personnel communication between Cynthia Fortin (TRC), and Dean Anson (Anson Environmental) on June 21, 1993.

TRC. 1993b. Personnel communication between Cynthia Fortin (TRC), and Dean Anson (Anson Environmental) on June 4, 1993.

Versheuren, K. 1983. Handbook of Environmental Data on Organic Chemicals. Van Nostrand-Reinhold Company, New York.

APPENDIX A

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE

302426

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE.

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE
UNITS: all water analyses are ug/L; organic soils are ug/Kg; inorganic soils are mg/Kg.

TYPE=Ground Water

Class	Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Arithmetic Mean Conc.	95 Pct. Upp. Conf. Limit	Lowest Observed Det.Limit	Highest Observed Det.Limit
VOCs	1,1,1-Trichloroethane	6	22	2.00	0.00	MW-3	3.40	4.13	5.00	10.00
	1,1-Dichloroethane	1	22	2.00	2.00	MW-5S-II	3.61	4.21	5.00	10.00
	1,2-Dichloroethene (tot)	1	22	3.00	3.00	MW-5S-II	3.66	4.23	5.00	10.00
	Acetone	1	22	150.00	150.00	MW-1S-II	12.59	12.53	10.00	21.00
	Chloromethane	1	22	2.00	2.00	MW-1S-II	4.06	5.27	10.00	10.00
BNAs	4-Methylphenol	1	22	3.00	3.00	MW-1S-II	5.02	6.52	10.00	50.00
	Butylbenzylphthalate	7	22	1.00	2.00	MW-1D-II	4.75	7.13	10.00	50.00
	Chrysene	1	22	1.00	1.00	MW-7D-II	5.73	7.01	10.00	50.00
	Di-n-octylphthalate	2	21	1.00	3.00	MW-4-II	5.67	7.06	10.00	50.00
	Diethylphthalate	3	22	1.00	2.00	MW-1S-II	4.55	5.57	10.00	10.00
	Fluoranthene	1	22	1.00	1.00	MW-7D-II	5.73	7.01	10.00	50.00
	Pyrene	3	22	1.00	3.00	MW-5S-II	5.45	7.22	10.00	50.00
	bis(2-Ethylhexyl)phthalate	13	22	5.00	160.00	MW-7S-II	28.40	53.84	10.00	20.00
Pest/PCBs	Heptachlor epoxide	1	22	0.08	0.00	MW-1S-II	0.03	0.03	0.05	0.08
Inorganics	Aluminum	17	22	314.00	53100.00	MW-2-II	6954.61	29941.17	190.00	520.00
	Arsenic	3	22	6.40	9.20	MW-2-II	2.37	3.09	2.00	10.00
	Barium	20	22	7.00	230.00	MW-2-II	57.94	138.80	3.00	19.20
	Cadmium	1	22	4.00	4.00	MW-7S	2.07	2.45	1.90	9.70
	Calcium	22	22	5000.00	50500.00	MW-5D-II	15860.41	20430.01	.	.
	Chromium	22	22	11.00	1440.00	MW-2-II	204.10	496.06	.	.
	Cobalt	3	22	10.50	20.30	MW-3-II	5.31	8.91	2.00	14.00
	Copper	17	22	26.00	179.00	MW-3-II	67.98	126.23	17.00	27.00
	Iron	22	22	450.00	40900.00	MW-2-II	6400.34	14695.15	.	.
	Lead	19	19	10.20	240.00	MW-2-II	48.66	66.40	.	.
	Magnesium	22	22	550.00	3070.00	MW-3-II	1740.95	2218.94	.	.
	Manganese	22	22	8.20	402.00	MW-3-II	91.42	151.15	.	.
	Mercury	5	22	0.06	0.20	MW-3	0.00	0.13	0.04	0.20
	Nickel	17	22	8.20	97.40	MW-1S-II	30.46	61.22	7.90	10.00
	Potassium	19	22	1010.00	61500.00	MW-5D	5798.07	7523.74	1690.00	1960.00
	Sodium	22	22	2090.00	52500.00	MW-2-II	18529.55	34908.10	.	.
	Vanadium	6	22	6.00	72.40	MW-2-II	10.48	24.75	1.00	6.00
	Zinc	10	19	45.00	342.00	MW-5D-II	99.20	131.41	65.50	65.50

SUMMARY S.ATISTICS FOR THE ANCHOR CHEMICAL SITE (CONTINUED).

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE

UNITS: all water analyses are ug/L; organic soils are ug/Kg; inorganic soils are mg/Kg.

-- TYPE=MW-6S and MW-6D/Ground Water --

Class	Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Arithmetic Mean Conc.	95 Pct. Upp. Conf. Limit	Lowest Observed Det.Limit	Highest Observed Det.Limit
BNAs	Butylbenzylphthalate	2	4	1.00	2.00	MW-6S-II	3.25	39.59	10.00	10.00
	Di-n-octylphthalate	1	4	1.00	1.00	MW-6S-II	4.00	57.96	10.00	10.00
	bis(2-Ethylhexyl)phthalate	2	4	5.00	6.00	MW-6S	7.50	21.34	10.00	28.00
Inorganics	Aluminum	2	4	353.00	803.00	MW-6S-II	387.75	2373.68	371.00	419.00
	Barium	4	4	7.00	75.70	MW-6S-II	40.70	2959.42	.	.
	Calcium	4	4	12400.00	24200.00	MW-6S-II	17450.00	28924.41	.	.
	Chromium	4	4	13.00	54.40	MW-6S-II	36.50	200.84	.	.
	Copper	2	4	33.00	79.80	MW-6S-II	33.83	1464.39	18.00	27.00
	Iron	4	4	458.00	1370.00	MW-6S-II	792.25	2025.63	.	.
	Lead	2	2	25.20	29.40	MW-6S-II	27.30	29.40	.	.
	Magnesium	4	4	1670.00	2760.00	MW-6S-II	2212.50	3089.16	.	.
	Manganese	4	4	20.90	92.00	MW-6D	53.58	270.84	.	.
	Nickel	2	4	20.90	22.00	MW-6D	12.96	375.63	7.90	10.00
	Potassium	4	4	2270.00	5010.00	MW-6D	3645.00	7596.05	.	.
	Sodium	4	4	10600.00	14000.00	MW-6S	12100.00	14331.68	.	.
	Zinc	3	3	48.00	97.00	MW-6S-II	79.73	354.88	.	.

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE (CONTINUED).

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE

UNITS: all water analyses are ug/L; organic soils are ug/Kg; inorganic soils are mg/Kg.

TYPE=Subsurface Soils

Class	Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Arithmetic Mean Conc.	95 Pct. Upp. Conf. Limit	Lowest Observed Det.Limit	Highest Observed Det.Limit
BNAs	4-Chloroaniline	1	6	78.00	78.00	MW-5D(5-7')	153.82	214.65	330.00	340.00
	bis(2-Ethylhexyl)phthalate	3	6	130.00	190.00	CP01_14-16'	162.49	185.36	339.90	350.00
Pest/PCBs	Dieldrin	2	6	3.40	7.50	CP02_14-16'	7.23	10.60	16.00	16.80
	Methoxychlor	1	6	14.00	14.00	CP02_14-16'	30.82	89.52	17.00	84.00
Inorganics	Aluminum	6	6	759.00	2150.00	MW-5D(5-7')	1364.00	2095.13	.	.
	Arsenic	6	6	0.55	1.50	IB-1(10-12')	0.88	1.33	.	.
	Barium	4	6	3.20	7.30	IB-1(10-12')	4.88	9.29	3.50	3.70
	Beryllium	1	4	0.22	0.22	CP02_14-16'	0.16	0.43	0.15	0.38
	Cadmium	2	6	0.92	1.20	IB-1(10-12')	0.59	1.27	0.61	0.80
	Calcium	4	6	134.00	4150.00	CP01_14-16'	1069.88	1457098.80	33.60	355.00
	Chromium	2	6	4.00	11.60	IB-1(10-12')	3.92	12.60	2.70	5.30
	Cobalt	1	6	0.59	0.59	CP01_14-16'	0.71	0.97	1.00	1.80
	Copper	2	4	2.70	2.70	CP01_14-16'	2.37	5.01	2.50	5.70
	Iron	6	6	3440.00	8410.00	IB-4(10-12')	4941.67	6982.20	.	.
	Lead	5	6	1.98	13.60	MW-5D(5-7')	3.77	16.61	1.98	1.90
	Magnesium	5	5	179.08	2640.08	CP01_14-16'	796.60	11879.20	.	.
	Manganese	6	6	26.50	78.00	MW-5D(5-7')	50.15	86.23	.	.
	Potassium	4	6	101.00	207.00	IB-2(10-12')	131.25	178.50	214.00	217.00
	Sodiu	3	6	132.00	174.00	IB-1(10-12')	119.37	1171.76	35.70	445.00
	Vanadium	6	6	2.30	8.00	IB-1(10-12')	4.22	6.74	.	.
	Zinc	3	5	5.88	8.70	IB-2(10-12')	5.26	19.99	4.20	4.20

SUMM. ‹ STATISTICS FOR THE ANCHOR CHEMICAL SITE (CONTINUED).

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE

UNITS: all water analyses are ug/L; organic soils are ug/Kg; inorganic soils are mg/Kg.

-- TYPE=Deep Subsurface Soils --

Class	Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Arithmetic Mean Conc.	95 Pct. Upp. Conf. Limit	Lowest Observed Det.Limit	Highest Observed Det.Limit
VOCs	2-Butanone	2	20	32.00	37.00	MW-7D(56-58')	8.78	10.77	10.00	21.00
BNAs	bis(2-Ethylhexyl)phthalate	6	20	36.00	400.00	IB-6(40-42')	186.77	253.53	330.00	693.00
Pest/PCBs	Heptachlor	1	20	0.20	0.20	MW-7D(114-116)	4.49	7.46	8.00	16.00
Inorganics	Aluminum	20	20	290.00	2130.00	IB-3(30-34')	888.35	1150.63	.	.
	Arsenic	16	20	0.24	1.90	IB-1(15-17')	0.70	1.03	0.40	0.40
	Barium	11	19	0.82	12.30	IB-3(30-34')	3.04	6.91	0.60	3.10
	Cadmium	4	20	0.59	0.92	IB-3(35-39')	0.48	0.58	0.40	1.40
	Calcium	1	14	160.00	160.00	IB-1(15-17')	31.06	44.94	21.50	74.00
	Chromium	14	20	1.40	23.20	MW-7D(56-58')	4.19	5.61	2.90	4.50
	Cobalt	2	20	1.50	1.60	IB-3(30-34')	0.95	1.09	1.00	2.00
	Iron	19	19	655.00	9140.00	IB-3(35-39')	3114.26	4574.32	.	.
	Lead	13	17	0.62	2.10	MW-6D(60-62')	1.00	1.27	0.03	1.20
	Magnesium	16	16	7.50	473.00	IB-3(30-34')	124.79	379.05	.	.
	Manganese	20	20	7.20	93.30	IB-3(30-34')	30.34	45.47	.	.
	Nickel	1	19	1.50	1.50	IB-3(30-34')	1.02	1.40	0.20	2.40
	Potassium	3	20	91.50	220.00	IB-3(30-34')	156.21	245.63	50.00	420.00
	Selenium	1	20	0.76	0.76	MW-5D(115-117)	0.36	0.42	0.40	0.80
	Sodium	2	18	164.00	201.00	MW-7D(56-58')	186.17	517.23	14.00	890.00
	Vanadium	8	20	1.40	6.10	IB-3(35-39')	1.97	2.58	1.20	3.50
	Zinc	7	13	3.10	12.90	IB-3(35-39')	4.47	10.36	1.70	4.00

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE (CONTINUED).

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE

UNITS: all water analyses are ug/L; organic soils are ug/Kg; inorganic soils are mg/Kg.

------------------ TYPE=MW-6S and MW-6D/Deep Subsurface Soils ------------------

Class	Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Arithmetic Mean Conc.	95 Pct. Upp. Conf. Limit	Lowest Observed Det.Limit	Highest Observed Det.Limit
BNAs	bis(2-Ethylhexyl)phthalate	1	2	36.00	36.00	MW-6D(60-62')	117.00	36.00	396.00	396.00
Inorganics	Aluminum	2	2	515.00	811.00	MW-6D(60-62')	663.00	811.00	.	.
	Arsenic	2	2	0.80	0.85	MW-6S(74-76')	0.82	0.85	.	.
	Chromium	2	2	3.40	3.70	MW-6S(74-76')	3.55	3.70	.	.
	Iron	2	2	1360.00	1960.00	MW-6D(60-62')	1660.00	1960.00	.	.
	Lead	2	2	1.30	2.10	MW-6D(60-62')	1.70	2.10	.	.
	Magnesium	2	2	28.50	34.60	MW-6S(74-76')	31.55	34.60	.	.
	Manganese	2	2	14.80	25.40	MW-6D(60-62')	20.10	25.40	.	.

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE (CONTINUED).

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE

UNITS: all water analyses are ug/L; organic soils are ug/Kg; inorganic soils are mg/Kg.

-- TYPE=Sediments --

Class	Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Arithmetic Mean Conc.	95 Pct. Upp. Conf. Limit	Lowest Observed Det.Limit	Highest Observed Det.Limit
VOCs	1,1,1-Trichloroethane	1	10	3300.00	3300.00	DW-2	390.30	102396.91	5.50	1150.00
	1,1-Dichloroethane	1	10	1600.00	1600.00	DW-2	220.30	29760.35	5.50	1150.00
	Carbon Disulfide	2	10	5.00	21.00	DW-9	167.28	13739.66	5.50	2100.00
	Ethylbenzene	1	10	4800.00	4800.00	DW-2	540.30	205239.90	5.50	1150.00
	Toluene	4	10	5.00	4000.00	DW-2	571.43	881540.52	5.50	1100.00
	Total Xylenes	1	10	67000.00	67000.00	DW-2	6760.30	79976639.93	5.50	1150.00
BNAs	2-Methylnaphthalene	1	10	3900.00	3900.00	DW-2	1381.98	2994.05	339.90	2999.70
	Benzo(a)pyrene	2	10	500.00	1100.00	DW-8	1034.91	1908.96	339.90	2999.70
	Benzo(b)fluoranthene	2	10	1000.00	2700.00	DW-8	1236.91	2480.21	339.90	2999.70
	Benzo(k)fluoranthene	2	10	560.00	1700.00	DW-8	1092.91	2106.12	339.90	2999.70
	Benzoic Acid	1	10	73.00	73.00	DW-5	5412.90	65294.75	8720.00	14544.00
	Butylbenzylphthalate	6	10	40.00	5200.00	DW-2	1676.77	13698.72	1798.50	2999.70
	Chrysene	4	10	230.00	2000.00	DW-8	956.87	2578.16	339.90	2999.70
	Di-n-butylphthalate	3	10	72.00	2500.00	DW-2	1185.15	4062.68	1798.50	2999.70
	Fluoranthene	4	10	410.00	3400.00	DW-0	1151.87	2729.00	339.90	2999.70
	Indeno(1,2,3-cd)pyrene	1	10	1500.00	1500.00	DW-8	1131.92	2101.22	339.90	2999.70
	Naphthalene	1	10	9500.00	9500.00	DW-2	1941.98	5128.06	339.90	2999.70
	Phenanthrene	5	10	190.00	1500.00	DW-8	769.94	2068.05	339.90	2999.70
	Pyrene	5	10	34.00	4500.00	DW-8	1238.27	9186.62	1798.50	2999.70
	bis(2-Ethylhexyl)phthalate	3	10	21000.00	27000.00	DW-2	9610.00	60724.93	1000.00	12000.00
Pest/PCBs	4,4'-DDE	6	9	10.00	146.00	DW-2	42.44	479.35	6.00	9.44
	Dieldrin	6	9	16.00	106.00	DW-2	33.33	233.65	6.00	9.44
	Endrin	2	9	12.00	36.00	DW-2	0.95	17.27	6.00	12.00
	Methoxychlor	7	9	7.50	126.00	DW-4	35.66	81.56	34.40	30.40
	alpha-BHC	1	9	183.80	183.00	DW-2	22.47	106.43	3.44	6.00
	beta-BHC	1	9	8.20	8.20	DW-9	3.09	4.40	3.44	6.00
	gamma-chlordane	2	9	3.40	29.00	DW-8	22.58	46.84	34.40	60.00
Inorganics	Aluminum	10	10	1410.00	11100.00	DW-8	4723.50	9376.31	.	.
	Antimony	3	10	9.50	12.00	DW-1	5.35	8.29	6.40	6.40
	Arsenic	9	10	0.79	3.70	DW-2	2.18	3.52	0.00	0.00
	Barium	10	10	9.70	90.20	DW-2	39.20	78.12	.	.
	Calcium	10	10	1300.80	22980.00	DW-3	10363.00	26585.34	.	.
	Chromium	10	10	17.40	463.00	DW-2	124.46	400.50	.	.
	Cobalt	8	10	3.10	11.30	DW-8	5.82	11.08	3.40	3.40
	Copper	10	10	5.30	266.00	DW-2	67.90	257.42	.	.
	Iron	10	10	3810.80	22700.00	DW-8	12444.50	28604.89	.	.
	Lead	10	10	81.30	1620.00	DW-8	542.33	2004.60	.	.
	Magnesium	10	10	950.00	14100.00	DW-8	5797.50	15318.92	.	.
	Manganese	10	10	51.45	189.00	DW-9	107.55	159.68	.	.
	Mercury	2	9	0.26	0.34	DW-7	0.11	0.22	0.10	0.10
	Nickel	10	10	4.70	21.50	DW-2	13.04	19.71	.	.
	Potassium	10	10	186.00	615.00	DW-6	333.10	453.81	.	.
	Sodium	6	6	447.00	1240.00	DW-0	766.00	1296.77	.	.
	Thallium	2	10	0.28	0.33	DW-2	0.14	0.28	0.20	0.20
	Vanadium	10	10	5.30	81.00	DW-8	38.25	92.46	.	.
	Zinc	10	10	24.60	1770.00	DW-2	386.00	1944.01	.	.

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE (CONTINUED).

SUMMARY STATISTICS FOR THE ANCHOR CHEMICAL SITE

UNITS: all water analyses are ug/L; organic soils are ug/Kg; inorganic soils are mg/Kg.

-- TYPE=Deep Soils + Deep Sediments --

Class	Analyte	Num. Times Detected	Num. Samples Analyzed	Lowest Detected Conc.	Highest Detected Conc.	Highest Conc. Locat.	Arithmetic Mean Conc.	95 Pct. Upp. Conf. Limit	Lowest Observed Det.Limit	Highest Observed Det.Limit
VOCs	2-Butanone	2	29	32.00	37.00	MW-7D(56-58')	229.21	466.38	10.00	7780.00
	Ethylbenzene	1	29	4800.00	4800.00	DW-2(15-17')	212.76	297.41	5.00	650.00
	Toluene	1	29	2300.00	2300.00	DW-2(15-17')	126.55	221.78	5.00	650.00
	Total Xylenes	1	29	82000.00	82000.00	DW-2(15-17')	2874.83	1231.38	5.00	650.00
BNAs	2-Methylnaphthalene	1	29	150.00	150.00	DW-2(15-17')	188.81	198.27	330.00	693.00
	4-Chloro-3-methylphenol	1	29	570.00	570.00	DW-2(25-27')	204.31	228.58	330.00	693.00
	Butylbenzylphthalate	1	29	410.00	410.00	DW-2(15-17')	197.77	211.09	330.00	693.00
	Di-n-butylphthalate	1	29	100.00	100.00	DW-2(15-17')	187.08	199.06	330.00	693.00
	Fluoranthene	1	29	69.00	69.00	DW-2(15-17')	186.01	202.03	330.00	693.00
	Naphthalene	1	29	290.00	290.00	DW-2(15-17')	193.63	204.33	330.00	693.00
	Phenanthrene	1	29	58.00	58.00	DW-2(15-17')	185.63	204.04	330.00	693.00
	Pyrene	1	29	66.00	66.00	DW-2(15-17')	185.91	202.51	330.00	693.00
	bis(2-Ethylhexyl)phthalate	6	29	36.00	400.00	IB-6(40-42')	254.39	321.85	330.00	2700.00
Pest/PCBs	Aroclor-1254	1	29	230.00	230.00	DW-2(15-17')	84.32	101.99	67.20	336.00
	Endosulfan II	1	29	1.10	1.10	DW-2(25-27')	7.72	9.94	6.72	33.60
	Heptachlor	1	29	0.20	0.20	MW-7D(114-116)	3.84	5.55	3.36	16.80
	alpha-BHC	1	29	1.30	1.30	DW-2(25-27')	3.89	4.72	3.36	16.80
	delta-BHC	1	29	2.00	2.00	DW-2(25-27')	3.91	4.68	3.36	16.80
Inorganics	Aluminum	29	29	290.00	3730.00	DW-2(15-17')	1086.64	1356.67	.	.
	Antimony	1	28	9.60	9.60	DW-2(15-17')	4.13	4.63	4.80	10.00
	Arsenic	23	29	0.24	2.30	DW-6(35-37')	0.82	1.18	0.40	0.80
	Barium	19	28	0.82	20.50	DW-2(15-17')	4.52	10.23	0.60	3.10
	Cadmium	5	29	0.59	2.00	DW-2(15-17')	0.51	0.59	0.40	1.40
	Calcium	3	16	160.00	11000.00	DW-2(15-17')	724.87	946.95	21.50	74.00
	Chromium	18	24	1.40	392.00	DW-2(15-17')	21.79	21.02	2.90	4.50
	Cobalt	3	29	1.50	4.20	DW-2(15-17')	1.24	1.45	1.00	3.40
	Copper	3	25	2.40	36.90	DW-2(15-17')	3.04	3.19	1.50	7.40
	Cyanide	2	22	1.60	6.20	DW-2(15-17')	0.88	1.08	0.50	2.00
	Iron	28	28	655.00	12000.00	DW-2(15-17')	3256.82	4223.35	.	.
	Lead	22	26	0.62	130.00	DW-2(15-17')	6.43	4.77	0.03	1.20
	Magnesium	23	23	7.50	473.00	IB-3(30-34')	132.52	278.39	.	.
	Manganese	29	29	7.20	93.30	IB-3(30-34')	31.99	42.95	.	.
	Nickel	1	28	1.50	1.50	IB-3(30-34')	1.54	2.20	0.20	7.60
	Potassium	5	29	91.50	228.00	IB-3(30-34')	141.16	188.15	50.00	420.00
	Selenium	1	29	0.76	0.76	MW-5D(115-117)	0.39	0.44	0.40	1.00
	Sodium	8	24	47.60	593.00	DW-2(15-17')	101.79	369.03	14.00	890.00
	Vanadium	17	29	1.40	41.40	DW-2(15-17')	3.55	4.02	1.20	3.50
	Zinc	16	22	3.10	432.00	DW-2(15-17')	26.32	34.08	1.70	4.80

APPENDIX B

HUMAN HEALTH RISK SPREADSHEETS

TABLE : 1
SITE : ANCHOR CHEMICAL
ENV. MEDIUM : GROUND WATER
EXPOSURE TYPE : INGESTION/FUTURE/RESIDENT
RISK TYPE : CARCINOGENIC

Contaminant of Concern	*Concentration (mg/l)*	*Exposure Factor (a) (l/kg/day)*	*Ingested Dose (b) (mg/kg/day)*	*Oral Slope Factor (mg/kg/day)-1*	*Increased CA Risk*
1,1,1-TRICHLOROETHANE	4.05E-03	1.17E-02	4.76E-05	NA	
BUTYLBENZYLPHTHALATE	2.00E-03	1.17E-02	2.35E-05	NA	
DI-N-OCTYLPHTHALATE	3.00E-03	1.17E-02	3.52E-05	NA	
DIETHYLPHTHALATE	2.00E-03	1.17E-02	2.35E-05	NA	
PYRENE	3.00E-03	1.17E-02	3.52E-05	NA	
BIS(2-ETHYLHEXYL)PHTHALATE	5.02E-02	1.17E-02	5.89E-04	1.40E-02	8E-06
ALUMINUM	2.84E+01	1.17E-02	3.33E-01	NA	
ARSENIC	2.96E-03	1.17E-02	3.48E-05	2.00E+00	7E-05
BARIUM	1.29E-01	1.17E-02	1.51E-03	NA	
CHROMIUM	5.02E-01	1.17E-02	5.89E-03	NA	
COBALT	8.61E-03	1.17E-02	1.01E-04	NA	
COPPER	1.21E-01	1.17E-02	1.42E-03	NA	
IRON	1.44E+01	1.17E-02	1.69E-01	NA	
LEAD	6.64E-02	1.17E-02	7.80E-04	NA	
MANGANESE	1.45E-01	1.17E-02	1.70E-03	NA	
MERCURY	1.20E-04	1.17E-02	1.41E-06	NA	
NICKEL	5.82E-02	1.17E-02	6.83E-04	NA	
VANADIUM	2.20E-02	1.17E-02	2.58E-04	NA	
ZINC	1.31E-01	1.17E-02	1.54E-03	NA	

TOTAL RISK	8E-05

a. Exposure Factor = (2l/day * 350 day/yr * 30 yr)/(70 kg * 25550 day) = 0.0117 l/kg/day
b. Ingested Dose = Concentration * Exposure Factor

TABLE : 2
SITE : ANCHOR CHEMICAL
ENV. MEDIUM : GROUND WATER
EXPOSURE TYPE : INGESTION/FUTURE/RESIDENT
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/l)*	*Exposure Factor (a) (l/kg/day)*	*Ingested Dose (b) (mg/kg/day)*	*Chronic Protective Body Dose (mg/kg/day)*	*Chronic Hazard Quotient*
1,1,1-TRICHLOROETHANE	4.05E-03	2.74E-02	1.11E-04	9.00E-02	1E-03
BUTYLBENZYLPHTHALATE	2.00E-03	2.74E-02	5.48E-05	2.00E-01	3E-04
DI-N-OCTYLPHTHALATE	3.00E-03	2.74E-02	8.22E-05	2.00E-02	4E-03
DIETHYLPHTHALATE	2.00E-03	2.74E-02	5.48E-05	8.00E-01	7E-05
PYRENE	3.00E-03	2.74E-02	8.22E-05	3.00E-02	3E-03
BIS(2-ETHYLHEXYL)PHTHALATE	5.02E-02	2.74E-02	1.38E-03	2.00E-02	7E-02
ALUMINUM	2.84E+01	2.74E-02	7.77E-01	1.00E+00	8E-01
ARSENIC	2.96E-03	2.74E-02	8.11E-05	3.00E-04	3E-01
BARIUM	1.29E-01	2.74E-02	3.53E-03	7.00E-02	5E-02
CHROMIUM	5.02E-01	2.74E-02	1.37E-02	8.76E-01	2E-02
COBALT	8.61E-03	2.74E-02	2.36E-04	NA	
COPPER	1.21E-01	2.74E-02	3.32E-03	5.50E-02	6E-02
IRON	1.44E+01	2.74E-02	3.95E-01	5.00E-01	8E-01
LEAD	6.64E-02	2.74E-02	1.82E-03	NA	
MANGANESE	1.45E-01	2.74E-02	3.97E-03	5.00E-03	8E-01
MERCURY	1.20E-04	2.74E-02	3.29E-06	3.00E-04	1E-02
NICKEL	5.82E-02	2.74E-02	1.59E-03	2.00E-02	8E-02
VANADIUM	2.20E-02	2.74E-02	6.02E-04	7.00E-03	9E-02
ZINC	1.31E-01	2.74E-02	3.60E-03	3.00E-01	1E-02

HAZARD INDEX	3E+00

a. Exposure Factor = (2l/day * 350day/yr * 30yr)/(70kg * 10950day) = 0.0274 l/kg/day
b. Ingested Dose = Concentration * Exposure Factor

TABLE : 3
SITE : ANCHOR CHEMICAL
EXPOSURE ROUTE : GROUND WATER/SHOWER VAPORS
RECEPTOR : INHALATION/FUTURE/RESIDENT
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/l)*	*Molecular Weight (g/mole)*	*Henry's Law Constant (atm-m3/mole)*	*Chemical-Specific kl (cm/hr)*	*Chemical-Specific kv (cm/hr)*	*Adjusted Overall Mass Transfer Coefficient (cm/hr)*	*Emitted Concentration (mg/l)*	*VOC Generation Rate (mg/m3-min)*	*Single Inhaled Dose (mg/kg/event)*	*Exposure Factor (b) (event/day)*	*Chronic Inhaled Dose (c) (mg/kg/day)*	*Chronic Protective Body Dose (mg/kg/day)*	*Chronic Hazard Quotient*
1,1,1-TRICHLOROETHANE	4.05E-03	133.4	1.44E-02	1.15E+01	1.10E+03	1.52E+01	1.61E-03	2.68E-03	5.38E-05	9.59E-01	5.16E-05	0.3	2E-04

HAZARD INDEX	2E-04

a Henry's Law Constant is the average of the values for cis- and trans- isomers
b Exposure Factor = (350 days/year * 30 years)/(365 days/year * 30 years) = 0.959
c Chronic Inhaled Dose = Single Inhaled Dose * Exposure Factor

NOTE: Formulae for the calculation of values in the above table are provided in Appendix B of this report

TABLE : 4
SITE : ANCHOR CHEMICAL
EXPOSURE ROUTE : SEDIMENT/SUBSURFACE SOIL
RECEPTOR : INGESTION/FUTURE/EXCAVATION WORKER
RISK TYPE : CARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Exposure Factor (a)*	*Daily Exposure Dose (b) (mg/kg/day)*	*Oral Slope Factor (mg/kg/day)-1*	*Increased CA Risk*
1,1,1-TRICHLOROETHANE	5.29E-01	1.74E-08	9.23E-09	NA	
1,1-DICHLOROETHANE	2.97E-01	1.74E-08	5.19E-09	NA	
CARBON DISULFIDE	2.10E-02	1.74E-08	3.66E-10	NA	
ETHYLBENZENE	7.30E-01	1.74E-08	1.27E-08	NA	
TOLUENE	2.80E+00	1.74E-08	4.88E-08	NA	
TOTAL XYLENES	1.13E+01	1.74E-08	1.97E-07	NA	
2-METHYLNAPHTHALENE	2.34E+00	1.74E-08	4.08E-08	NA	
4-CHLOROANILINE	7.80E-02	1.74E-08	1.36E-09	NA	
BENZO(A)PYRENE	1.10E+00	1.74E-08	1.92E-08	7.30E+00	1E-07
BENZO(B)FLUORANTHENE	2.01E+00	1.74E-08	3.51E-08	7.30E-01	3E-08
BENZO(K)FLUORANTHENE	1.67E+00	1.74E-08	2.91E-08	7.30E-01	2E-08
BENZOIC ACID	7.30E-02	1.74E-08	1.27E-09	NA	
BUTYLBENZYLPHTHALATE	3.65E+00	1.74E-08	6.36E-08	NA	
CHRYSENE	1.38E+00	1.74E-08	2.40E-08	7.30E-02	2E-09
DI-N-BUTYLPHTHALATE	2.17E+00	1.74E-08	3.78E-08	NA	
FLUORANTHENE	1.71E+00	1.74E-08	2.98E-08	NA	
INDENO(1,2,3-CD)PYRENE	1.50E+00	1.74E-08	2.62E-08	7.30E-01	2E-08
NAPHTHALENE	3.32E+00	1.74E-08	5.79E-08	NA	
PHENANTHRENE	9.99E-01	1.74E-08	1.74E-08	NA	
PYRENE	2.53E+00	1.74E-08	4.42E-08	NA	
BIS(2-ETHYLHEXYL)PHTHALATE	2.70E+01	1.74E-08	4.71E-07	1.40E-02	7E-09
4,4'-DDE	1.08E-01	1.74E-08	1.88E-09	3.40E-01	6E-10
DIELDRIN	5.11E-02	1.74E-08	8.92E-10	1.60E+01	1E-08
ENDRIN	1.23E-02	1.74E-08	2.15E-10	NA	
METHOXYCHLOR	5.47E-02	1.74E-08	9.54E-10	NA	
ALPHA-BHC	1.90E-02	1.74E-08	3.31E-10	6.30E+00	2E-09
BETA-BHC	4.43E-03	1.74E-08	7.73E-11	1.80E+00	1E-10
GAMMA-CHLORDANE	2.90E-02	1.74E-08	5.06E-10	1.30E+00	7E-10
ALUMINUM	5.74E+03	1.74E-08	1.00E-04	NA	
ANTIMONY	6.32E+00	1.74E-08	1.10E-07	NA	
ARSENIC	2.47E+00	1.74E-08	4.31E-08	2.00E+00	9E-08
BARIUM	8.70E+01	1.74E-08	1.52E-06	NA	
BERYLLIUM	2.20E-01	1.74E-08	3.84E-09	4.30E+00	2E-08
CADMIUM	1.00E+00	1.74E-08	1.74E-08	NA	
CHROMIUM	4.63E+02	1.74E-08	8.08E-06	NA	
COBALT	9.98E+00	1.74E-08	1.74E-07	NA	
COPPER	2.66E+02	1.74E-08	4.64E-06	NA	
IRON	1.41E+04	1.74E-08	2.46E-04	NA	
LEAD	1.62E+03	1.74E-08	2.83E-05	NA	
MANGANESE	1.22E+02	1.74E-08	2.13E-06	NA	
MERCURY	1.20E-01	1.74E-08	2.09E-09	NA	
NICKEL	2.15E+01	1.74E-08	3.75E-07	NA	
THALLIUM	1.90E-01	1.74E-08	3.31E-09	NA	
VANADIUM	7.52E+01	1.74E-08	1.31E-06	NA	
ZINC	1.77E+03	1.74E-08	3.09E-05	NA	

TOTAL RISK	3E-07

a. Exposure Factor = (480 mg/day * 65 days/yr * 1 yr * 1E-06 kg/mg)/(70 kg * 365 days/year * 70 years) = 1.74 E-08
b. Daily Exposure Dose = Concentration * Exposure Factor

TABLE : 5
SITE : ANCHOR CHEMICAL
EXPOSURE ROUTE : SEDIMENT/SUBSURFACE SOIL
RECEPTOR : INGESTION/FUTURE/EXCAVATION WORKER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration* (mg/kg)	*Exposure Factor (a)*	*Daily Exposure Dose (b)* (mg/kg/day)	*Subchronic Protective Body Dose* (mg/kg/day)	*Hazard Quotient*
1,1,1-TRICHLOROETHANE	5.29E-01	4.90E-06	2.59E-06	9.00E-01	3E-06
1,1-DICHLOROETHANE	2.97E-01	4.90E-06	1.46E-06	1.00E+00	1E-06
CARBON DISULFIDE	2.10E-02	4.90E-06	1.03E-07	1.00E-01	1E-06
ETHYLBENZENE	7.30E-01	4.90E-06	3.58E-06	1.00E+00	4E-06
TOLUENE	2.80E+00	4.90E-06	1.37E-05	2.00E+00	7E-06
TOTAL XYLENES	1.13E+01	4.90E-06	5.52E-05	4.00E+00	1E-05
2-METHYLNAPHTHALENE	2.34E+00	4.90E-06	1.15E-05	NA	
4-CHLOROANILINE	7.80E-02	4.90E-06	3.82E-07	4.00E-03	1E-04
BENZO(A)PYRENE	1.10E+00	4.90E-06	5.39E-06	NA	
BENZO(B)FLUORANTHENE	2.01E+00	4.90E-06	9.85E-06	NA	
BENZO(K)FLUORANTHENE	1.67E+00	4.90E-06	8.16E-06	NA	
BENZOIC ACID	7.30E-02	4.90E-06	3.58E-07	4.00E+00	9E-08
BUTYLBENZYLPHTHALATE	3.65E+00	4.90E-06	1.79E-05	2.00E+00	9E-06
CHRYSENE	1.38E+00	4.90E-06	6.75E-06	NA	
DI-N-BUTYLPHTHALATE	2.17E+00	4.90E-06	1.06E-05	1.00E+00	1E-05
FLUORANTHENE	1.71E+00	4.90E-06	8.38E-06	4.00E-01	2E-05
INDENO(1,2,3-CD)PYRENE	1.50E+00	4.90E-06	7.35E-06	NA	
NAPHTHALENE	3.32E+00	4.90E-06	1.63E-05	NA	
PHENANTHRENE	9.99E-01	4.90E-06	4.89E-06	NA	
PYRENE	2.53E+00	4.90E-06	1.24E-05	3.00E-01	4E-05
BIS(2-ETHYLHEXYL)PHTHALATE	2.70E+01	4.90E-06	1.32E-04	2.00E-02	7E-03
4,4'-DDE	1.08E-01	4.90E-06	5.27E-07	NA	
DIELDRIN	5.11E-02	4.90E-06	2.50E-07	5.00E-05	5E-03
ENDRIN	1.23E-02	4.90E-06	6.02E-08	3.00E-04	2E-04
METHOXYCHLOR	5.47E-02	4.90E-06	2.68E-07	5.00E-03	5E-05
ALPHA-BHC	1.90E-02	4.90E-06	9.29E-08	NA	
BETA-BHC	4.43E-03	4.90E-06	2.17E-08	NA	
GAMMA-CHLORDANE	2.90E-02	4.90E-06	1.42E-07	6.00E-05	2E-03
ALUMINUM	5.74E+03	4.90E-06	2.81E-02	1.00E+00	3E-02
ANTIMONY	6.32E+00	4.90E-06	3.10E-05	4.00E-04	8E-02
ARSENIC	2.47E+00	4.90E-06	1.21E-05	3.00E-04	4E-02
BARIUM	8.70E+01	4.90E-06	4.26E-04	7.00E-02	6E-03
BERYLLIUM	2.20E-01	4.90E-06	1.08E-06	5.00E-03	2E-04
CADMIUM	1.00E+00	4.90E-06	4.90E-06	5.00E-04	1E-02
CHROMIUM	4.63E+02	4.90E-06	2.27E-03	8.77E-01	3E-03
COBALT	9.98E+00	4.90E-06	4.89E-05	NA	
COPPER	2.66E+02	4.90E-06	1.30E-03	5.50E-02	2E-02
IRON	1.41E+04	4.90E-06	6.91E-02	5.00E-01	1E-01
LEAD	1.62E+03	4.90E-06	7.93E-03	NA	
MANGANESE	1.22E+02	4.90E-06	5.98E-04	1.00E-01	6E-03
MERCURY	1.20E-01	4.90E-06	5.88E-07	3.00E-04	2E-03
NICKEL	2.15E+01	4.90E-06	1.05E-04	2.00E-02	5E-03
THALLIUM	1.90E-01	4.90E-06	9.31E-07	7.00E-04	1E-03
VANADIUM	7.52E+01	4.90E-06	3.68E-04	7.00E-03	5E-02
ZINC	1.77E+03	4.90E-06	8.67E-03	3.00E-01	3E-02

HAZARD INDEX	4E-01

a. Exposure Factor = (480 mg/day * 65 days/yr * 1 yr * 1E-06 kg/mg)/(70 kg * 91 days) = 4.90 E-06
b. Daily Exposure Dose = Concentration * Exposure Factor

TABLE : 6
SITE : ANCHOR CHEMICAL
EXPOSURE ROUTE : SEDIMENT/SUBSURFACE SOIL
RECEPTOR : DERMAL CONTACT/FUTURE/EXCAVATION WORKER
RISK TYPE : NONCARCINOGENIC

Contaminant of Concern	*Concentration (mg/kg)*	*Exposure Factor (a)*	*Daily Exposure Dose (b) (mg/kg/day)*	*Chronic Protective Body Dose (c) (mg/kg/day)*	*Chronic Hazard Quotient*
CADMIUM	1.00E+00	1.91E-07	1.91E-07	5.00E-04	4E-04
				HAZARD INDEX	4E-04

a Exposure Factor = (3120 cm2 * 0.6 mg/cm2 * 0.01 * 65 days/year * 1 year * 1E-06 kg/mg)/(70 kg * 91 days) = 1.91 E-07
b Daily Exposure Dose = Concentration * Exposure Factor
c Oral RFD for cadmium is based on an absorbed dose; therefore an oral absorption factor was not applied.

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APPENDIX C

TOXICITY PROFILES

VOLATILES

CARBON DISULFIDE

Use

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

Chemical and Physical Properties

Chemical Formula: CS_2

MW:	76.13	BP:	46.3°C
Sol. (water):	2,940 mg/liter	MP:	-108.6 to -116.6°C
Sol. (organics):	miscible with anhydrous methanol, ethanol, ether, benzene, chloroform, carbon tetrachloride, and oil	VP:	297 mmHg at 20°C

Fate and Transport

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

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

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

Pharmacokinetics

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

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

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

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

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

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

Vigliani (1954) reported that exposure to carbon disulfide at 144-321 ppm may cause psychosis, polyneuritis (absent of weakened achilles or patellar reflexes) tremors, weakness of limbs, myopathy and vertigo in a study from the viscose rayon industry.

Teratogenic and Other Development Effects

Jones-Price et. al. (1984a,b) exposed pregnant CD rats and New Zealand White Rabbits to carbon disulfide in corn oil by gavage. CD rats were exposed to 0, 100, 200, 400, or 600 mg/kg/day for days 6-15 of gestation and killed on day 20. Rabbits were exposed to 0, 25, 75, or 150 mg/kg/day on days 6-19 of gestation and killed on day 30. Maternal toxicity effects (abnormal posture, rigidity or paralysis of hind limbs, ataxia, lethargy, rough or erect coat, decreased body weight, decreased gravid uterine weight, increased liver weight), occurred in rabbits at 75 and 150 mg/kg/day and in rats at all doses, with the most severe effects of 400 and 600 mg/kg/day. Rats exhibited no compound-related effects on number of implants/litter, proportion of litters with deal, resorbed or affected fetuses, percent resorbed, dead or affected fetuses/litter, number of live fetuses/live litter, and percent males/live litter. They did exhibit a significant dose-related decrease in average fetal body weight/litter. The rabbits exposed to 150 mg/kg/day exhibited an increase in percent resorbtions (litter and percent malformations/litter). The U.S. EPA (1986) states that a significantly increased number of resorptions/litter in rabbits at a level where no maternal toxicity occurred (25 mg/kg/day) suggests that ingested carbon disulfide may have a primary effect on the developing fetus.

Gondzik (1971) exposed "mongrel" rats to 12.5 mg/kg or 25 mg/kg distilled carbon disulfide dissolved in peanut oil intraperitoneally every second day for 60 days or 120 days. Rats exposed to 12.5 mg/kg/2 days for 60 days showed no testicular effects. Those exposed to 25 mg/kg/2 days for 60 days exhibited thickened vascular walls in the testis vessels engorged with RBCs, disorganization of the layers of seminiferous epithelium, and a reduction in the number of spermatozoa in the tubular lumen. Rats exposed to this higher dose for 120 days exhibited advanced regressive lesions involving all structural parts of the testis; including folding and shrinking of the tubular basement membrane, scant stomal tissue, and loss of spermatogonia. Spermatogenesis was absent but Sertoli cells were present in every tubule. The number of leadig cells in the intertubular spaces was reduced and the cells showed signs of degenerative vacuolation. The authors suggest that the nature of these effects indicate irreversibility.

Mutagenic Effects

Hedenstedt et. al. (1979) found that carbon disulfide was not mutagenic in bacterial reverse mutation assays with *Salmonella typhimurium* strain TA100 with or without S-9. Donner et. al. (1981) reported similar results for strains. TA98 and TA100 and Haworth et. al. (1983) also had similar results for strains TA98, TA100, TA1535, and TA1537. Donner et. al. (1981) also reported negative results in sex-linked recessive lethal studies in *Drosophila melanogaster* and Beliles et. al. (1980) reported a lack of mutagenicity in bone marrow cytogenesis and in dominant-lethal studies in rats.

Carcinogenic Effects

Checkoway et. al. (1984) and Wilcosky et. al. (1984) reported an association between lymphocytic leukemia and multiple solvent exposure. Eleven male hourly workers from the U.S. rubber industry whose deaths were attributed to lymphocytic leukemia were compared to a control group. Of the 24 solvents, the workers were exposed to, carbon disulfide and carbon tetrachloride, had the strongest association with leukemia. There was no association between exposure to carbon disulfide and any other cancer. Wilcosky et. al. (1984) however, suggests "cautious interpretation" due to the large number of variables and that the interpretation of this apparent association between carbon disulfide and leukemia is unclear. In contradiction to these studies, Nurimen and Hernberg (1984) found no association between occupational exposure to carbon disulfide and cancer mortality in a 15-year prospective follow-up study on viscose rayon workers.

Ecotoxicity

No data regarding the toxicity of carbon disulfide to aquatic or terrestrial life, domestic or wild, were found in the literature reviewed.

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity.

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	1×10^{-1} mg/kg/day
Chronic Inhalation RfD:	2.86×10^{-3} mg/kg/day
Subchronic Oral RfD:	1.0×10^{-1} mg/kg/day

Subchronic Inhalation RfD: 2.86 x 10^{-3} mg/kg/day
MCL: NA
AWQC: NA

BIBLIOGRAPHY

Atkinson, R. et. al., Rate Constants for the Reaction of Hydroxyl Radicals with Carbonyl Sulfide, Carbon Disulfide and Dimethyl Thither Over the Temperature Range 299-430°K, *Chem. Phys, Lett.* 54: 14-18, 1978, (Cited in HEEP, 1986).

Beliles, R.P. et. al., *Teratogenic-Mutagenic Risk of Workplace Contaminants: Trichlorocthylene, Perchloroethylene and Carbon Disulfide*, Prepared by Litton Bionetics, Inc., under contract number 210-77-0047, NIOSH, Washington, D.C., 1980, (Cited in HEEP, 1986).

Berg, G.L., *1981 Farm Chemical Handbook*, Mister Publishing Co., Willoughby, OH, p. C-62 - C-63, 1981, (Cited in HEEP, 1986).

Bergman, K. et. al., Tissue Disposition of Carbon Disulfide: I. Whole-Body Autoradiography of ^{35}S- and ^{14}C- Labeled Carbon Disulfide in Adult Male Mice, *Acta Pharmacol. Toxicol.* 54(2): 141-150, 1984, (Cited in HEEP, 1986).

Bremner, J.M., and W.L. Banwait, Sorption of Sulfur Gases by Soils, *Soil Biol. Biochem.* 8:79-83, 1976, (Cited in HEEP, 1986).

Checkoway, H. et. al., An evaluation of the associations of leukemia and rubber industry solvent exposures, *Am. J. Ind. Med.*, 5(3): 239-249, 1984.

CMR (Chemical Marketing Reporter), *Chemical Profile: Carbon Disulfide*, January 24, 1083, (Cited in HEEP, 1986).

Donner, M. et. al., Carbon Disulfide is not Mutagenic in Bacteria or Drosophila, *Mutat. Res.* 91(3): 163-166 1981, (Cited in HEEP, 1986).

Gondzik, M., Effect of carbon disulfide on testicles and kidneys, *Med. Pr.*, 27(1): 21, 1976.

Hedenstet, A. et. al., Mutagenicity and Metabolism Studies on 12 Thorium and Dithiocarbamate Compounds Used as Accelerators in the Swedish Rubber Industry, *Mutat. Res.* 68: 313-325, 1979.

Jones-Price, C. et. al., Teratologic evaluation of carbon disulfide (CAS No. 75-15-0) administered to CD rats on gestational days 6 through 15. *Gov. Rep. Announce. Index (U.S.)*, 84(15)80, FDA/NCTR-84/78, 1984a.

McKenna, K.J. and V. DiStefano, Carbon Disulfide I., The Metabolism of Inhaled Carbon Disulfide in the Rat, *J. Pharmacol, Exp. Ther.* 202(2): 245-252, 1977, (Cited in HEEP, 1986).

Nurimen, M. and S. Hernberg, Cancer mortality among carbon disulfide exposed workers, *J. Occ. Med.*, 26(5): 341, 1984.

Peyton, T.O. et al., *Carbon Disulfide, Carbonyl Sulfide, Literature Review and Environmental Assessment,* ORD, U.S., EPA, Washington, D.C., EPA 600/9-78-009, 1978, (Cited in HEEP, 1986)

Pilarski, K. et. al., Effect of carbon disulfide on the hematopoietic system of rats, I, Evaluation of peripheral blood, *Acta Haematol. Pol.*, 4(1): 33, 1973.

Timmerman, R.W., *Carbon Disulfide In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 4, 3rd ed.,* M. Grayson and D. Eckroth, Ed., John Wiley and Sons, Inc., New York, p. 742-757, 1978, (Cited in HEEP, 1986)

U.S. EPA, *Drinking Water Regulations and Health Advisories.*

U.S. EPA, *Health and Environmental Effects Profile for Carbon Disulfide*, EPA/600/X-86/155, June, 1986.

U.S. EPA, *Integrated Risk Information System (IRIS).*

U.S. EPA, *Health Effects Assessment Summary Tables (HEAST).* FY1991.

U.S. EPA, OERR. *CERCLA Compliance with Other Laws Manual Interim Final.* August, 1988.

U.S. EPA, *Oil and Hazardous Materials Technical Assistance Data System,* On line, 1985, (Cited in HEEP, 1986).

Vigliani, E.C., Carbon disulfide poisoning in viscose rayon factories, *Br. J. Ind. Med.*, 11:235-244, 1954.

Wilcosky, T.C. et. al., Cancer mortality and solvent exposures in the rubber industry, *Am. Id. Hyg. Assoc. J.*, 45(12): 809-811, 1984.

Windholz, M., Ed, *The Merck Index,* 10th ed., Merck and Co., Rahway, NJ, p. 1,796, 1983, (Cited in HEEP, 1986).

Wood, W.P. and J. Heicklan, *The Photooxidation of Carbon Disulfide*, J. Phys. Chem., 75: 854-860, 1971, (Cited in HEEP, 1986).

1,1-DICHLOROETHANE

Use

1,1-dichloroethane, also known as asymmetrical dichloroethane, ethylidene chloride and 1,1-ethylidene chloride; is used as a solvent and cleaning and degreasing agent. It is also an intermediate in organic synthesis. (Sittig, 1991)

Physical and Chemical Properties

Chemical Formula: CH_2 Cl CH_2 Cl

MW: 98.96	BP: 83-84°C
SG: 1.253 at 20°C	MP: -35.4°C
Sol. (water): 8 g/liter	VP: 61 mmHg at 20°C

Sol. (organics): miscible with alcohol, chloroform, and ether

Fate and Transport

Volatilization is the most significant means of escape of 1,1-dichloroethane from surface waters (ICF, 1985). The chemical is rapidly broken down by hydroxylation in the atmosphere. Some may be absorbed by atmospheric water and return to the earth by precipitation, however (ICF, 1985). Due to a low octanol/water partition coefficient (1.48) and reasonable solubility in water, leaching through soil into the ground water is an expected fate (ICF, 1985).

Pharmacokinetics

Specific data regarding the uptake and metabolism of 1,1-dichloroethane was not found in the literature reviewed. Data on the uptake of its isomer, 1,2-dichloroethane, was found, however. A similar uptake of the two isomers may be suspected.

Reitz et al. (1982) administered 150 mg ^{14}C-1,2-dichloroethane/kg bw in corn oil to rats. Recovery of radioactivity in exhaled air, urine and carcass at the end of 48 hours was virtually complete. Spreafico et al. (1978, 1979, 1980) found that peak blood levels occurred within 20 minutes and appeared to be linearly related to dose level when rats were exposed to 25, 50, or 150 mg 1,2-dichloroethane/kg bw in corn oil by gavage. Tissue levels, however were not linearly related. The authors concluded from this that passive transport across the GI tract occurred.

Urusova (1953) reported that women exposed to $\simeq$ 15.5 ppm 1,2-dichloroethane in air during a normal work day accumulated the chemical in breast milk. Immediately following exposure, exhaled air contained 14.5 ppm 1,2-dichloroethane indicating absorption through their lungs and the achievement of blood and total body equilibrium with inspired air within the daily work period. Reitz et al. (1980, 1982) found that equilibrium was reached in approximately 1 hour and was maintained at $\simeq$ 9 mg/liter when 4 Osborne-Mendel rats were exposed to 150 ppm 1,2-dichloroethane for 6 hours. Blood levels approached zero 1.5 hours after exposure was terminated.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Smyth (1956) reported that rats survived an 8-hour exposure to 4000 ppm but were killed by 16000 ppm. Lehmann and Plaa et al. (1965) reported no renal necrosis in mice exposed to 1000 mg/kg dose. Some tubular swelling occurred at this dose and at higher doses urinary protein (2000 mg/kg) and urinary glucose (4000 mg/kg) increased. Hoffman et al. (1971) exposed rats, guinea pigs, rabbits, and cats to 500 ppm concentration of 1,1-dichloroethane to 6 hours/day, 5 days/week for 13 weeks. No adverse effects were observed. After an additional 13 weeks however, the cats exhibited evidence of kidney injury histologically and by increased blood urea.

Teratogenic and Other Development Effects

Schwetz et al. (1974) exposed pregnant female rats on days 6-15 of gestation to 3800-6000 ppm 1,1-dichloroethane vapors for 7 hour/day. No adverse effects were observed in the dams or the fetuses except for slight, although statistically significant, decreases in food consumption and weight gain by the dams and delayed ossification in the fetuses. No teratological effects were related to exposures.

Mutagenic Effects

Riccio et al. (1983) and Mitoma et al. (1984) reported positive results in *S. typhimurium* strains TA1535, TA98, and TA100 when tested by plate incorporation in a desiccator in the presence and absence of metabolic activation systems. Strain TA1537, however, yielded negative results in this test. Similarly, Tu et al. (1985) and A.D. Little, Inc. (1983) reported negative results for 1,1-dichloroethane in a cell transformation assay with BALB/C-3T3 cells tested in the absence of an exogenous metabolic activation system in a sealed glass incubation chamber. When tested in a similar manner, however, 1,1-dichloroethane produced positive results in a DNA repair assay with hepatocyte primary cultures from rats or mice (Williams, 1977).

Carcinogenic Effects

Limited evidence of carcinogenicity was revealed in an NCI (1978a) bioassay. In this study Osborne-Mendel rats and B6C3F1 mice were administered technical grade 1,1-dichloroethane in corn oil by gavage, 5 days/week for 78 and 70 weeks, respectively.

The dosing was incontinuous (3 weeks on, 1 week off) due to toxicity resulting in doses of 382 (low) and 764 (high) mg/kg/day for male rats, 475 (low) and 950 (high) for female rats, 1442 (low) and 2885 (high) mg/kg/day for male mice, and 1665 (low) and 3331 (high) mg/kg/day for female mice. Unexposed and vehicle-control groups were monitored as well. Female rats exhibited a statistically significant dose-related increase in the incidence of hemangiosarcomas and those females surviving 52 weeks showed a significant increase in the incidence of mammary gland adenocarcinomas. Male rats exhibited no significant incidences of carcinomas. Female mice exhibited liver carcinomas in the vehicle-control and low-dose groups, an increase in benign uterine endometrial stromal polyps in the high-dose group. A dose-related increase in the incidence of hepatocellular carcinomas was observed in male mice.

In another NCI (1978b) study, the isomer of 1,1-dichloroethane, 1,2-dichloroethane, produced an increase in the incidence of forestomach squamos cell carcinomas and hemangiosarcomas in male rats and an increase in the incidence of mammary adenocarcinomas in female rats and mice. Additionally, mice of both sexes exhibited alveolar and bronchiolar adenomas, females exhibited endometrial stromal polyps and sarcomas, and males exhibited hepatocellular carcinomas.

Klaunig et al. (1986) concluded that 1,1-dichloroethane was not carcinogenic based on a 56-week study in B6C3F1 mice. The mice were exposed to 0, 835, or 2500 mg/liter in drinking water following a 4-week exposure to 10 mg/liter diethyl nitrosamine (DENA-initiated groups) or deionized water (uninitiated groups). Mice were sacrificed at 24 and 52 weeks and no adverse effects were observed. IRIS, however, has questioned the adequacy of the duration of the study.

Chlorinated ethanes and ethylenes were investigated by Milman et al. (1988) and Story et al. (1986) to detect their potential tumor initiating or promoting effects in a liver foci assay in Osborne-Mendel rats. 1,1-dichloroethane did not show any signs of initiation or complete carcinogenicity in the absence of initiation or promotion. It did exhibit promotional effects with DENA as initiator. However, the assumption that the liver foci seen in this assay are precancerous has not been validated (IRIS).

Ecotoxicity

The available freshwater data for chlorinated ethanes indicate that toxicity increases greatly with increasing chlorination (AWQC, 1986). ICF (1985) reports that 1,1,1-trichloroethane is less active than the 1,1,2-isomer. Based on this, 1,1-dichloroethane is probably no more toxic than the 1,2-isomer. 1,2-dichloroethane is acutely toxic at 100-500 mg/liter concentrations and chronically toxic at 20 mg/liter concentration (ICF, 1985). In saltwater systems, 1,2-dichloroethane was acutely toxic at 113 mg/liter (AWQC, 1986).

No data regarding the toxicity of 1,1-dichloroethane to terrestrial life, wild or domestic, was found in the literature reviewed.

Standards, Criteria, and Guidelines

EPA Class C Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	1.0 x 10^{-1} mg/kg/day
Chronic Inhalation RfD:	1.0 x 10^{-1} mg/kg/day
Subchronic Oral RfD:	1.0 x 10^{0} mg/kg/day
Subchronic Inhalation RfD:	1.0 x 10^{0} mg/kg/day
MCL:	NA
AWQC:	NA

BIBLIOGRAPHY

Arthur D. Little, Inc., *Cell transformation assays of 11 chlorinated hydrocarbon analogs.* Microfiche No. 0750509392, Doc. No. 40-8324457. 1983. (cited in IRIS).

Clayton, G.D. and F.E. Clayton Ed., *Patty's Industrial Hygiene and Past 3rd rev. ed.* 1981.

Hofman, H.T. et al., on the inhalation toxicity of 1,1- and 1,2-dichloroethane *Arch. Toxicol* 27:248 (ber.) 1971 (cited in HEA, 1984).

ICF Clements Association, *Physical, Chemical, and Biological Properties of Chemicals Present at Hazardous Waste Sites,* 1985.

Klaunig, J.E. et al., Carcinogenicity of chlorinated methane and ethane compounds administered in drinking water to mice. *Environ. Health Perspect.* 69:89-95. 1986 (cited in IRIS).

Milma, H.A. et al., Rat liver foci and vitro assays to detect initiating and promoting effects of chlorinated ethanes and ethylenes. *Ann. NY Acacl. Sci.* 534:521-530. 1988 (cited in IRIS).

Mitoma, C. et al., *Investigations of the species sensitivities and mechanism of carcinogenicity of halogenated hydrocarbons.* Microfiche No. OTS0509408, Doc. No. 40-8244225. 1984 (cited in IRIS).

NCI (National Cancer Institute), Bioassay of 1,1-dichloroethane for possible carcinogenicity. *NCI/NTP Technical Report No. 066.* SHEW Publ. No. (NIH) 78-136, Washington, DC 1978a (cited in IRIS).

NCI, Bioassay of 1,2-dichloroethane for possible carcinogenicity. *NCI Carcinogenesis Technical Report Series No. 55.* DHEW Publ. No. (NIH) 78-1361, Washington, DC 1978b (cited in IRIS).

Plaa, G.L. and R.E. Larson, *Toxicol. Appl. Pharmacol.* 7:37. 1965 (cited in Pattys, 1981).

Reitz, R.H. et al., Ethylene dichloride: A potential health risk? *In: Banbury Report No. 5* B. Ames, P. Infante, and R. Reitz, Eds. Cold Spring Harbor Laboratory, p. 135-148. 1980 (cited in HEA, 1984).

Reitz, R.H. et al., Pharmacokinetics and micromolecular interactions of ethylene dichloride in rats after inhalation or gavage. *Toxicol. Appl. Pharmacol.* 62:190-204. 1982 (cited in HEA, 1984).

Riccio, E. et al., A comparative mutagenicity study of volatile halogenated hydrocarbons using different metabolic activation systems. *Environ. Mutagen.* 5:472 (Abstract). 1983 (cited in IRIS).

Schwetz, B.A. et al., *Toxicol. Appl. Pharmacol.* 28:452 1974 (cited in Pattys, 1981).

Sittig, M., *Handbook of Toxic and Hazardous Chemicals,* 1991.

Smyth, H.F. Jr., *Am. Ind. Hyg. Assoc. Q.* 17:129. 1956 (cited in Pattys, 1981).

Spreafico, F. et al. Metabolism of 1,2-dichloroethane in experimental animals. *Report Nos. 1 and 2 to Chemical Manufactures Assoc.* New York, NY 1978 (cited in HEA, 1984).

Story, D.L. et al. Differences in rat liver enzyme-altered foci product by chlorinated aliphatics and phenobarbital. *Toxicol. Ind. Health.* 2(4):351-362. 1986 (cited in IRIS).

Tu, A.S. et al., Invitor transformation of BALB/C-3T3 cells by chlorinated ethanes and ethylenes. *Cancer Lett.* 28 (1): 85-92. 1985 (cited in IRIS).

Urusova, T.P. The possible presence of dichloroethane in human milk with exposure in industrial conditions. *Gig. Sonit.* 18:36-37. 1953 (cited in HEA, 1984).

U.S. EPA, *Drinking Water Regulations and Health Advisories.*

U.S. EPA, *Health Effects Assessment for 1,2-dichloroethane (HEA)* EPA-540/1-86/002 September 1984.

U.S. EPA, *Health Effects Assessment Summary Tables (HEAST).* FY1991.

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA, Office of Water Reg. and Standards, *Quality Criteria for Water (AWQC),* 440/5-86-001. May 1, 1986.

U.S. EPA, *Integrated Risk Information System (IRIS).*

ETHYLBENZENE

Use

Ethylbenzene is a volatile aromatic hydrocarbon that is colorless and highly flammable. It is used as an anti-knock agent in airplane fuels; a solvent; a constituent of asphalt and naphtha; and in the manufacture of styrene and acetophenone (EPA, 1985).

Chemical and Physical Properties

Chemical Formula: C_8H_{10}

MW: 106.2	BP: 136.2°C
SG: 0.867 at 20°C	MP: -95°C
FP: 17.2°C	VP: 7 mmhg at 20°C
Sol. (water): 161 mg/l at 25°C	
Sol. (organics): soluble in most organic solvents	

Fate and Transport

The transport of ethylbenzene in the environment is not well documented. The major route of elimination from surface water and soils is most likely volatilization. High quantities of organics in the soil would likely cause retention and adsorption of ethylbenzene.

In the atmosphere, ethylbenzene is photooxidized rapidly (EPA, 1985).

Pharmacokinetics

Ethylbenzene is absorbed through the lung, gastrointestinal tract, and skin into the bloodstream. Inhalation studies with ethylbenzene have shown that humans absorb approximately 64 percent of the inhalation dose. Absorbed ethylbenzene is distributed throughout the body but is concentrated in the kidneys, lung, adipose tissue, digestive tract, and liver. The primary metabolites of ethylbenzene formed in humans are mandelic acid and phenylglyoxylic acid, while 1-phenylethanol, benzoic acid, and mandelic acid are the major metabolites formed in rodents. The inhaled ethylbenzene dose is almost completely excreted by humans within 24 hours after exposure is ceased (EPA, 1985).

When ethylbenzene is coadministered with xylenes, xylenes are preferentially metabolized, causing ethylbenzene metabolism to be delayed (EPA, 1985).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Acute dermal exposure (17.8 ml/kg or 15,400 mg/kg) has been demonstrated to produce death in humans. Death occurred in rats after acute exposure to 4,000 ppm (17,400 mg/cu meter) via inhalation. The target organs of acute exposure are the central nervous system and lungs, however, toxic effects have also been observed in the liver and kidneys, (EPA, 1984). A concentration of 100 ppm (435 mg/m^3) did not produce adverse health effects in humans following an 8-hour inhalation exposure. Higher concentrations (values not specified) produced sleepiness, fatigue, headache, and mild eye and respiratory irritation (EPA, 1985).

Chronic oral exposure of ethylbenzene to rats resulted in liver and kidney changes. Increases in liver and kidney weights, cloudiness and swelling of the hepatocytes and renal tubular epithelium were produced by doses of 408 mg/kg/day. Doses of 13.6 mg/kg/day did not produce effects (EPA, 1985).

Ethylbenzene potentiates the toxicity of acrylonitrile (EPA, 1985).

Teratogenic and Other Developmental Effects

Embrytoxicity, fetotoxicity, and teratogenicity were not observed in rats or rabbits exposed to ethylbenzene via inhalation. Inhalation of 1,000 ppm of ethylbenzene elicited slight maternal toxicity in rats (EPA, 1985).

Mutagenic Effects

There is no evidence of mutagenic activity in *S. typhimurium* following ethylbenzene exposure in assays with and without metabolic activation at concentrations up to 3 mg/plate. No mutations were observed in yeast cells nor in rat liver epithelial cells exposed to ethylbenzene at 0.2 to 2,000 µg/plate. In *Drosophila melanogaster*, there was no increased frequency of recessive lethals (EPA, 1985).

Carcinogenic Effects

Due to the lack of animal and human studies, EPA has not classified ethylbenzene as a Class D carcinogen, not classifiable as to carcinogenicity.

Ecotoxicity

At concentrations greater than 23 mg/L, freshwater species experienced acutely toxic effects. No chronic effects were observed following exposure to 440 μg/L. A bioconcentration factor of 95 was calculated based on the log octanol/water partition coefficient (EPA, 1985). With regard to impacts on vegetation, no adverse effects on chlorophyll production by *Selenatrum capriconatum* or *Skeletonema costratum* was observed at concentrations as high as 438,00 μg/L (EPA, 1980).

Standards, Criteria and Guidelines

EPA Class D carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	1.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	2.86×10^{-1} mg/kg/day
Subchronic Oral RfD:	1.0×10^{0} mg/kg/day
Subchronic Inhalation RfD:	2.86×10^{-1} mg/kg/day
MCL:	0.7 mg/L
AWQC:	Water and Fish Consumption - 1.4 mg/L Fish Consumption - 3.3 mg/L

BIBLIOGRAPHY

Andrew, F.D. et al. Teratologic assessment of ethylbenzene and 2-ethoxyethanol. Battelle Pacific Northwest Laboratory, Richland, WA. PB83-208074,108, 1981, (Cited in IRIS).

U.S. EPA, *Ambient Water Quality Criteria for Ethylbenzene,* Office of Water Regulations and Standards, October, 1980.

U.S. EPA, *Drinking Water Criteria Document for Ethylbenzene.* Office of Drinking Water, March, 1985, final draft.

U.S. EPA, *Drinking Water Regulations and Health Advisories.*

U.S. EPA, *Integrated Risk Information System (IRIS).*

U.S. EPA, *Health Effects Assessment for Ethylbenzene,* EPA 540/1-86/008, Office of Emergency and Remedial Response, September, 1984, final draft.

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

Wolf, M.A. et al. Toxicological studies of certain alkylated benzenes and benzene. *Arch. Ind. Health* 14:387-398, 1956, (Cited in IRIS).

TOLUENE

Use

Toluene is a clear, colorless, organic compound with a benzene-like odor. It is highly flammable and extremely volatile. In industry, toluene is used in the production of benzene, as a solvent in paint thinners, and as additive to gasoline and other petroleum products. It is estimated that 100,000 workers in the United States are exposed to toluene annually (Sittig, 1991).

The majority of toluene releases to the environment occur from spills of gasoline and from improper disposal of toluene containing products. Every-day exposure to toluene occurs from gasoline and from the use of commercial paints and paint thinners.

Chemical and Physical Properties

Chemical Formula: $C_6H_5CH_3$

MW:	92	BP:	110.6°C
SG:	0.867 at 20°C	MP:	-95°C
FP:	4.4°C	VP:	28.7 mmhg at 25°C

Sol. (Water): 0.05%

Sol. (organics): acetone, carbon disulfide; miscible with alcohols, ether, benzene, chloroform, and glacial acetic acid.

Fate and Transport

Volatilization is the predominant route of removal of toluene from soils and aquatic environments. Toluene degrades rapidly in the air where it has a half life of 1.3 days (EPA, 1985c). It readily biodegrades in soils and surface waters. Toluene is transported easily in ground water, where it is known to remain stable. A 1988 EPA study found toluene present at 29 percent of hazardous waste sites surveyed. The average ground water concentration was 21 ppb.

Toluene occurs at low levels in drinking water, food and air. In urban settings, toluene is found in the air at levels of approximately 10 ppb. According to an EPA National Screening Survey, approximately 3 percent of all surface water derived drinking water systems are contaminated with toluene at levels higher than 0.1 µg/l (EPA, 1985b).

Pharmacokinetics

Studies on humans and animals have shown that toluene is absorbed quickly through the respiratory tract. In humans, inhalation exposures of 100 to 130 ppm for 4 hours resulted in a 40 to 60 percent uptake and retention of toluene. Absorption in the gastrointestinal tracts of male rats was relatively rapid with maximal blood-toluene levels being reached within 2 hours after gastric incubation (EPA, 1985); maximal blood levels following inhalation were reached in 15 to 30 minutes. Dermal absorption of aqueous toluene across human skin is related directly to concentration. Due to its lipophilic nature and low water solubility, toluene is expected to distribute to and accumulate in lipid tissue. A study with male rats revealed that toluene was distributed through the body with the greatest accumulation in lipid tissue. Toluene and its metabolites were also found in relatively high concentrations in tissues active in metabolism and excretion such as the liver and kidneys (EPA, 1985c). Toluene appears to be metabolized in humans and in animals through similar pathways (EPA, 1985). From inhalation studies, it is seen that side-chain hydroxylation to benzyl alcohol occurs. Benzyl alcohol is then conjugated with glycine to form hippuric acid, and is then excreted rapidly in the urine, generally within 12 hours of exposure. The half-life for toluene in adipose tissue of male humans exposed to 300 ppm toluene for 2 hours ranged from 0.5 to 2.7 days (ATSDR, 1989).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Human exposure to toluene is usually a result of inhalation of vapors in occupational settings. Intentional abuse and experimental inhalation also frequently occur (EPA, 1985b). Acute exposure to 200 ppm toluene for 8 hours caused fatigue, headache and nausea (EPA, 1985c). Subacute exposure resulted in similar but proportionately less severe symptoms. Studies of workers exposed to 200-500 ppm toluene occupationally for "many years" show that coordination, memory, and visual aptitude are all impaired. Cerebral dysfunction, such as tremors and ataxia, were also noted (EPA, 1985c). In a study done by Greenberg et al, it was shown that chronic exposure to toluene can also cause kidney dysfunction. A number of studies have reported chromosomal damage in the bone marrow (EPA, 1984). There is, however, no definitive evidence that toluene causes serious irreversible organ damage following chronic exposure (EPA 1985c).

Teratogenic and Other Developmental Effects

In one abstract, scientists reported an increase in fetal mortality in mice. In this study, gavage doses of 0.3, 0.5, and 1.0 ml/kg bw were administered daily on days 6-15 of the gestation period (EPA, 1985a).

Mutagenic Effects

Several studies cited by IRIS report no signs of mutagenic activity with toluene exposure. IRIS did cite a few Russian studies, however, that report toluene as effective in causing chromosomal damage in bone marrow cells of rats.

Carcinogenic Effects

There is no evidence that toluene is carcinogenic to humans.

No carcinogenic effects were seen in studies done on acute or chronic exposure of toluene to rats. These studies include topical, inhalation, and gavage exposures.

Ecotoxicity

In a 13 week gavage study on rats, animals that received 5,000 mg/kg-bw/day died during the first week (U.S. DHHS, 1990). Lower doses over the same period resulted in a reduction in body weight (16 percent) and an increase in organ size. The livers, lungs, hearts and kidneys were all seen to be greater in size and weight than those of the control rats.

Inhalation studies on rats showed that levels of 1200 ppm toluene over a period of 2 years resulted in an increase in the degeneration of olfactory and respiratory epithelial tissues. No weight changes or survival differences were noted (U.S. DHHS, 1990).

Five freshwater species of zooplankton displayed LC_{50} values for toluene of 12,700 to 313,000 μg/l. This is considered "practically nontoxic" (U.S. DHHS, 1990).

Standards, Criteria and Guidelines

EPA Class D carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	1.14×10^{-1} mg/kg/day
Subchronic Oral RfD:	2.0×10^{0} mg/kg/day
Subchronic Inhalation RfD:	5.72×10^{-1} mg/kg/day
MCL:	1.0 mg/L
AWQC:	Water and Fish Consumption - 14.3 mg/L Fish Consumption - 424 mg/L

BIBLIOGRAPHY

Agency for Toxic Substances and Disease Registry, (ATSDR), *Toxicological Profile for Toluene,* December 1989.

Anderson, I. et al. Human response to controlled levels of toluene in six-hour exposure. *Scand. J. Work Environ. Health* 9:405-418, 1983, (Cited in HEAST).

NIOSH, *Pocket Guide to Chemical Hazards,* 1987.

NTP (National Toxicology Program). Toxicology and carcinogenesis studies of toluene in F344 rats and B6C3F1 mice. Technical Report Series No. 371, 1989 (Cited in HEAST).

Sittig, Marshall, *Handbook of Toxic and Hazardous Chemicals*, 1991.

U.S. Dept. of Health and Human Services, *Toxicology and Carcinogenesis Studies of Toluene*, February 1990.

U.S. EPA, *Advisory Opinion for Toluene*, Office of Drinking Water (undated).

U.S. EPA 1985c, *Chemical Integration Methods for Toluene*, October 1985.

U.S. EPA, *Drinking Water Regulations and Health Advisories.*

U.S. EPA, 1985a, *Health Effects Assessment for Toluene*, EPA 540/1-86/033, 1985.

U.S. EPA, 1985b, *Health Advisory for Toluene*, Office of Drinking Water, 1985.

U.S. EPA, *Integrated Risk and Information System (IRIS).*

U.S. EPA, *Health Effects Assessment Summary Tables (HEAST).* FY1991.

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

1,1,1-TRICHLOROETHANE

Use

1,1,1-trichloroethane is a colorless, nonflammable liquid with an odor similar to chloroform. It is sometimes referred to as methyl chloroform.

Relatively recently, it has become widely substituted for carbon tetrachloride. It is used as a degreaser, and for dip-cleaning, bucket cleaning, and cold-cleaning of metals. 1,1,1-trichloroethane's solvent properties compel its use as a dry-cleaning agent, a vapor-degreasing agent and as a propellant (Sittig, 1991).

Physical and Chemical Properties

Chemical Formula: CH_3CCl_3
M.W.: 133.41
Sol. (water): insoluble
Sol. (organics): alcohol, ether, chloroform

BP: 74°C
MP: -30.4°C
VP: 123 mm Hg at 25°C

Fate and Transport

The half-life of 1,1,1-trichloroethane in water was reported by Callahan et al. (1979) to be 20-25 minutes. Volatilization to the atmosphere is the most likely route of escape. Singh et al. (1981) and Makide and Rowland (1981) reported the half-life of 1,1,1-trichloroethane in air to be 2.2 - 4.8 years. This suggests that the compound may move up into the stratosphere where it could contribute to ozone depletion.

Evaporation is expected to be the major fate of 1,1,1-trichloroethane from surface soil (Bouwer et al., 1981). Tabak et al. (1981) concluded that biodegradation of 1,1,1-trichloroethane is a slow process in subsurface soils. Coupled with low water solubility and a relatively low octanol/water partition coefficient, this suggests that the compound will remain substantially undegraded in subsurface soils creating the potential for leaching into ground water. In fact, Page (1981) detected the presence of this compound in ground water at a frequency of 78 percent.

Pharmacokinetics

It was determined by Stewart (1971) that 1,1,1-trichloroethane is "rapidly and completely" absorbed from the GI tract of humans and distributed preferentially and rapidly to the CNS. The U.S. EPA investigated the possibility of using 1,1,1-trichloroethane as an anesthetic and considered it to be more potent than trichloroethylene and safer than chloroform (U.S. EPA 1984).

Pulmonary absorption of inhaled 1,1,1-trichloroethane is initially rapid, but then slows dramatically until equilibrium is reached (U.S. EPA, 1984) Monster et al. (1979) and Humbert and Fernandez (1977) exposed volunteers to 70 or 140 ppm of 1,1,1-trichloroethane for four and eight hours. Equilibrium was reached in four hours and, at that time, Monster et al. (1979) reported a retention of 30 percent of the inhaled dose. This is 40 percent less than that reported by Humbert and Fernandez (1977). These data led the EPA to classify the compound as a poorly absorbed, partially soluble vapor (U.S. EPA, 1984).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Stewart et al. (1975) exposed 20 human subjects to 1,1,1-trichloroethane for three weeks, five days/week, 7.5 hours/day to a 500 pm concentration. The only effects detected were complaints of fatigue, irritation and headache by the subjects.

Similarly, Seki et al. (1975) found no dose-related effects in 196 male workers exposed to varying concentrations of 1,1,1-trichloroethane for greater than five years. Maroni et al. (1977), as well, found no signs of neurotoxicity when comparing 21 women exposed to 110-345 ppm 1,1,1-trichloroethane for 6.5 years to seven unexposed control subjects.

Torkelson et al. (1958), however, detected statistically significant increased liver weights in female guinea pigs exposed to 1,1,1-trichloroethane vapor at a concentration of 1,000 ppm for three hours/day, five days/week for three months. Females exposed to 500 ppm showed no adverse effects after exposure for seven hours/day, five days/week, for six months. Adams et al. (1950), as well, reported a slight depression in weight gain when guinea pigs were exposed to 1,1,1-trichloroethane at a concentration of 650 ppm for seven hours/day, five days/week for two to three months.

Teratogenic and Other Developmental Effects

In studies by Leong et al. (1975) and Schwetz et al. (1975), "no remarkable malformations were observed" in the fetuses of mice or rats exposed to 1,1,1-trichloroethane. A similar lack of adverse effect was reported when Charles River albino rats were exposed to 300 ppm 1,1,1-trichloroethane (U.S. EPA, 1984).

Mutagenic Effects

Both positive and negative results have been reported in the literature. Farber (1977) and Nestmann et al. (1980) found 1,1,1-trichloroethane to be mutagenic in *S. typhimurium* strain TA1535 and Simmon et al. (1977) produced positive results in *S. typhimurium* strain TA100.

Farber (1977) and Simmon et al. (1977) found no gene conversion of mitotic recombination in *Saccharomyces cerevisiae* upon exposure to 1,1,1-trichloroethane and the chemical also failed to produce chromosomal aberrations in the bone marrow of cats (Rampy et al., 1977).

Carcinogenic Effects

Quast et al. (1978) exposed 96 Sprague-Dawley rats of both sexes to 875 of 1,750 ppm vapor concentrations of 1,1,1-trichloroethane for six hours/day, five days/week for 12 months, followed by a 19-month observation period. There were no signs of carcinogenicity other than a significant increased incidence of focal hepatocellular alterations in female rats at the highest dosage.

Neither Quost et al. (1978) nor NCI (1977) found significant dose-related incidences of neoplasms. In the NCI study, Osborne-Mendel rats and B6C3F1 hybrid mice were treated with 750 or 1,500 mg/kg and 2,807 or 5,615 mg/kg of 1,1,1-trichloroethane, respectively, five times/week for 78 weeks. Although a variety of neoplasms were observed in the treated animals, they were not dose-related, nor were they statistically different from the occurrence of neoplasms in untreated animals.

Ecotoxicity

The available data on the toxicity of chlorinated ethanes in freshwater systems indicate that acute toxicity for trichloroethanes occurs at concentrations as low as 18,000 µg/liter. chronic toxicity occurs at concentrations as low as 9,400 µg/liter for 1,1,2-trichloroethane, a similar chlorinated ethane. In saltwater systems, acute toxicity to fish and invertebrate species occurs at 31,200 µg/liter for 1,1,1-trichloroethane. Both acute and chronic toxicity values are expected to be lower for species more sensitive to chlorinated ethanes than those tested (U.S. EPA, 1986).

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	9×10^{-2} mg/kg/day
Chronic Inhalation RfD:	3.0×10^{-1} mg/kg/day
Subchronic Oral RfD:	9.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	3.0×10^{0} mg/kg/day
MCL:	0.2 mg/l
AWQC:	Water and Fish Consumption - 18.4 mg/l Fish Consumption - 1030 mg/l

BIBLIOGRAPHY

Adams, E.M. et al., Vapor toxicity of 1,1,1-trichloroethane (methyl chloroform) determined by experiments on laboratory animals *Arch. Ind. Hyg. Occup. Med.* 1:225 1950 (cited in IRIS).

Bouwer, E.J. et al., Trace organic behavior in soil columns during rapid infiltration of secondary wastewater. *Water Res.* 15:151-159 1981 (cited in HEA, 1984).

Callahan, M.A. et al., *Water-related environmental fate of 129 priority pollutants, Vol. II* OWPS, OWWM U.S. EPA Washington, D.C. EPA-440/4-79-029-b 1979 (cited in HEA, 1984).

Farber, H. Manager of Environmental Affairs, Dow Chemical letter to James Price, Chief of Air Quality Data Analysis, Texas Air Control Board, Austin, TX. NCI, 1977 (cited in HEA, 1984).

Humbert, B.E. and J.G. Fernandez, Exposure to 1,1,1-trichloroethane: Contribution to the Study of absorption, excretion and metabolism in human subjects. *Arch. Mal. Prof.* 38:415-425 1977 (cited in HEA, 1984).

Leong, B.K.J. et al., Embryo - and fetotoxicity of inhaled trichloroethylene, perchloroethylene, methylchloroform, and methylene chloride in mice and rats. *Toxicol. Appl. Pharmacol* 33(1): 136 (Abstr. 34) 1975 (cited in HEA, 1984).

Makide, Y. and F. S. Rowland, Trospheric concentrations of methyl chloroform, CH_3CCl_3, in January 1978 and estimates of the atmospheric residence times of halocarbons *Proc. NAS* 78:5933-5937 1981 (cited in HEA, 1984).

Maroni, M. et al., A clinical, neurophysical and behavioral study of female workers exposed to 1,1,1-trichloroethane. *Scand. J. Work. Environ. Health* 3:16-22 1977 (cited in HEA, 1984).

Monster, A.C., et al., Kinetics of 1,1,1-trichloroethane in volunteers, influence of exposure concentrations and work load. *Int. Arch. Occup. Environ. Health* 42:293-301 1979 (cited in HEA, 1984).

NCI (National Cancer Institute), Bioassay of 1,1,1-trichloroethane for possible carcinogenicity. *Carcinog. Tech. Rep.* Ser. No. 3 NCI-CG-TR-3 p. 70, 1977 (cited in IRIS).

Nestman, E.R. et al., Mutagenicity of constituents identified in pulp and paper mill effluents using the Salmonella/mammalian microsome assay. *Mutat. Res.* 79:203-212 1980 (cited in HEA, 1984).

Page, G.W., Comparison of groundwater and surface water for patterns and levels of contamination by toxic substances. *Environ. Sci. Technol.* 15:1475-1481 1981 (cited in HEA, 1984)

Quast, J.F. et al., *Toxicologic and carcinogenic evaluation of a methylchloroform (1,1,1-trichloroethane) formulation by chronic inhalation in rats - interim reports at 24 months.* Dow Chemical Co., Midland, MI. p. 14, 1978, (cited in IRIS).

Rampy, L.W. et al., Results of long-term inhalation toxicity studies on rats of 1,1,1-trichloroethane and perchloroethylene formulations. In: *Proc. Int. Cong. Toxicol.* Toronto p. 27 1977 (cited in HEA, 1984).

Schwetz, B.A., et al., The effect of maternally inhaled trichloroethylene, perchloroethylene, methylchloroform, and methylene chloride on embryonal and fetal development in mice and rats. *Toxicol. Appl. Pharmacol.* 32:84-86 1975 (cited in HEA, 1984).

Seki, Y. et al., Trichloro-compounds in the urine of humans exposed to methyl chloroform at sub-threshold levels. *Int. Arch. Arbeitsmed.* 34: 39-49 1975 (cited in HEA, 1984).

Simmon V.F. et al. Mutagenic activity of chemicals identified in drinking water. *In-Progress in Genetic Toxicology.* D. Scott et al., Ed. Elsevier/North Holland Biomedical Press, Amsterdam 1977 (cited in HEA, 1984).

Singh, H.B. et al., Measurements of some potentially hazardous organic chemicals in urban environments. *Atmos. Environ.* 15:601-612 1981 (cited in HEA, 1984).

Sittig, M., *Handbook of Toxic and Hazardous Chemicals,* 1991.

Stewart, R.D., Methyl chloroform intoxication: Diagnosis and treatment. *J. Am. Med. Assoc.* 1789 - 1792 1971 (cited in HEA, 1984).

Stewart, R.D. et al., *1,1,1-trichloroethane: Development of a biological standard for the industrial worker by breath analysis.* Medical College of Wisconsin and National Institute of Occup. Safety and Health NIOSH- MCOW-ENVM-1,1,1-T-75-4, 1975 (cited in HEA, 1984).

Tabak, H.H., et al., Biodegradability studies with organic priority pollutant compounds. *J. Wales Pollut. Control. Fed.* 53:1503-1518 1981 (cited in HEA, 1984).

Torkelson, T.R. et al., Toxicity of 1,1,1-trichloroethane as determined on laboratory animals and human subjects. *Am. Ind. Hyg. Assoc. J.* 19:353-362 1958 (cited in HEA, 1984).

U.S. EPA, *Drinking Water Regulations and Health Advisories.*

U.S. EPA, *Health Effects Assessment for 1,1,1-trichloroethane (HEA).* EPA 540/1-86-005, 1984.

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA, Office of Water Regs. and Standards *Quality Criteria for Water* 440/5-86-001, May 1, 1986.

U.S. EPA, *Health Effects Assessment Summary Tables (HEAST).* FY1991.

U.S. EPA, *Integrated Risk Information System (IRIS).*

XYLENES

Use

Xylenes refer to a mixture of orth-, meta-, and para-xylenes. Xylenes are components of petroleum and gasoline products; are used in iron and steel manufacturing, foundries, pulp and paper mills; and are used as solvents for paints, inks, and adhesives (ICF, 1985; EPA, 1987). Xylenes occur naturally as a constituent of petroleum oil, and are produced in large volumes during gasoline refinement. The release of xylenes to the environment occurs mostly to air as a result of volatilization. Releases of xylenes to water and soil are primarily attributed to spills and leaks of gasoline and other petroleum products, with lesser releases due to disposal of waste paints, inks, and other industrial products (EPA, 1987).

Chemical and Physical Properties

Chemical Formula: $C_6H_4(CH_3)_2$

MW: 106.17
SG: 0.860 at 25°C
FP: 25°C
Sol. (water): 160 mg/l at 25°C
Sol. (organics): alcohol, ether, and numerous other organic solvents

BP: 137 - 140°C (mixed)
MP: meta - 48°C
ortho - 25°C
para - 13°C
VP: 10 mmHg at 25°C

Fate and Transport

Volatilization is the most important means by which xylene is removed from soils and surface water. Xylene is adsorbed by organics in moist soils and transport to ground water is unlikely (EPA, 1985).

In the atmosphere, xylene is photohydroxylated to produce carbon dioxide, cresol, and peroxyacetylnitrate.

Pharmacokinetics

Inhalation of mixed xylenes by humans showed that xylenes are absorbed readily to an extent of 64 percent. Animal studies indicate that xylenes are rapidly distributed to the brain and adipose tissue and reach maximum tissue levels within one hour after inhalation; xylenes are also distributed to the kidneys, subcutaneous fat, sciatic nerve, blood, liver, lungs, spleen, and muscles. Metabolism of xylenes is generally accomplished by oxidation of methyl groups and ring hydroxylation, thus producing methyl hippuric acid (95 percent) and xylenols (1-2 percent) as metabolites (EPA, 1985).

Synergistic effects result from metabolic interactions when xylenes are coadministered with other chemicals. Ethanol potentiates the effect of xylenes by delaying its metabolism and elevating blood xylene levels (EPA, 1985). Depressed metabolism was also observed when xylene were coadministered with any of the following compounds: 1,1,1-trichloroethane (EPA, 1984), benzene, ethylbenzene, or toluene (EPA, 1985). Xylenes induce enzyme activity and thus potentiate the hepatoxicity of carbon tetrachloride by elevating the levels of toxic metabolites (EPA, 1985).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The lowest oral dose to cause death in humans was reported at 50 mg/kg. Xylenes produce central nervous system disturbances in humans that include alterations in numerative ability, short-term memory, and electroencephalographic patterns. No adverse effects were observed in males following inhalation of xylene for 70 minutes at concentrations of 435 and 1,300 mg/m^3. However, inhalation of xylene at 1,300 mg/m^3 after 30 minutes of strenuous exercise caused decreased mental abilities. Psychophysiological functions were impaired in humans following exposure to xylene at 391 mg/m^3 for five consecutive days. The effects included depression of manual coordination and impairment of body balance (EPA, 1985).

NTP (1986) studies of rats and mice administered gavage doses of 0, 250, or 500 mg/kg/day and 0, 500, or 1000 mg/kg/day for five days/week for 103 weeks revealed the following: increased dose-related mortality observed in rats; and hyperactivity in mice given the high dose.

Chronic inhalation of 770, 2,200 and 3,500 mg/m^3 of mixed xylenes by rodents for six days/week or five days/weeks for 13 weeks did not produce any effects at the lower two doses. The higher dose produced renal tubular degeneration. Inhalation of 337 and 3,358 mg/m^3 of o-xylene for thirty and ninety days continuously by rats, guinea pigs, monkeys, and dogs did not produce significant effects with respect to body weight, hematology, and histopathology. The only observed effect was tremors produced in dogs. Oral exposure to o-xylene at 200 mg/kg diet for six months produced hepatotoxicity in rats. Inhalation of 4,750 mg/m^3 of xylene for eight hours/day, seven days/week for one year produced hepatotoxicity in rats (EPA, 1985).

Teratogenic and Other Developmental Effects

Increased incidence of fused sternebrae and extra ribs were observed in rats inhaling 1,000 mg/m^3 of mixed xylenes for 24 hours/day during days 9 to 14 of pregnancy. No maternotoxic effects were observed in the rats. In a study which exposed pregnant rats to 0, 434, and 1,730 mg/m^3 of xylenes during days 6 to 15 of pregnancy, no teratogenic effects were observed (EPA, 1985). Maternotoxicity appears to occur in mice exposed to xylenes (EPA, 1985).

Mutagenic Effects

Short-term *in vitro* assays and the Ames test indicate that xylenes are not mutagenic (EPA, 1985).

Carcinogenic Effects

Xylene is designated by EPA as a Group D -- not classified Weight-of-Evidence category for potential carcinogens (EPA, 1985). Several studies including one by NTP (1986) report no increase in the incidence of cancer in laboratory animals exposed to xylenes.

Ecotoxicity

Xylenes adversely affect trout at concentrations as low as 3.6 mg/L and have a LC_{50} value of 13.5 mg/L. LC_{50} values for other fresh water fish average 13.5 mg/L. Information regarding the toxicity of xylenes to terrestrial animals was not available (EPA, 1980).

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2.0×10^0 mg/kg/day (mixed xylenes)
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	4.0×10^0 mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	10 mg/l
AWQC:	NA

BIBLIOGRAPHY

Clayton and Clayton, *Patty's Industrial Hygiene and Toxicology*. Third Edition, 1981.

Hake, C.L. et al. Development of a biologic standard for the industrial worker by breath analysis. NIOSH-MCOW-ENUM-XY-77-3. NTIS PB82-152844, 1981, (Cited in HEAST).

ICF, Clement Associates, *Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites,* 1985.

National Toxicology Program (NTP), *NTP Technical Report on the Toxicology and Carcinogenesis of Xylenes (mixed) (60.2% m-xylene, 13.6% p-xylene, 17.0% ethylbenzene, and 9.1% s-xylene) (cas no. 1330-20-7) in F344/N rats and B6C3F1 mice (gavage studies).* U.S. DHHS, NIH, NTP, Research Triangle Park, NC NTP TR 327, NIH Publ. No. 86-2583. 1986 (cited in IRIS).

U.S. EPA, *Ambient Water Quality Criteria for Xylene,* Office of Water Regulations and Standards, October 1980.

U.S. EPA, *Drinking Water Criteria Document for Xylene.* Final Draft. Office of Drinking Water, March 1985.

U.S. EPA, *Drinking Water Regulations and Health Advisories.*

U.S. EPA, *Health Effects Assessment for Xylene*, Office of Emergency and Remedial Response, September 1989.

U.S. EPA, *Health Effects Assessment Summary Tables (HEAST).* FY1991.

U.S. EPA, *Integrated Risk Information System (IRIS).*

U.S. Geological Survey, *Physical and Chemical Properties and Health Effects of Thirty-Three Organic Chemicals,* U.S. Department of the Interior, August 1987.

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

SEMI-VOLATILES

BENZOIC ACID

Use

Benzoic acid is used as a food preservative, in pharmaceutical and cosmetics preparation, in the manufacture of resins and as an intermediate in the synthesis of other industrial chemicals (Verscheuren, 1977).

Chemical and Physical Properties

Chemical Formula: C_6H_5COOH

MW:	122.1	MP:	121.7°C
SG:	1.27	BP:	249°C
Sol. (water):	2,900 mg/L	VD:	4.21

Fate and Transport

Benzoic acid is known to biodegrade in soils within 1 day of release (Verscheuren, 1977). No other information regarding the fate and transport of benzoic acid was located in the available literature.

Pharmacokinetics

Benzoic acid is absorbed rapidly in the stomach and gastrointestinal tracts of humans (IRIS). No other information regarding the fate and transport of benzoic acid was located in the available literature.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The Food and Drug Administration (FDA) (1983) reports that daily per capita intakes of 0.9-34 mg of benzoic acid have resulted in no toxic effects on humans. The FDA has given benzoic acid the status of Generally Recognized As Safe (GRAS).

Chronic oral doses of 40 mg benzoic acid/kg/day administered to mice and rats for 17 months resulted in a decreased resistance to stress and a possible reduction in food and water intake (Shtenberg and Ignat'ev, 1970). Another study resulted in the same effects in rats exposed orally to 750 mg benzoic acid/kg/day for a lifetime (IRIS).

Levels between 0.9 and 34 mg benzoic acid do not appear to induce toxic effects in humans when exposed via ingestion (IRIS).

Gerlach (1909) reported no visible ill effects in humans ingesting benzoic acid at 0.5 - 1.0 g/day for 44 days or 82 of 86 days. Wiley and Bigelow (1908), however, noted irritation and discomfort in humans given oral doses less than 1.75 g/day.

Teratogenic and Other Developmental Effects

No information regarding the teratogenic or developmental effects of benzoic acid were located in the available material.

Mutagenic Effects

Benzoic acid has been tested for mutagenicity and genotoxicity in prokaryotes, eukaryotes and mammalian cell cultures. Reports have all been negative (IRIS).

Carcinogenic Effects

Toth (1984) administered 3502 and 3367 mg benzoic acid/kg/day to male and female Swiss mice, respectively. After a lifetime, examinations revealed no toxic or carcinogenic effects.

Shtenberg and Ignat'ev (1970) reported an 8 percent incidence of malignant tumors in mice exposed to 40 mg benzoic acid/kg/day for 17 months. Tumor incidences of the controls were not reported. In a separate study, Dinerman and Ignat'ev (1966) reported that mice exposed to 0.2 percent benzoic acid for 3 months displayed increased susceptibility to carcinomas induced by inoculations with Erlich ascites carcinoma cells. The incidence of tumors more than doubled when benzoic acid was co-administered.

Ecotoxicity

Benzoic acids LD_{50} for rats was found to be 1.7 g/kg. *Daphnia magna* are immobilized by 146 mg benzoic acid/L water when exposed for a prolonged period of time (Verscheuren, 1977).

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor: NA
Inhalation Slope Factor: NA
Chronic Oral RfD: 4 x 10^0 mg/kg/day
Chronic Inhalation RfD: NA
Subchronic Oral RfD: 4 x 10^0 mg/kg/day
Subchronic Inhalation RfD: NA
MCL: NA
AWQC: NA

BIBLIOGRAPHY

Dinerman, A.A. and A.D. Ignat'ev, Effect of Certain Food Preservatives on the Development of Tumors in Mice, 1966, *Gig. Sanit.*, 31(9): 38-42 (Eng. Trans.), (Cited in IRIS).

Food and Drug Administration (FDA), *Evaluation of the Health Aspects of Benzoic Acid and Sodium Benzoate as Food Ingredients.* DHEW, Wash. D.C., Report No. SC065-7. NTIS PB-223837/6. 1973 (cited in IRIS)

Gerlach, V. VII, Summary of the Results, In: Physiological Activity of Benzoic Acid and Sodium Benzoate, V. Gerlach, Ed., *Verlag von Heinrich Staadt*, Weisbaden, 1909, page 90-92, (Cited in IRIS).

Shtenberg, A.J. and A.D. Ignat'ev, Toxicological Evaluation of Some Combination of Food Preservatives, 1970, *Food Cosmet. Toxicol.*, 8(4) 309-380, (Cited in IRIS).

Toth, B., Lack of Tumorigenicity of Sodium Benzoate in Mice, 1984; *Fund. Appl. Toxicol.*, 4(3): 494-496, (Cited in IRIS).

U.S. EPA, *Drinking Water Regulations and Health Advisories.*

U.S. EPA, *Integrated Risk Information System* (IRIS).

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

Verscheuren, Karel, *Handbook of Environmental Data on Organic Chemicals*, 1977.

Wiley, H.M. and W.D. Bigelow, Influence of Benzoic Acid and Benzoates on Digestion and Health, 1908, *Bulletin 84 pt IV, Bureau of Chemistry*, U.S. Department of Agriculture, (Cited in IRIS).

POLYCYCLIC AROMATIC HYDROCARBONS

Background

Polycyclic Aromatic Hydrocarbons (PAHs) constitute a class of materials which are characterized as containing more than one benzene ring. Because of the similarities between them, PAHs have been summarized in one toxicity profile. Discussions of specific PAHs are provided in cases where available literature exists. Otherwise, PAHs are discussed in general terms. The most common and the most hazardous PAH is benzo (a) pyrene (BaP). As a result, most of the information located in the available literature deals with BaP.

Use

As a class, PAHs are used industrially in the production of automobile tires, rubber stoppers, dyes, and glass and can be found in yeasts, whiskeys, dried prunes, and cigarette smoke (ICF, 1985). PAHs are often found as byproducts to the refining processes of petroleum, shale, coal, and coke.

Chemical and Physical Properties

Summarized in Table 1.

Fate and Transport

In general, PAHs are expected to exist as vapor and particulates in the atmosphere. Once in the atmosphere, PAHs may be removed through photochemical reactions, chemical reactions, or by wet and dry deposition. In aquatic media, PAHs are expected to volatilize, react photochemically, and be degraded microbially. In high water and wind flow conditions, volatilization would occur readily. In water, PAHs would adsorb to organic matter and would most likely fall out of the water column (EPA, 1984a).

In soils, PAHs are subject to microbial degradation and adsorption. Because of their affinity to organic matter, PAHs are not expected to be highly mobile in soils, therefore, leaching to ground water is not considered to be a significant fate process.

Pharmacokinetics

Although few studies have been performed on human ingestion of PAHs, it is thought that they would be absorbed readily in the gastrointestinal tract. Benzo (a) pyrene (BaP), chrysene, and benzo (a) anthracene (BaA), three of the more common PAHs, are reported to transport passively across the gastrointestinal mucosa (EPA, 1984a). Chang (1943) noted that rats given BaP by gavage absorbed approximately 50 percent of the administered dose. Certain PAHs require metabolic activation by specific enzymatic systems in order to acquire carcinogenic properties. PAHs and their metabolites are excreted through the feces and through the hepatobiliary system. There is little evidence that PAHs bioaccumulate extensively (EPA, 1984a).

TABLE 1. CHEMICAL AND PHYSICAL PROPERTIES OF PAHs (U.S. EPA, 1986)

Compound	Chemical Formula	MW	SP. GR. (at 20°C)	BP (°C)	MP (°C)	VP (mmHg at 20°C)	Solubility (at 25°C)
Acenapthene	$C_{12}H_{10}$	154.21	1.225 at 0°C	279	96.2	$1.55x10^{-3}$	water: 3.42 mg/l organics: ethanol, toluene, chloroform, benzene
Acenaphthylene	$C_{12}H_8$	152.21			92.0*	$1x\ 10^{-3}$	water: 3.93 mg/1
Anthracene	$C_{14}H_{10}$	178.2	1.25	342	218	$1.95x10^{-4}$	water: 0.0446 mg/kg organics: benzene, chloroform, methanol
Benzo(a)anthracene	$C_{18}H_{12}$	228.30		435	167.0	2.2x10 at 20°C	water: 9.4 µg/kg organics: alcohol, ether, acetone, benzene,
Benzo(a)pyrene	$C_{20}H_{12}$	252.30		311	179.15	$5.6x10^{-9}$	water: 1.2 µg/kg organics: most
Benzo(b)fluoranthene	$C_{20}H_{12}$	252.3			168.3	$5.0x10^{-7}$	water: 0.014 mg/l organics: benzene, acetone
Benzo(k)fluoranthene	$C_{20}H_{12}$	252.3		480	215.7	$5.1x10^{-7}$	water: 0.0043 mg/l organics: acetic acid, benzene, ethanol
Benzo(g,h,i)perylene	$C_{22}H_{12}$	276.30			278.3	$1.03x10^{-10}$	water: 0.7 µg/kg organics: benzene, acetone
Dibenzo(a,h)anthracene	$C_{22}H_{14}$	278.4			266.6	$1.1x10^{-10}$	water: insoluble organics: benzene, toluene, xylene, oils

(Continued)

TABLE 1. (Continued)

Compound	Chemical Formula	MW	SP. GR. (at 20°C)	BP (°C)	MP (°C)	VP (mmHg at 20°C)	Solubility (at 25°C)
Chrysene	$C_{18}H12$	228.20	1.274	448	255.5	$6.3x10^{-9}$	water: 1.8 µg/kg organics: benzene, ether, alcohol
Fluoranthene	$C_{16}H_{10}$	202.24	1.252	250.5	111.1	$5.0x10^{-6}$	water: 0.206 mg/kg organics: acetic acid, benzene, chloroform, ethanol
Fluorene	$C_{13}H_{10}$	166.2		295	116.5	10 at 146°C	water: insoluble organics: most
Indeno(1,2,3-c,d)pyrene	$C_{22}H_{12}$	276.3			163.6	10^{-10} torr	water: insoluble organics:
2-Methylnapthalene	$C_{11}H_{10}$	142.20		241.05	34.58		water: insoluble organics: most
Naphthalene	$C_{10}H_8$	128.16	1.15	217.9	80.55	0.082	water: 31.7 mg/l organics:
Phenanthrene	$C_{14}H_{10}$	178.22	1.025	340.0	100	6.8x10	water: 1 mg/kg organics: ethanol, toluene, benzene
Pyrene	$C_{16}H_{10}$	202.24		385	149.5	$2.5x10^{-6}$	water: 0.132 mg/kg organics: benzene, diethyl ether, ethanol, toluene, acetone

Human Toxicity

Acenapthene

Knobloch, et al. (1969) reported that, when administered orally to rats, acenapthene causes changes in renal function, lowers body weight and causes unspecified changes in the peripheral vascular system (EPA, 1984f). Mild morphological damage to the kidneys and liver were also noted. U.S. EPA (1989a) reports liver weight changes accompanied by microscopic alterations in mid- to high-dose mice exposed to acenapthene ranging in concentration form 0 to 700 mg/kg/day. Nonspecific pneumonia was noted by Reshetyuk et al. (1970) in rats exposed to 12 mg/m^3 acenapthene by inhalation for four hours/day, six days/week for five months (EPA, 1984f).

Acenapthene has not been shown to be mutagenic; however, it is known to cause changes in the DNA content of a number of plant and microbial species. These changes are a result of disruptions of the spindle mechanisms during mitosis (ICF, 1985).

At high exposure levels, acenapthene is known to cause liver and kidney damage, but is not known to be carcinogenic (ICF, 1985).

Acenaphthylene

The U.S. EPA is currently reviewing the noncarcinogenic risk assessment of this substance (IRIS). No data relative to the toxicity of this chemical were found in the literature reviewed.

Kaden et al. (1979) found that acenaphthylene (1mM) yielded positive results in a *Salmonella typhimurium* forward mutation assay. However, Bos et al. (1988) reported negative results in *S. typhimurium* strains TA98 and TA100 in the presence of hepatic homogenates.

Cook (1932) observed no carcinogenic effect in a lifetime study of the effect of dermally introduced 0.25 percent acenaphthylene in mice. Survival, however, was only 65 percent at 6 months and 35 percent at 12 months.

Anthracene

Chronic exposure to anthracene is thought to cause dermatitis, hyperkeratoses, and other skin disorders in workers (ICF, 1985). Numerous studies of chronic and acute exposure of laboratory animals to anthracene have suggested that it does not cause any systemic toxic effects (i.e., U.S. EPA, 1989b).

Anthracene has been shown to cause reproductive effects in mice given a single oral dose during the last week of gestation. A lower survival rate was seen in experimental mice than in controls (IARC, 1983).

In twenty experiments on the mutagenicity of anthracene, very few have resulted in positive effects (IARC, 1983); therefore, not enough evidence is available to consider anthracene a mutagen. There are no epidemiologic studies available that suggest anthracene is carcinogenic to humans. In studies of the effects of subcutaneous injections of anthracene to laboratory animals, it was noted to cause local tumors. The carcinogenic effects of subcutaneous injections of anthracene appear to be enhanced by ultra-violet light. Schmahl (1955), however, found no incidence of tumors following exposure of rats to 4.5 g anthracene/rat over 78 weeks.

However, this evidence was determined to be inadequate in proving anthracene's carcinogenicity (IARC, 1983).

Benzo (a) Anthracene

Benzo (a) anthracene (BaA) is known to cause skin disorders, such as hyperplasia and hyperkeratosis, in workers exposed occupationally (ICF, 1985). Cutaneous exposure to BaA causes destruction of the sebaceous glands of laboratory mice and, when injected repeatedly, BaA produces gross changes in the lymphoid tissues of mice and rats (ICF, 1985).

It is also known that many carcinogenic PAHs, such as BaA, cause immunosuppressive effects, although studies on BaA have not been conclusive (ICF, 1985).

BaA is known to be mutagenic in *Salmonella typhimurium* and *Drosophila melanogaster*. It is also known to cause sister-chromatid-exchange in cultured mammalian cells.

Several studies indicate that BaA is carcinogenic to laboratory animals. Oral administrations and sub-cutaneous injections have resulted in statistically significant increases in tumors and adenomas (Klein, 1963; IARC, 1983).

Benzo (a) Pyrene

From laboratory studies performed on mice, it appears as though BaP's toxicity to organisms is dependent upon the constitution of a specific gene locus. The particular locus determines whether or not aryl-hydrocarbon-hydroxylase, an enzyme which alters the chemical makeup of aromatic hydrocarbons, is easily released (induced) into the body (EPA, 1985). Those animals that cannot easily induce the release of aryl hydrocarbon hydroxylase are more susceptible to BaP's toxic effects. Robinson, et al. (1975) administered 120 mg/kg-bw BaP in food to "poorly inducible" and "easily inducible" mice. The "poorly inducible" mice developed aplastic anemia and died within four weeks whereas the "easily inducible" mice remained healthy for at least six months.

In a study a carried out by Rigdon and Rennels (1964), only one of seven pregnant female rats carried viable fetuses to term, after having been fed a diet containing BaP at a level of 50 mg/kg/day for up to 3.5 months. Of four pups delivered, two were stillborn, one of which was grossly malformed. A third was killed for observational purposes, while the fourth died of starvation because it did not appear to be lactating.

In a teratogenicity and reproduction study, Rigdon and Neal (1965) fed diets containing BaP at a level of 0, 250, 500, or 1,000 mg/kg to male and female mice over various time spans during mating, gestation, and lactation. No apparent reproductive, teratogenic, embryotoxic or fetotoxic effects were observed.

MacKenzie and Angevine (1981) administered BaP orally at a level of 10 mg/kg/bw to CD-1 mice during pregnancy. There was no effect on fetal body weight; however, reduced fertility and reproductive capacity were observed in the offspring.
BaP has been used as a positive control in a variety of short-term tests. It has yielded positive results in assays for bacterial mutation, mutation in *Drosophila melanogaster*; DNA binding, DNA repair, sister chromatid exchange (SCE), chromosomal aberration, point mutation and transformation in mammalian cells in culture; and *in vivo*, including DNA binding, SCE, chromosomal aberration, sperm abnormality and the specific locus (spot) test (IARC, 1982; deSerres and Ashby, 1981; Hollstein and McCann, 1979).

PAH mixtures containing BaP have been shown to induce lung cancer in humans as a result of chronic exposure to cigarette smoke, roofing tar, and coke oven emissions (IRIS). It is impossible to conclude from these studies however, that BaP is the responsible agent.

Cottini and Mazzone (1939) applied a 1 percent solution of BaP to the skin of 26 patients. The skin of the patients developed regressive verrucae, reversible and apparently benign cysts that are thought to represent the early stages of neoplasia.

BaP is known to be carcinogenic to mice when exposed subcutaneously. Neal and Rigdon (1967) noted a dose-response relationship in the incidence of stomach tumors in male and female CFW-Swiss mice treated orally with 1-250 ppm BaP for 197 days. Individuals treated with greater than 20 ppm doses exhibited a significant increase in stomach carcinomas and papillomas. Mice treated with 250 ppm BaP exhibited an increase in the incidence of lung adenoma and leukemia.

In an inhalation study, Thyssen, et al. (1981) exposed hamsters to 2.2, 9.5 or 45 mg/m^3 BaP for 4.5 hours/day for 10 weeks and three hours/day seven days/week for up to 675 days. Animals exposed to 9.5 mg/m^3 developed tumors of the nasal cavity, larynx trachea and pharynx. Animals exposed to 45 mg/m^3 BaP developed a significant number of tumors in the respiratory tract and upper digestive tract.

Benzo (b) Fluoranthene

No data concerning the systemic effects of benzo (b) fluoranthene (BbF) on humans or laboratory animals were located in the available literature.

One study has demonstrated that BbF caused chromosomal aberrations in the bone-marrow cells of Chinese hamsters (IARC, 1983). In this study, hamsters were given two doses of 450 mg BbF/kg-bw. In separate studies, unspecified mutations in *Salmonella typhimurium* cultures were noted when exposed to 100 μg BbF (IARC, 1983).

BbF is known to be carcinogenic to laboratory mice and rats. three-month old female Osborne-Mendel rats exposed to BbF through lung implants illustrated a dose-related increase in the incidence of epidermoid carcinoma and pleomorphic sarcomas in the lung and thorax (Deutsch-Wenzel et al., 1983). A 0.5 percent solution of BbF produced papilloma in 100 percent of laboratory mice that were painted three times per week (IARC, 1983). In one study, researchers were able to induce local sarcoma in 18 of 24 mice that were subcutaneously injected with 0.6 mg BbF. The lowest carcinogenic dose of BbF painted on mice was noted to be a 0.01 percent solution (IARC, 1983).

No experiments concerning the carcinogenic effects of BbF on humans were located in the available literature.

Benzo (k) Fluoranthene

No data concerning the systemic effects of benzo (k) fluoranthene (BkF) on humans or laboratory animals were located in the available literature. BkF was reported to be mutagenic in bacteria such as *Salmonella typhimurium* (IARC, 1983).

The International Agency for Research on Cancer (IARC) has determined that there is sufficient evidence to prove that BkF is carcinogenic to laboratory animals. Tumors were noted in 69 percent of NMRI mice treated with 9.2 mg BkF/kg-bw. In this study, 3.4, 5.6 or 9.2 mg BkF were applied to the mice's skin. In the lowest test group, 8 of 34 individuals exhibited local tumors (IARC, 1983).

When injected into the pulmonary tissues of rats, BkF caused squamous cell carcinomas (IARC, 1983). Female Osborne-Mendel rats exposed to BkF through lung implants illustrated a dose-related increase in the incidence of epidermoid carcinomas in the lung and thorax (Deutsch-Wenzel, et. al., 1983).

Benzo (g,h,i) Perylene

No data concerning the systemic effects of benzo (g,h,i) perylene (B(g,h,i)P) on humans or laboratory animals were located in the available literature. IARC states that there is inadequate evidence to prove that B(g,h,i)P is toxic when exposure is short-term.

B(g,h,i)P was shown to be mutagenic to *Salmonella typhimurium* when administered in various doses (IARC, 1983).

In seven studies evaluated by IARC, B(g,h,i)P caused no visible carcinogenic effects. The tests included five skin application assays, one intrapulmonary injection study, and one co-administration study. In the latter study, B(g,h,i)P was administered with BaP. A higher number of skin tumors was noted in the test group than in the group administered BaP alone (IARC, 1983).

There is not sufficient evidence to classify B(g,h,i)P as carcinogenic to humans or laboratory animals (IARC, 1983).

Chrysene

Chrysene's toxic effects to humans and animals have not been studied extensively. It is expected that chrysene causes damage to epidermal tissues in workers exposed daily. Although not specific to chrysene, numerous studies indicate that PAHs cause immunorepressive effects (IARC, 1983).

Chrysene was shown to be mutagenic to *Salmonella typhimurium* when administered at doses of 10 mg/plate. Another study concluded that chrysene causes embryonic cell transformations in Syrian hamsters. Chrysene is known to cause sister-chromatid-exchange in Chinese hamsters and aberrations in the oocyte development of laboratory mice (IARC, 1983).

Chrysene is thought to be weakly carcinogenic to laboratory animals. It does not appear to be locally or systemically carcinogenic to laboratory animals when exposed epidermally although some studies provide evidence to the contrary. A number of these studies were ignored due to contamination to stock by methylchrysenes (IARC, 1983). Perinatal and subcutaneous administrations have resulted in similar effects and conclusions.

Although some studies have indicated chrysene to be carcinogenic to laboratory animals (Wislocki et al., 1986), IARC has determined that only limited evidence of chrysene's carcinogenicity exists.

Dibenzo (a,h) Anthracene

Researchers reported a decreased growth rate in young rats when exposed to between 3 and 90 mg/kg bw dibenzo (a,h) anthracene (D(a,h)A), (IARC, 1983). No other evidence of systemic or local noncarcinogenic toxic effects were located in the available literature.D(a,h)A was found to be mutagenic to a number of cultured and *in vivo* cells. D(a,h)A was highly mutagenic to *Salmonella typhimurium*. It also induced unscheduled DNA syntheses in the presence of an exogenous metabolic system in cultured mammalian cells. D(a,h)A was found to be embryotoxic to rats when administered in high doses (IARC, 1983).

D(a,h)A has produced tumors in rats, guinea pigs, mice, frogs, pigeons, and chickens (Snell and Stewart; 1962, 1963). Carcinogenic affects, both local and systemic, have been noted as a result of oral, intratracheal, and cutaneous applications (IARC, 1983).

Fluoranthene

Male and female CD-1 mice (20/sex/group) were exposed to 0, 125, 250, or 500 mg/kg/day fluoranthene by gavage for 13 weeks (U.S. EPA, 1988a). Mice exhibited increased food consumption and body weight gain at the highest dose. Increased SGPT values and increased absolute and relative liver weights occurred at 250 and 500 mg/kg/day. Compound-related microscopic liver lesions were observed in 65 and 87.5 percent of the mid- and high-dose mice, respectively.

Fluoranthene was found to be mutagenic in *Salmonella typhimurium* and *in vitro* human lymphoblastoid cells in the presence of an exogenous metabolic system (IARC, 1983). There have been no studies done that indicate fluoranthene to be carcinogenic to humans or laboratory animals. Of eight studies reviewed by IARC, none provided sufficient evidence to conclude that fluoranthene is carcinogenic. However, one study noted twice as many tumors in mice administered fluoranthene in conjunction with BaP than in mice administered BaP alone (IARC, 1983).

Fluorene

CD-1 mice (25/sex/group) were exposed to 0, 125, 250, or 500 mg/kg/day fluorene by gavage for 13 weeks (U.S. EPA, 1989c). Increased spleen, liver, and kidney weights were observed at the high doses. Other systemic effects included a decreasing trend in BUN and an increasing trend in serum bilirubin.

Fluorene does not appear to be mutagenic, teratogenic, or embryotoxic to laboratory animals. Three studies were reviewed by IARC, all were inconclusive as to the reproductive effects of fluorene (IARC, 1983).

Fluorene did not cause cancer in laboratory animals from skin applications, subcutaneous injections, or oral administrations (IARC, 1983). Due to insufficient studies, there is inadequate evidence to evaluate the carcinogenicity of fluorene (IARC, 1983).

Indeno (1,2,3-cd) Pyrene

No data regarding the systemic, mutagenic, teratogenic, or developmental effects of indeno (1,2,3-cd) pyrene (IP) were located in the available literature.

IP is carcinogenic to laboratory mice when administered by skin painting at a dose of 250 µg. Researchers noted that doses of 0.01 and 0.05 percent IP produced no tumors. A dose of 0.1 percent IP produced a total of 6 papillomas and 3 carcinomas in 20 mice. Seven papillomas and five carcinomas were noted in 20 mice painted with 0.5 percent IP. The same study demonstrated that 10 paintings at two-day intervals, resulting in a total dose of 250 mg initiated skin carcinogenesis (IARC, 1983).

When administered subcutaneously to mice, 0.6 mg IP given at one-month intervals, produced 10 sarcomas in 14 male mice and 1 sarcoma in 14 female mice (IARC, 1983).

In a lung implantation study (Deutsch-Wenzel et al., 1983), IP produced epidermoid carcinomas.

2-Methylnapthalene

No data were located in the available literature.

Naphthalene

Naphthalene appears to effect ocular function in humans, rats, and rabbits. Ghetti and Mariani (1956) reported that eight of 21 workers exposed to an unspecified concentration of naphthalene in a dye-manufacturing process developed cataracts. All of these workers were less than 50 years of age. Fitzhugh and Buschke (1949) observed cataracts in young rats exposed to two percent naphthalene by ingestion for 60 days (approximately 1 g/kg bw/day). Ghetti and Mariani (1956) noted similar effects in rabbits.

Naphthalene is known to be fetotoxic because of its ability to cross the placental wall (EPA, 1984). It is also known to cause DNA damage in mice (ICF, 1985). The offspring of rats injected with unspecified amounts of naphthalene displayed retarded cranial and heart development (ICF, 1985).

Wolf (1976) reported that 6 of 15 workers exposed, via inhalation, to naphthalene develop laryngeal carcinomas and neoplasms of the pylorus and cecum. A study on the effects to rats of subcutaneously injected naphthalene concluded with negative results (Schmahl, 1955). The rats were injected with either 10 or 0.82 g naphthalene for an unspecified amount of time. No tumors were noted. (EPA, 1984b).

Phenanthrene

No data regarding the systemic effects of phenanthrene to humans or laboratory animals were located in the available literature.

The majority of the studies concerning the developmental effects of phenanthrene concluded with negative results. One study reported that *Salmonella typhimurium* mutated when exposed to 12 mg phenanthrene (IARC, 1983). Abnormally high concentrations of exogenous metabolites were introduced into the culture before mutations were seen. In two other experiments, phenanthrene was reported to induce mutations *in vitro* human cells and *in vivo* hamster cells. These studies do not provide enough evidence to classify phenanthrene as a mutagenic compound (IARC, 1983).

Experiments indicate that phenanthrene is not carcinogenic to laboratory animals (Higgins and Yang, 1962). No case studies of human exposure to phenanthrene were located. Mice and rats were exposed to phenanthrene via painting, subcutaneous injections, intraperitoneal injections, and ingestion. None of the studies resulted in the induction of tumors (IARC, 1983).

Pyrene

Cd-1 mice were exposed to 0, 75, 125, or 250 mg/kg/day pyrene by gavage for 13 weeks (U.S. EPA, 1989d). Nephropathy and reduced relative and absolute kidney weights were observed in the high dose groups.

It was noted in one study of the effects of pyrene exposure that the growth of young rats was inhibited when fed 2000 mg pyrene/kg/day for 100 days. In the same study, it was noted that the rats' livers were enlarged after prolonged exposure (IARC, 1983). No toxic effects to humans or animals resulting from pyrene exposure were noted in the available studies, although one researcher reported an LD_{50} for mice of 678 mg pyrene/kg-bw for four days (IARC, 1983). Pyrene induced unscheduled DNA synthesis in cultured rat hepatocytes (EPA, 1984d) and cultured human fibroblast cells (IARC, 1983). It induced sister-chromatid-exchange in Syrian hamster embryonic cells in one instance and, in another, it mutated *Salmonella typhimurium* cultures (IARC, 1983).

The carcinogenic effects of pyrene on laboratory animals have been studied extensively. Oral, inhalation, injection, and topical studies have all been performed and have all concluded that pyrene is noncarcinogenic (EPA, 1984d). Pyrene did not initiate tumors in mouse skin, although it did enhance the carcinogenic effects of benzo (a) pyrene when co-applied. Evidence regarding the carcinogenicity of intratracheal administration were considered inadequate for evaluation (IARC, 1983).

Ecotoxicity

The ecotoxic effects of PAHs have not been widely studied. It appears as though the effects of PAHs on aquatic organisms are more variable than the effects on humans.

Acenapthene resulted in 96-hour LC_{50} values of 970 and 2,230 mg/l for mysid shrimp and sheepshead minnows, respectively. Two freshwater species subjected to acenapthene exposure displayed EC_{50} values of 41,200 and 1,700 mg/l (*Daphnia magna* and bluegill, respectively) (ICF, 1985).

Fluoranthene appears to be less toxic to freshwater species than does acenapthene. The 96-hour LC_{50} value for bluegill was 3,970 mg/l and the 48-hour EC_{50} value for *Daphnia magna* was 325,000 mg/l. The 96-hour LC_{50} value for mysid shrimp, a saltwater species, was 40 mg/l, significantly lower than the value for acenapthene (ICF, 1985). Fluoranthene is known to bioaccumulate but, to what extent is unknown.

The medium effect concentration of naphthalene for freshwater species was reported to be greater than 2,300 mg/l. Acute values for saltwater species (polychaetes, oysters, shrimp) are reported to be greater than 2,350 mg/l (ICF, 1985).

Standards Criteria and Guidelines

Summarized in Table 2.

TABLE 2. STANDARDS, CRITERIA AND GUIDELINES FOR PAHs(a)

Compound	EPA Carc. Class	Slope Factor Inh/Oral (mg/kg/day)$^{-1}$*	Chronic Oral RfD (mg/kg/day)	Chronic Inhalation RfD (mg/kg/day)	Subchronic Oral RfD(b) (mg/kg/day)	Subchronic Inhalation RfD (mg/kg/day)	MCL(c) (mg/l)	Ambient Water Quality Criteria (d) Fish & Water Consumption	Ambient Water Quality Criteria (d) Fish Consumption
Acenapthene	--	--	6.0x10^{-2}	--	6.0x10^{-1}	--	--	2.8 ng/l**	
Acenapthylene	D	--	p	--	--	--	--		
Anthracene	D	--/7.3	3.0x10^{-1}	--	3.0x10^{0}	--	--		
Benzo(a)anthracene	B2	--/7.3 x 10^{-1}	--	--	--	--	0.0001		
Benzo(a)pyrene	B2	--/7.3 x 10^{0}	--	--	--	--	0.0002		
Benzo(b)fluroanthene	B2	--/7.3 x 10^{-1}	--	--	--	--	0.0002		
Benzo(k)fluroanthene	B2	--/7.3 x 10^{-1}	--	--	--	--	0.0002		
Benzo(g,h,i)perylene	D	ND/ND	--	--	--	--	--		
Chrysene	B2	--/7.3 x 10^{-2}	--	--	--	--	0.0002		
Dibenz(a,h)anthracene	B2	--/7.3 x 10^{0}	--	--	--	--	0.0003		
Fluoranthene	D	--	4.0x10^{-2}	--	4.0x10^{-1}	--	--	42 µg/l	54 µg/l
Fluorene	D	--	4.0x10^{-2}	--	4.0x10^{-1}	--	--		
Indeno(1,2,3-c,d)pyrene	B2	--/7.3 x 10^{-1}	--	--	--	--	0.0004		
2-Methylnapthalene	--	--	--	--	--	--	--		
Naphthalene	D	--	--	--	--	--	--		
Phenanthrene	D	--	--	--	--	--	--		
Pyrene	D	--	3.0x10^{-2}	--	3.0x10^{-1}	--	--		

*Values derived from B(a)P slope factor listed on IRIS by applying Toxic Equivalency Factors (TEFs).
-- = no data
ND = not determined
** = PAHs, in general (from U.S. EPA, Quality Criteria for Water, May 1986)
p = pending; currently under review by EPA.
References:
(a) U.S. EPA, Integrated Risk Information System (IRIS).
(b) U.S. EPA, Health Effects Assessment Summary Tables (HEAST).
(c) U.S. EPA, Drinking Water Regulations and Health Advisories.
(d) U.S. EPA, OERR, CERCLA Compliance With Other Laws Manual, Interim Final, August 1988.

BIBLIOGRAPHY

Adkins, B., et al. 1986. Oncogenic response of strain A/J mice to inhaled chemicals. *J. Toxicol. Environ. Health* 17:311-3222. (cited in IRIS).

American Conference of Governmental Industrial Hygienists. 1984. *Documentation of the Threshold Limit Values.*

Badger, G.M., et al. 1944. The production of cancer by pure hydrocarbons. *V. Proc. R. Soc. London Ser. B* 129: 439-467. (cited in IRIS).

Bos, R.P., et al. 1988. Mutagenicity of bi, tri-, and tetra-cyclic aromatic hydrocarbons in the "taped-plate assay" and in the conventional Salmonella Mutagenicity assay. *Mutat Res.* 204: 203-206. (cited in IRIS).

Chang, L.H. The fecal Excretion of Polycyclic Hydrocarbons Following Their Administration to the Rat. *J. Biol. Chem.* 151: 93-1943. (Cited in EPA, 1984a).

Clayton and Clayton. 1978. *Patty's Industrial Hygiene and Toxicology Vol. IIB.* (Patty's)

Cook, J.W. 1932. The production of cancer by pure hydrocarbons-Part II. *Proc. Royal Soc. London S.B.* 11:485-496. (cited in IRIS).

Cottini, G.B. and G.B. Mazzone. 1939. The effects of 3,4-Benzpyrene on Human Skin. *Am J. Cancer.* 37: 186. (Cited in EPA, 1984e).

deSerres, F. and J. Ashby, Ed. 1981. Evaluation of Short-Term Tests for Carcinogens. *Reports on the International Collaborative Program.* Elsevier/North-Holland Biomedical Press, New York, p. 180, 190. (Cited in EPA, 1984e).

Deutsch-Wenzel, R., et al. 1983. Experimental studies in rat lungs on the carcinogenicity and dose-response relationships of eight frequently occurring environmental polycyclic aromatic hydrocarbons. *J. Nat'l. Cancer Inst.* 71 (3): 539-543. (cited in IRIS).

Fitzhugh. O.G. and W.H. Buschke. 1949. Production of Cataracts in rats by betatetralol and other derivatives of naphthalene. *Arch. Opthal.* 41: 572-583. (Cited in EPA 1984b).

Ghetti, C. and L. Mariani. 1985. Eye changes due to naphthalene. *Med. Lav.* 47: 524. (Cited in EPA, 1984b).

Hollstein, M. and J. McCann. 1979. Short-term tests for carcinogens and mutagens. Mutat. Res. 65: 133-226. (Cited in EPA, 1984e).

Huggins, C. and N.C. Yang. 1962. Induction and extinction of mammary cancer. Science 137 (3562): 257-262. (cited in IRIS).

IARC (International Agency for Research in Cancer). 1982. Chemicals, Industrial Processes and Industries Associated with Cancer in Humans, In: *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans.* WHO, IARC, Lyon, France. Suppl. 4. (Cited in EPA, 1984e).

ICF, Clement Associates. 1985. *Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Site.*

International Agency for Research on Cancer. 1983. *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Human.*

Kaden, D.A., et al. 1979. Mutagenicity of Soot and Associated polycyclic aromatic hydrocarbons to Salmonella typhimurium. *Cancer Res.* 39:4152-4159. (cited in IRIS).

Kennaway, E.L. 1924. On cancer-producing tars and tar-fractions. *J. Ind-Hyg.* 5(12): 462-488. (cited in IRIS).

Klein, M. 1963. Susceptibility of strain B6AF/J Hybrid infant mice to tumorigenesis with 1,2-benzanthracene, deoxycholic acid, and 3-methyl-cholanthrene. *Cancer Res.* 23: 1701-1707. (cited in IRIS).

Knobloch K., S. Szendzikowski, and A. Slusarcyk - Zalobna. 1969. Acute and subacute toxicity of acenaphthene and acenaphthylene. *Med. Pracy.* 20(3): 210-222. CA 72(7) 299117b, (Cited in EPA, 1984f).

Mackenzie, K.M. and D.M. Angevine. Infertility in Mice Exposed *in utero* to Benzo(a)pyrene. *Biol. Reprod.* 24: 183-191. (Cited in EPA, 1983).

National Toxicology Program (NTP). 1980. Unpublished subchronic toxicity study: Naphthalene (C52904), Fischer 344 rats. Prepared by Battelle's Columbus Laboratories under subcontract no. 76-34-106002. (cited as principal RfD study in HEAST).

Neal, J. and R.H. Rigdon. 1967. Gastric Tumors in Mice Fed Benzo(a)pyrene: A quantitative Study. *Tex. Rep. Boil. Med.*, 25: 553. (Cited in EPA, 1984e).

Reshetyuk, A.L., E.I. Talakina, and P.a. En' yakova. 1970. Toxicological evaluation of acenaphthene and acenaphthylene. *Gig. Tr. Prof. Zabol.* 14(6): 46-47 CA 73(23)118650x, (Cited in EPA, 1984f).

Rigdon, R.H. and E.G. Rennels. 1964. Effect of Feeding Benzo(a)pyrene on Reproduction in the Rat. *Experientia.* 20: 224-226. (Cited in EPA, 1984e).

Rigdon, R.H. and J. Neal. 1969. Relationship of leukemia to lung and stomach tumors in mice feed benzo(a)pyrene. *Proc. Soc. Exp. Biol.* NY. 130: 146. (Cited in EPA, 1984e).

Robinson, J.R., J.S. Felton, R.C. Levitt, S.C. Thorgiersson, and D.W. Neber. 1975.

Relationship Between "Aromatic Hydrocarbon Responsiveness" and the Survival Times in Mice Treated with Various Drugs and Environmental Compounds. *Mol. Pharmacal.* II: 850-865. (Cited in EPA, 1984e).

Schmahl, D. 1955. Examination of the carcinogenic action of naphthalene and anthracene in rats. Z. *Krebsforsch* 60:697-710. (cited in IRIS).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

Stanton, M.F., et al. 1972. Experimental induction of epidermoid carcinoma in the lungs of rats by cigarette smoke condensate. *J. Nat'l Cancer Inst.* 49(3): 867-877. (cited in IRIS).

Snell, K.C. and H.L. Stewart. 1962. Pulmonary adenomatose induced in DBA/2 mice by oral administration of dibenz (a,h) anthracene. *J. Natl Cancer Inst.* 28(5): 1043-1049. (cited in IRIS).

Snell, K.C., and H.L. Stewart. 1963. Induction of pulmonary adenomatose in DBA/2 mice by the oral administration of dibenz (a,h) anthracene. *Acta. Un. Int. Cancer* 19:692-694. (cited in IRIS).

Thyssen, J., J. Althoff, G. Kimmerle, and U. Mohr. 1981. Inhalation Studies with Benzo(a)Pyrene in Syrion Golden Hamsters. *J. Natl. Cancer Inst.* 66(3): 575-577. (Cited EPA, 1984e).

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1984a. *Health Effects Assessment for Polycyclic Aromatic Hydrocarbons*. EPA 540/1-86/013.

U.S. EPA. 1984b. *Health Effects Assessment for Naphthalene*. EPA 600/8-89/094.

U.S. EPA. 1984c. *Health Effects Assessment for Phenanthrene*. EPA 540/1-86/029.

U.S. EPA. 1984d. *Health Effects Assessment for Pyrene*. EPA 540/1-86/030.

U.S. EPA. 1984e. *Health Effects Assessment for Benzo(a)pyrene*. Final Draft.

U.S. EPA. 1984f. *Health Effects Assessment for Acenaphthene.*

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System* (IRIS).

U.S. EPA. 1986. *Superfund Public Health Evaluation Manual.*

U.S. EPA. 1988a. *Subchronic toxicity in mice with anthracene.* Final Report. Hazleton Laboratories, Ltd. Muskegon, MI. for the Office of Solid Waste, Wash, D.C. (cited in IRIS).

U.S. EPA. 1988b. *13-Week mouse oral subchronic toxicity study.* Toxicity Research Laboratories, Ltd. Muskegen, MI. for the office of Solid Waste, Wash, D.C. (cited in IRIS).

U.S. EPA. 1989d. *Mouse oral subchronic toxicity for Pyrene.* Study conducted by Toxicity Research Laboratories, Muskegon, MI. for the Office of Solid Waste, Wash., D.C. (cited in IRIS).

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* EPA 440/5-86-001.

U.S. EPA. 1989a. *Mouse oral subchronic study with acenaphthene.* Study conducted by Hazleton Laboratories, America, Inc. for the Office of Solid Waste, Wash., D.C. (cited in IRIS).

U.S. EPA. 1989b. *Subchronic toxicity in mice with anthracene.* Final Report Hazleton Laboratories, America, Inc. Prepared for the Office of Solid Waste, Wash. D.C. (cited in IRIS).

U.S. EPA. 1989c. *Mouse oral subchronic toxicity study.* Prepared by Toxicity Research Laboratories, Ltd. Muskegon, MI. for the Office of Solid Waste, Wash., D.C. (cited in IRIS).

Wislocki, P.G., et al., Tumorigenicity of nitrated derivatives of pyrene, benz(a)anthracene, chrysene, and benzo(a)pyrene in the newborn mouse assay. *Carcinogenesis.* 7(8):1317-1322. (cited in IRIS).

Wolf, O. 1976. Cancer Diseases in Chemical Workers in a Former Naphthalene Cleaning Plant. *Deutsche Gesundheituesen.* 31: 996. (Cited in EPA, 1984b)

Wynder, E.L. and D. Hoffman. 1959. A study of tobacco carcinogenic. VII. The role of higher polycyclic hydrocarbons. *Cancer* 12: 1079-1089. (cited in IRIS).

Van Duuren, B.L., et al. 1966. The tumor-promoting agents of tobacco leaf and tobacco smoke condensate. *J. Nat'l Cancer Inst.* 37(4): 519-526. (cited in IRIS).

BUTYL BENZYL PHTHALATE

Use

No information regarding the use of butyl benzyl phthalate (BBP) was located in the available literature.

Chemical and Physical Properties

Chemical Formula: $C_{19}H_{20}O_2$

MW: 312	BP: 377°C at 760 torr
Sol (water): 2.9 mg/L	MP: -35°C

Fate and Transport

Volatilization, photolysis and hydrolysis appear to be insignificant as fate processes of BBP. It is thought that, because phthalate esters, the class in which BBP is grouped, generally sorb to organic material and bioaccumulate, BBP would to do the same (EPA, 1979).

Pharmacokinetics

BBP appears to effect the kidneys, liver and testes of male rats (IRIS, 1990).

Human Toxicity

Noncarcinogenic

Systemic Effects

The National Toxicology Program (NTP) administered doses between 17 and 1417 mg BBP/kg/day in the diet of rats. Rats in the highest dose groups displayed depressed body weight and shrunken testes. Mean organ weight decreased for the heart, kidneys, lungs and seminal vesicles in the highest dose group only. An increase in liver weight was observed at the next to highest dose administration. Only the highest dose group showed evidence of abnormal morphology in any organ. In an addendum to this study, NTP noted a significant reduction in the total marrow cell count in the high dose group.

Two 14-day studies support the NTP results. In the first, significant increases in the liver and kidney weights of rats administered 5.0 percent doses of BBP per day for 14 days were noted. In the other, it was reported that only liver weights increased in rats fed 500,750 or 1000 mg BBP/kg/day (IRIS).

Teratogenic and Other Developmental Effects

No information regarding the teratogenic or developmental effects of BBP were located in the available literature.

Mutagenic Effects

Studies indicate that BBP is not mutagenic to *Salmonella typhimurium, E. Coli* of Chinese hamster ovary cells (IRIS).

Carcinogenic Effects

NTP (1985) noted that, among female rats given 12,000 ppm BBP daily for 103 weeks, the number of mononuclear cell leukemia or lymphoma significantly increased after 83 weeks. When given similar doses, male rats exhibited high mortality rates within the first 30 weeks due to internal hemorrhaging (IRIS). In the same study, however, mice displayed no toxic or carcinogenic effects when exposed to the same dosage. Theiss et al. (1977) noticed no induction of lung adenomas in Strain A mice administered intraperitoneal injections of 160, 400 or 800 BBP mg/kg.

Ecotoxicity

No information regarding the ecotoxic effects of BBP were located in the available literature.

Standards, Criteria and Guidelines

EPA Class C Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2×10^{-1} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	2×10^{0} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.1 mg/L
AWQC:	NA

BIBLIOGRAPHY

National Toxicity Program (NTP). 1985. *Twenty-six Weeks Subchronic Study and Modified Matting Trial in F344 Rats, Butyl Benzyl Phthalate,* Final Report. Project No. 12307-02, -03; Hazelton Laboratories America, Inc. Unpublished Study. (Cited in IRIS).

Theiss, J.C., G.D. Stoner, M.B. Shimkin, and E.K. Weisburger. 1977. Test for carcinogenicity of organic contaminants of United States drinking waters by pulmonary tumor response in Strain A mice. *Cancer Res. 37: 2717-2720.* (Cited in IRIS).

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1979. *Water Related Environmental Fate of 129 Priority Pollutants.*

BIS(2-ETHYLHEXYL)PHTHALATE

Use

The phthalate esters, such as bis(2-ethylhexyl)phthalate (BEHP), are widely used in PVC resins and vinyl copolymer resins to impart flexibility to the finished product. Other reported uses include as an inert ingredient in pesticides, a component in dielectric fluids (replacing PCBs) in electric capacitors, a solvent for erasable ink, acarid in orchids, in vacuum pump oils, and as a testing agent for air filtration systems. Consumer products using BEHP include vinyl upholstery, table cloths, shower curtains, raincoats, and food wrap. Annual consumption of BEHP is approximately 130 million kg.

Chemical and Physical Properties

Chemical Formula: $C_6H_{40}\ (COOCH_2CH\ (C_2H_5)C_4Hg)_2$

MW:	3,190	BP:	386.9 C at 5 mmHg
SG:	0.985 at 20 C	MP:	-50 C
FP:	218.33 C	VP:	2×10^{-7} mmHg at 20 C

Sol. (water): 0.4 mg/L at 25 C
Sol. (organics): mineral oil and hexaney

Fate and Transport

In aquatic media, BEHP does not volatilize or photo-oxidize readily. Apparently, adsorption to suspended solid and particular matter are probably the most important of BEHPs fate processes (ICF, 1985). Bioaccumulation is another important fate process for BEHP. Several unicellular and multicellular aquatic organisms are known to accumulate BEHP (ICF, 1985).

In soils, BEHP would be expected to sorb to organic matter. Very little volatilization and leaching would be expected (ICF, 1985).

Pharmacokinetics

Studies indicate that, following an oral dose, BEHP is initially hydrorolyzed by a nonspecific lipase in the gastrointestinal tract to produce mono(ethylhexyl)phthalate (MEHP) (and 2-ethylhexanol) which is readily absorbed from the gastrointestinal tract. One study indicated that BEHP is poorly absorbed following dermal application. In acute inhalation toxicity studies in rats, it has been demonstrated that BEHP is absorbed by the lung. Information on the oral absorption of BEHP in humans is limited, and data is not available on the absorption of BEHP by humans exposed via inhalation or through dermal exposure (ATSDR, 1989).

Absorbed BEHP and is metabolites are distributed rapidly to tissues and organs with only a slight cumulative potential. The liver appears to be the major, initial repository organ. BEHP is eliminated from the body mainly through urinary excretion. Urinary metabolites appear to differ amongst species (ATSDR, 1989).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Various rodent studies revealed LD_{50}s ranging from 26,000 to 49,000 mg/kg following oral exposure. No data are available on the effects of oral, ingestion, or dermal exposure to BEHP on lethality in humans (ATSDR, 1989).

The liver and the testes have been shown to be the primary target organs of BEHP. Morphological and biochemical changes in the liver of exposed rodents have been observed following exposure to high doses of BEHP. No data are available on the hepatic toxicity of BEHP in humans via inhalation, oral, or dermal exposure. Testicular effects, including a decrease in relative organ weight and histological changes in the seminiferous tubules have been observed in the rat and mouse, but not in the hamster, ferret, or marmoset following exposure to BEHP and MEHP (ATSDR, 1989).

Teratogenic and Other Developmental Effects

BEHP is a reproductive toxicant in male and female mice; reduced fertility and both production of fewer litters by breeding pairs and decreased litter size has been observed (ATSDR, 1989). Available data suggests that BEHP is developmentally toxic in rats and mice. One study indicated that, following administration of 0.05, 0.1, 1.0, 2.5, 5.0, or 10.0 Ml/kg BEHP by gavage on day 7 of gestation, a decrease in body weight of live fetuses occurred at the 0.05 mL/kg dose. At doses administered at or above 0.1, mL/kg, a decrease in fetal body weight was observed, and the fetuses were deformed or dead. In a study in which pregnant Fisher 344 rats were exposed to BEHP in their diets during 0 to 20 of gestation, the number and percentage or resorptions, non-live fetuses, and malformed fetuses were increased in a dose-related manner; with a statistically significant increase in the high-dose group (20,000 ppm/1,055 mg/kg/day) (ATSDR, 1989). The NOAEL for teratogenic effects or maternal toxicity in a study of pregnant CD-1 mice exposed to BEHP in their diets was 250 ppm. BEHP was found to be developmentally toxic in ICR mice when administered orally (at 1,000 mg/kg and 2,000 mg/kg), but not when administered by intraperitoneal injection. One hundred percent of live fetuses were malformed when pregnant mice were given 1 mL/kg MEHP on day 8 of gestation (ATSDR, 1989).

Mutagenic Effects

BEHP has not been shown to be mutagenic in most microbial and mammalian assay systems. Most of the data also suggest that MEHP and 2-ethylhexanol are not mutagenic (ATSDR, 1989).

Carcinogenic Effects

EPA has evaluated the weight of evidence on the carcinogenicity of BEHP and has concluded that it is a probable human carcinogen (Group B2). Evidence on potential carcinogenicity from animal studies is "sufficient", however there is no adequate human data. Data from a bioassay using Fisher 344 rats and B6C3F1 mice have been used by EPA to calculate the upper-bound incremental unit carcinogenic risk to humans (the unit risk value is estimated to be 4.0×10^{-7} for drinking water containing 1 µg/L BEHP). These rodents were fed diets containing 0, 6,000 or 12,000 ppm for 103 weeks. A statistically significant increase in hepatocellular carcinomas and neoplastic nodules was observed in the high dose groups (NTP, 1982).

Ecotoxicity

The LC_{50} values for the midge, scud, and bluegill all exceeded the highest concentrations tested, which were 18,000 32,000 and 770,000 µg/liter, respectively. Because these values are greater than the water solubility of the chemical, it is unlikely that BEHP will be acutely toxic to organisms in natural waters. In a chronic toxicity test with *Daphnia magna,* significant reproductive impairment was found at the lowest concentration tested, 3µg/liter. These data imply that some chronic toxicity will be observed in freshwater aquatic life subsequent to long-term exposure to BEHP (ICF, 1985).

BEHP is removed from water primarily through uptake by suspended matter, sediments, and biota. BEHP is absorbed by both single- and multi-cellular organisms. The tendency for BEHP to undergo bioaccumulation is lessened because it is degraded by microorganisms and metabolized by invertebrates, fish and other animals. Very rapid bioaccumulation and concentration factors ranging from several hundred to several thousand times the concentration of BEHP in water, however, have been observed for various aquatic organisms, seen mostly in smaller aquatic invertebrates (ATSDR, 1989).

Acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 940 and 3 µg/L, respectively, according to available data; more sensitive species than those tested would be expected to be affected by even lower concentrations. For saltwater aquatic life, acute toxicity occurs at concentrations as low as 2,944 µg/L. No data are available to enumerate the chronic toxicity of phthalate esters to saltwater aquatic life; however, toxicity of one species of algae occurs at concentrations as low as 3.4 µg/L (EPA, 1986).

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	1.4 x 10^{-2} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2 x 10^{-2}mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	2 x 10^{-2} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 15 mg/l Fish Consumption - 50 mg/l

BIBLIOGRAPHY

Agency for Toxic Substances and Disease Registry. U.S. Public Health Service. 1989. *Toxicological Profile for di(2-ethylhexyl)phthalate,* ATSDR/TP-88/15.

Carpenter, C.P., et. al. 1953. Chronic oral toxicity of di(2-ethylhexyl) phthalate for rats and guinea pigs. *Arch. Indust. Hyg. Occup. Med.* 8: 219-226. (Cited in IRIS).

Carpenter, C.P., et. al. 1953. Chronic oral toxicity of di(2-ethylhexyl) phthalate for rats, guinea pigs, and dogs. *AMA Arch. Ind. Hyg. Occup. Med.* 8:219-226. (cited in IRIS).

ICF Clement Associates. 1985. *Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites,* Final Report.

National Toxicity Program (NTP). 1982. Carcinogenesis bioassay of bis-(2-ethylhexyl) phthalate (CAS No. 1117-81-7) in F344 rats and B6C3F1 mice (feed study). *NTP Tech. Rep. Ser. Tr. No. 217,* NTP, Research Triangle Park, NC. (Cited in IRIS).

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water,* EPA 440/5-86-001.

4-CHLOROANILINE

Use

4-Chloroaniline is used in the synthesis of dyestuffs, chemical products, insecticides and many other industrial products.

Chemical and Physical Properties

Chemical Formula: $C_6H_4NH_2Cl$

MW: 127.57	BP: 231°C
FP: >104°C	MP: 70°C

Sol. (water): Insoluble
Sol. (organics): Ether

Fate and Transport

No information on this topic was found in the literature.

Pharmacokinetics

Routes of entry include inhalation of vapor, skin contact and ingestion (Sittig, 1991).

Human Toxicity

Noncarcinogenic

Systemic Effects

Exposure to 4-chloroaniline may decrease the blood's ability to carry oxygen. This can cause headaches, breathing difficulty, weakness, a bluish color to the nose and lips, collapse and death. Contact can severely irritate and burn the eyes (Sittig, 1991).

Rats fed 250 and 500 ppm 4-chloroaniline for 78 weeks showed increased mortality and decreased body weight in the 500 ppm group. Lesions of the spleen were observed in most of the treated rats (NCI, 1979).

Teratogenic and Development Effects

No information on this subject was found in the literature.

Mutagenic Effects

No information on this subject was found in the literature.

Carcinogenic Effects

No information on this subject was found in the literature.

Ecotoxicity

No information on this subject was found in the literature.

Standards, Criteria, and Guidelines

Unclassified by EPA as to carcinogenicity.

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	4.0 x 10^{-3} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	4.0 x 10^{-3} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

BIBLIOGRAPHY

NCI (National Cancer Institute). 1979. Bioassay of p-chloroaniline for possible carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 189. NTIS PB 295896 (cited in IRIS).

Sittig, M. 1991. Handbook of Toxic and Hazardous Chemicals and Carcinogens.

U.S. EPA. Integrated Risk Information System (IRIS).

DIETHYL PHTHALATE

Use

Diethyl phthalate (DEP) is used as a solvent for cellulose esters, as a vehicle in pesticidal sprays, as a fixitive and solvent in perfumery, as an alcohol denaturant, and as a plasticizer in solid rocket propellants (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: $C_6H_4\ (OCOC_2H_5)_2$

MW:	222.24	BP:	298°C
SG:	1.1175 at 20°C	MP:	-40.5°C
Sol.(water):	896 mg/liter	VP:	0.05 mm Hg at 70°C

Sol.(organics): Acetone, benzene

Fate and Transport

Much of the information on the fate and transport of DEP is for phthalate esters in general. ICF (1985) evaluated this general information in relation to DEP. ICF reports that hydrolysis of DEP in surface waters is likely although this process occurs at such a slow rate that it is not environmentally significant. Photolysis and oxidation are not significant fate processes either. Although volatilization is not a significant fate in natural waters, it may occur slowly from DEP-containing materials at relatively high temperatures.

The most important environmental fate for DEP is absorption onto suspended solids and particulate matter, and complexation with natural organic substances. ICF (1985) reports that the octanol/water partition coefficient for DEP suggests that it would be absorbed onto particulates high in organic matter and, in fact, phthalate esters are often found in sediment samples. DEP readily forms water-soluble complexes with humic substances which may lead to its dispersal is aquatic and terrestial systems.

Bioaccumulation is considered an important fate process. Many unicellular and multicellular organisms take up and accumulate phthalate esters (ICF, 1985). Phthalate esters are biodegraded under most conditions, however, making long-term bioaccumulation unlikely.

Pharmacokinetics

EPA (1980) reports that phthalate esters and their metabolites are readily absorbed from the intestinal tract, the intraperitoneal cavity, and the lungs. Shaffer et al. (1945) reports that a single oral dose of 10g of di-2-ethylhexyl phthalate (DEHP) in a human subject resulted in the recovery of a phthalate equivalent equal to 4.5 percent of the original dose in the urine after 24 hours, 5g of DEHP resulted in a two percent recovery. A study by Dillingham and Pesh-Imam (unref.) indicated that dermal absorption may also occur. 24 hours after labeled

DEHP had been applied to rabbit skin, nine percent was detected in urine. After 48 hours, this level increased to 14 percent and after 72 hours, it reached 16-20 percent.

Absorbed phthalate esters and/or their metabolites are distributed quite rapidly to various organs and tissues in humans depending upon the route and physical form of the ester (EPA, 1980). Jaeger and Rubin (1970) reported the presence of DEHP in the spleen, liver, lung, and abdominal fat of two deceased patients who had received large volumes of blood stored in PVC blood bags. It also appears that, although distribution is rapid there is no apparent accumulation. Waddell, et al. (1977) found that DEHP accumulated in the kidney and liver was rapidly excreted into urine, bile, and the intestine.

However, EPA (1980) reports that patients having received large volume blood or blood products may have phthalate ester residues in their tissues and organs. Jacobson, et al. (1977) found trace amounts of DEHP 14 months after a transfusion in nonhuman primates. Dillingham and Pesh-Imam (unref.) report that dermal application of DEP resulted in its distribution to the lungs, heart, liver, kidneys, gonads, spleen, and brain after 3 days. Interestingly, no DEP was detected on the skin or subdermal fatty tissue at the site of application.

EPA (1980) states that significant biotransformation of phthalate esters in the gut is likely based on the Albro, et al. (1973) study with DEHP and rats. In their study, DEHP was converted to a monoester which is then further metabolized in the liver. Phthalate esters are, for the most part, readily excreted in urine and feces in humans (EPA, 1980). Lake, et al. (1975) found nearly all of a single oral dose of DEHP excreted in urine and feces within a four day period.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The phthalate esters may be considered as having a relatively low order of toxicity. In fact, it is now thought that the toxic effects of the esters are due to a metabolite, particularly the monoester (EPA, 1980). The low volatility of most of the esters makes acute toxicity from inhalation unlikely (EPA, 1980).

Several studies have indicated that chronic exposure to DEP affects weight gain and food consumption by laboratory animals. Brown et al. (1978) exposed groups of male and female CD rats to diets containing 0, 0.2, 1.0, or 5.0 percent DEP for 16 weeks. No changes in behavior or other clinical signs of toxicity were observed. Those rats exposed to 5.0 percent DEP exhibited significantly less weight gain throughout the 16 weeks, as did the females exposed to 1.0 percent DEP. These rats also exhibited reduced food consumption. Urinalysis, histologic and hematologic examinations did not reveal any dose- or time-related trends. Although the absolute weights of the brain, heart, spleen, and kidneys were decreased in rats exposed to 5.0 percent DEP, the relative weights of the brain, liver, kidneys, stomach, small intestines, and full caecum were significantly greater in these rats.

In another study by Brown et al. (1978), rats exposed to 5.0 percent DEP in the diet exhibited increased food consumption (although not statistically significant) and a 7-10 percent reduction in weight gain. Food Research Laboratories, Inc. (1955) also reports reduced weight gain in rats exposed to 5.0 percent DEP in the diet. They also report a significant decrease in the efficiency of food utilization in this group.

Teratogenic and Other Developmental Effects

Singh et al. (1972) exposed pregnant Sprague-Dawley rats to 0.506, 1.012, and 1.686 ml/kg DEP intraperitoneally on days 5, 10, and 15 of gestation. Offspring exhibited skeletal malformations and reduced fetal size. NTP (1984) reports that oral exposure to 0.25, 1.25, and 2.5 percent DEP does not effect the reproductive performance of mice, however, second generation breeding pairs exposed to 2.5 percent DEP exhibited increased right epididymis and prostrate weights in males and decreased pituitary weights in females.

Mutagenic Effects

U.S. EPA (IRIS) reports that several studies have found DEP to be a weak direct-acting mutagen in forward and reverse mutation assays in S. typhimurium. Several other studies reported by U.S. EPA indicate that DEP produced negative results in mammalian cell chromosomal aberration assays.

Carcinogenic Effects

No data on the carcinogenicity of DEP were available in the literature reviewed.

Ecotoxicity

EPA (1986) reports that the available data for phthalate esters indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 940 and 3 µg/l respectively. Acute toxicity to saltwater species occurs at concentrations as low as 2,944 µg/l and chronic toxicity to one species of algae occurs at 3.4 µg/l. It should be noted that these concentrations would be lower in species more sensitive than those tested.

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	8×10^{-1} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	8×10^{0} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption: 350 mg/L Fish Consumption: 1800 mg/L

BIBLIOGRAPHY

Albro, P.W. et al., Metabolism of dimethyl hexyl phthalate by rats. Isolation and characterization of the urinary metabolies. *Jour chromatogr.* 76:320. 1973 (cited in EPA, 1980).

Brown, D. et al., Short-term oral toxicity study of diethyl phthalate in the rat. *Food Cosmet. Toxicol.* 16:415-422. 1978 (cited in IRIS).

Dellingham and Pesh-Imam, unreferenced by EPA, 1980.

Food Research Laboratories, Inc. Toxicological studies of diethyl phthalate. Lab No. 67567. Celanese Corp of America. *Summit Research Laboratories* Summit, NJ. 1955 (cited in IRIS).

ICF, Clement Associates, *Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites*, 1985.

Jacobsen, M.S. et al., Effects of a plasticizer leached from polyvinyl chloride on the subhuman primate: A consequence of chronic transfusion therapy. *Jour. Lab. Clin. Med.* 89:1066. 1977 (cited in EPA, 1980).

Jaeger, R.J. and R.J. Rubin, Plasticizers from plastic devices: Extraction, metabolism, and accumulation by biological systems. *Science*: 170:460. 1970 (cited in EPA, 1980).

Lake, B.G. et al., Studies on the hepatic effects of orally administered di-(2-ethylhexyl) phthalate in the rat. *Toxicol. Appl. Pharmacal* 32:355. 1975 (cited in EPA, 1980).

NTP (National Toxicology Program). *Diethyl phthalate: Reproduction and fertility assessment in CD-1 mice when administered in the feed.* Final report. NTP, Research Triangle Park, NC. 1984 (cited in IRIS).

Rubin, R.J. et al., Ames mutagenic assay of a series of phthalic acid esters: Positive response of the dimethyl and dimethyl esters in TA 100. *Soc. Toxicol. Annu. Mtg.* New Orleans, March p.11 (cited in EPA, 1980).

Shaffer, C.B. et al., Acute and subacute toxicity of di(2-ethylhexyl) phthalate with note upon its metabolism. *Jour. Ind. Hyg. Toxicol* 27:130. 1945 (cited in EPA, 1980).

Singh, A.R. et al. Teratogenecity of phthalate esters in rats. *J. Pharmacol. Sci.* 61(1):51-55. 1972 (cited in IRIS).

Sittig, M. *Handbook of Toxic and Hazardous Chemicals*, 1991.

U.S. EPA, *Ambient Water Quality Criteria for Phthalate Esters*, October, 1980.

U.S. EPA, *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA, *Quality Criteria for Water*, 1986.

DI-N-BUTYL PHTHALATE

Use

Di-n-butyl phthalate, also known as dibutyl phthalate and dibutyl-1,2-benzenedicarbozylate, is used in plasticizing vinyl acetate emulsion systems and in plasticizing cellulose esters. It is also used as an insect repellant (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: $C_6H_4\ (COOC_4H_9)_2$	BP: 340°C
MW: 278.34	FP: 171°C
Sol. (water): 0.4 mg/liter	SG: 1.0484 at 20°C

Sol. (organics): very soluble in alcohol, ether, acetone, and benzene.

Fate and Transport

The U.S. EPA (1981) reports the dominant transport process for di-n-butyl-phthalate is probably that of phthalate esters as a group, that is, sorption onto particulates and complexation with organics. Direct photolysis does not occur and indirect photolysis appears to be too slow to be environmentally important. Oxidation is considered unimportant and hydrolysis of phthalate esters as a group is believed to be too slow to be significant. Volatilization, as well, does no appear to be an important transport process. Phthalate esters in general have been found bioaccumulated in many organisms, are known to biodegrade rapidly in natural soil, and undergo some biotransformation. In fact, all biological processes are considered important fates for phthalate esters (U.S. EPA, 1981).

Pharmacokinetics

The U.S. EPA (1980) reports that phthalic acid esters and/or their metabolites are readily absorbed from the lungs, intraperitoneal cavity, the intestinal tract, and possibly through the skin. Shaffer et al. (1945) reported that 4.5 percent of a single dose of 10g of DEHP (di-2-ethylhexyl phthalate) in a human subject was recovered in the urine after 24 hours. In another subject, two percent of a 5g dose was recovered in the urine after 24 hours. Wallen et al. (1974) found that a significant amount of orally administered DEHP is absorbed in the gastrointestinal tract as the intact compound. The U.S. EPA (1980) reports that Dillingham and Pesh-Imam detected nine percent of a labeled dose of DEHP that had been applied to a rabbit's skin in the urine after 24 hours. In 48 hours the level had risen to 14 percent and in 72 hours it had risen to 16-20 percent of the original dose.

The U.S. EPA (1980) reports that absorbed esters of phthalate acid esters or their metabolites distribute quite rapidly to various organs and tissues in animals and humans but accumulation apparently does not occur. Jaeger and Rubin (1970) reported on the distribution of DEHP in human tissues of two deceased patients who had received large volumes of blood stored in PVC blood bags. They detected the presence of DEHP in the spleen, liver, lung, and abdominal fat. Intravenously administered DEHP was found to disappear rapidly from the blood and within 2 hours 60-70 percent of the total dose was detected in the liver and lungs. Results from a study by Waddell et al. (1977) reveal a rapid accumulation of DEHP in the kidney and liver followed by a rapid excretion into urine, bile, and the intestine. Albro et al. (1973) concluded that the first step in the metabolism of DEHP in rats is conversion of the diester to the monoester mono-2-ethylhexyl phthalate followed by a series of oxidations. The U.S. EPA (1980) concluded from this that the same metabolism is possible for other diesters and it is possible in other animals, including man.

Illustrative of several studies, Lake et al. (1975) found that a single oral dose of DEHP was practically all excreted in the urine and feces within a 4-day period, leaving less than 0.1 percent of the DEHP in the organs and tissues.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Smith (1953) fed diets containing 0, 0.01, 0.05, 0.25, and 1.25 percent dibutyl phthalate to male Sprague-Dawley rats in groups of 10 for 1 year. One-half of all the rats receiving the highest dose died during the first week. The remaining animals all lived and exhibited no treatment-related adverse effects. Men'shikova (1971) exposed rats continuously for 93 days to chamber concentrations of 0.098, 0.256, and 0.98 mg/m^3. The only detected adverse effect was a dose-related increase in gamma globulin.

Men'shikova (1971) reported that atmospheric concentrations of 0.12 and 0.15 mg/m^3 resulted in abnormal encephalographic responses in 3 human subjects in the study. A reduced level of 0.093 mg/m^3 appeared to have no effect. In a study of 147 industrial workers (87 women and 60 men) exposed primarily to dibutyl phthalate with other esters present in lower concentration, Milkov et al. (1973) found varying degrees of toxic polyneuritis.

Teratogenic and Other Developmental Effect

In a rat teratogenic study, Singh et al. (1975) administered 0.305 ml/kg, 0.610 ml/kg, and 1.01 ml/kg dibutyl phthalate intraperitoneally to pregnant female rats on days 5, 10, and 15 of gestation. Dose-related gross and skeletal abnormalities were observed in the fetuses that included absence of tail, anophthalmia, twisted hands and legs, hematomas, elongated and fused ribs, absence of tail bones, abnormal or incomplete skull bones, and incomplete or missing leg bones. Reduced fetal weight was also observed.

Mutagenic Effects

Dibutyl phthalate did not induce mutations in *Salmonella* strains TA100 and TA98 in a modified reverse mutation plate incorporation assay at concentrations up to 1000 µg/plate in the presence or absence of 59 hepatic homogenate (Kozumbo et al., 1982). It was also negative for clastogenic activity in human leukocytes (Tsuchiya and Hattori, 1977).

However, Seed (1982) found that it was a weak direct-acting mutagen in a forward mutation assay in *S. typhimurium*. CMA (1986) also found it to be mutagenic in the mouse lymphoma forward mutation assay, but only in the presence of metabolic activation. Additionally, Ishidate and Odashima (1977) found some evidence of clastogenic activity in Chinese hamster fibroblasts.

Carcinogenic Effects

Pertinent data regarding the carcinogenicity of di-n-butyl phthalate were not found in the literature reviewed. Di-n-butyl phthalate is designated Group D (not-classified) for the weight-of-evidence category for potential carcinogens.

Ecotoxicity

LC_{50} values for 4 fish and 2 invertebrates with di-n-butyl phthalate were conducted. Values ranged from 730-6,470 µg/liter. Bluegills were the most sensitive fish and scuds the most sensitive invertebrate tested (U.S. EPA, 1980) For phthalate esters in general, acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 940 and 3 µg/liter, respectively, and is expected to be lower among more sensitive species (U.S. EPA, 1986).

The U.S. EPA (1986) reports that acute toxicity to saltwater aquatic life for phthalate esters occurs at concentrations as low as 2,944 µg/liter and is expected to be lower among more sensitive species than those tested. No data concerning the chronic toxicity of phthalate esters to saltwater aquatic life was found in the literature reviewed.

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	$1x10^{-1}$ mg/kg/day

Chronic Inhalation RfD: NA
Subchronic Oral RfD: 1 x 10^0 mg/kg/day
Subchronic Inhalation RfD: NA
MCL: NA
AWQC: Water and Fish Consumption - 34 mg/l
Fish Consumption - 154 mg/l

BIBLIOGRAPHY

Albro, P.W., et al. 1973. Metabolism of diethylhexyl phthalate by rats. Isolation and characterization of the urinary metabolites. *Jour. Chromatogr.* 76:320. (cited in AWQC, 1980)

CMA (Chemical Manufacturer's Association). 1986. *Mutagenicity of IC (di-n-butyl phthalate) in a mouse lymphoma mutation assay.* Final Report. Submitted to Hazelton Biotech Co. HB Project No. 20989. (cited in IRIS)

Ishidate, M. Jr. and S. Odashima. 1977. Chromosome tests with 134 compounds on Chinese hamster cells in vitro - A screening test for chemical carcinogens. *Mutat. Res.* 48: 337-354. (cited in IRIS)

Jaeger, R.J. and R.J. Rubin. 1970. Plasitcizers from plastic devices: Extraction, metabolism and accumulation by biological systems. *Science* 170: 460. (cited in AWQC, 1980)

Kozumbo, W.J., et al. 1982. Assessment of the mutagenicity of phthalate esters. *Environ. Health Perspect.* 45: 103-109. (cited in IRIS)

Lake, B.G., et al. 1975. Studies on the hepatic effects of orally administered di-(2-ethylhexyl) phthalate in the rat. *Toxicol. Appl. Pharmacol.* 32: 255. (cited in AWQC, 1980)

Men'shikova, T.A. 1971. Hygienic evaluation of dibutyl phthalate in relation to the use of polymeric materials for finishing living quarters on ships. *Gig. Sanit.* 36: 23. (translated title) (cited in AWQC, 1980)

Milkov, L.E., et al. 1973. Health status of workers exposed to phthalate plasticizers in the manufacture of artificial leather and films based on PVC resins. *Environ. Health Perspect. 3:* 175. (cited in AWQC, 1980)

Seed, J.L. 1982. Mutagenic activity of phthalate esters in bacterial liquid suspension assays. *Environ. Health Perspect.* 45: 11-114. (cited in IRIS)

Shaffer, C.B., et al. 1945. Acute and subacute toxicity of di-(2-ethylhexyl) phthalate with note upon its metabolism. *Jour. Ind. Hyg. Toxicol.* 27: 130. (cited in AWQC, 1980)

Singh, A.R., et al. 1974. Maternal-fetal transfer of ^{14}C-di-2-ethylhexyl phthalate and ^{14}C-diethyl phthalate in rats. *Jour. Pharmacol. Sci.* 64: 1347. (cited in AWQC, 1980)

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

Smith, C.C. 1953. Toxicity of butyl stearate, dibutyl sebacate, dibutyl phthalate, and methoxyethyl oleate. *Arch. Ind. Hyg.* 7: 310. (cited in AWQC, 1980)

Tsuchiya, K. and K. Hattori. 1977. Chromosomal Study on human leukocyte cultures treated with phthalic acid ester. *Hokkaidoritus Eisei Kenkyusho Ho.* 26:114 (Abstract). (cited in IRIS)

U.S. EPA. 1980. *Ambient Water Quality Criteria for Phthalate Esters.* EPA/440/5-80-067.

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1981. *Treatability Manual Vol. I Treatability Data.* EPA-600/2-82-001a, Office of Research and Development.

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA. 1986. *Quality Criteria for Water.* EPA/440/5-86-001.

Waddell, W.M., et al. 1977. The distribution in mice of intravenously administered ^{14}C-di-2-ethylhexyl phthalate determined by whole body autoradiography. *Toxicol. Appl. Pharmacol.* 39: 339. (cited in AWQC, 1980)

Wallen, R.F., et al. 1974. Di-(2-ethylhexyl) phthalate (DEHP) metabolism in animals and post-transfusion tissue levels in man. *Bull. Parenteral Drug Assoc.* 28:278. (cited in AWQC, 1980)

Windholz, M. ed. 1983. *The Merck Index, 10th ed.*

DI-N-OCTYL PHTHALATE

Use

Di-n-octyl phthalate (DOP) is used as a plasticizer in the manufacture of plastics (Sittig, 1991).

Chemical and Physical Properties

Chemical Formula: $C_6H_4\ (COOC_8H_{17})_2$

MW: 391.0
SG: 0.978
Sol. (water): 3 mg/L at 25°C

BP: 220°C at 5 mmHg
MP: 25°C
VP: <0.2 at 150°C

Fate and Transport

The fate and transport of DOP can only be inferred from data on other phthalate esters. Photolysis, oxidation, and hydrolysis are probably not significant fate processes for DOP. Volatilization does not appear to be an important fate process except during the manufacture of plastics (ICF, 1985). DOP is expected to adsorb to organic material in both soils and aquatic media. Bioaccumulation is known to occur in a variety of organisms (ICF, 1985).

Pharmacokinetics

Information regarding the pharmacokinetics of DOP was not located in the available literature.

Human Toxicity

Noncarcinogenic

Systemic Effects

DOP does not appear to be highly toxic to mice but is known to be a severe eye irritant and a mild skin irritant to rabbits (ICF, 1985). The LD_{50} value for mice was determined to be 1.3 mg DOP/kg when injected intraperitoneally for 5 days/week for 10 weeks (ICF, 1985).

Teratogenic and Other Developmental Effects

DOP was shown to be fetotoxic and teratogenic to rats. The same study indicated that rats, when intraperitoneally administered 5 g DOP/kg on days 5 to 15 of gestation, showed an increase in fetal resorption and fetal toxicity.

Mutagenic Effects

No evidence is available indicating that DOP is mutagenic.

Carcinogenic Effects

Information regarding the carcinogenicity of DOP was not located in the available literature.

Ecotoxicity

Freshwater species display seven to eight day LC_{50} values between 690 and 42,000 ug/L for DOP (ICF, 1985).

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	2.00×10^{-2} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	2×10^{-2} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

BIBLIOGRAPHY

ICF, Clement Associates, *Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites,* 1985.

Piekacz, H. Effect of dioctyl and dibutyl phthalates on the organism of rats after oral administration in prolonged experiment. II. subacute and chronic toxicity. *Rocz. Panstw. Zakl. Hig.* 22(3): 295-307. 1971 (cited as principal study for oral RfD development in HEAST).

Sittig, Marshall, *Handbook of Toxic and Hazardous Chemicals,* 1991.

U.S. EPA, *Drinking Water Regulations and Health Advisories.*

U.S. EPA, *Health Effects Assessment Summary Tables (HEAST).* FY1991.

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

PESTICIDES

BHC (Hexachlorocyclohexane)

Use

BHC, or hexachlorocyclohexane, is a broad spectrum insecticide consisting of five configurational isomers. It was produced between 1942 and 1978 when it was discovered that its insecticidal properties resided in the gamma isomer. Gamma-BHC, or Lindane, is available in the form of emulsifiable concentrates, wettable powders, dusts, crystals, and solids (EPA, 1980).

Chemical and Physical Properties

Chemical Formula: $C_6H_6Cl_6$

BHC Isomer	Melting Point (°C)	Vapor Pressure (mm Hg at 50°C)	Water Solubility (mg/l)	Solubility in Relatively Nonpolar Solvent (g/100 g ether at 20°C)
alpha	158	0.00087	10	6.2
beta	312	0.000014	5	1.8
delta	138	---	10	35.4
gamma	112.5	0.0008	10	20.8

*Source: EPA, 1980.

Fate and Transport

ICF (1985) reports that the main fate and transport of BHC in aqueous systems seems to be absorption to organic particles and transport to, and biodegradation by, anaerobic organisms. This biodegradation, however, yields other toxic products and, therefore, may not be a detoxification process. Volatilization is a major transport process in soils. Hardie (1972) reports that BHC-isomers are not susceptible to photolysis or strong acids but are with the exception of beta-BHC, dehydrochlorinated by alkalies to form primarily 1,2,4-trichlorobenzene. Mathur and Saha (1975) have demonstrated Lindane to be slowly degraded (10 percent after 6 weeks) by soil microorganisms. Several investigators cited by EPA (1980) report that microorganisms and plants are capable of isomerizing Lindane to alpha- and/or delta-BHC.

Pharmacokinetics

In general, the isomers of BHC tend to accumulate in fatty tissue. The metabolism of this compound involves primarily dehydrogenation, dehydrochlorination, and dechlorination. In mammals, including humans, common metabolites of this compound include pentachlorocyclohexanes, chlorinated benzenes and chlorinated phenols. These metabolites are usually excreted in the urine (HEEP, 1987). The isomers have also been found excreted in human milk (Herbst and Bodenstein, 1972; Welch and Findlay, 1981).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

There is data indicating that exposure of humans by inhalation to gamma-BHC can cause death (ATSDR, 1990). Exposure concentrations were not given. No information on quantified inhalation exposures was available, however, ATSDR (1990) reports that exposure to BHC, particularly gamma-BHC, via inhalation has resulted in mucous membrane irritation, abnormal ECG readings, anemia, hematological abnormalities, hepatic effects, urticaria (elevated itchy patches of skin), immunological effects, paresthesia, headache, and vertigo.

Death has also occurred in human following oral exposure to gamma-BHC (Storen, 1955).

An oral study (Shivanandappa and Krishnakumari, 1981) suggests that technical grade BHC, which is a mixture of the five isomers of this compound, may cause neurotoxic effects. In this study, rats fed 2,000 ppm showed signs of central nervous system stimulation. Signs of central nervous system stimulation were not reported in rats fed less than 1,500 ppm.

No significant compound related changes were observed in Wistar rats fed 500 and 1,000 ppm delta-BHC for up to 48 months (Ito et al., 1975). However Ito et al (1973) did observe elevation in absolute liver weights in male mice fed 500 ppm delta-BHC for 24 weeks. No effects on the liver of mice fed 100 or 250 ppm delta-BHC for 24 months were observed.

Teratogenic and Developmental Effects

Testicular atrophy has been observed in rats and mice fed technical grade BHC (HEEP, 1987). Gamma BHC has not been reported to cause developmental effects in animals. Adverse reproductive effects in male and female rats have been associated with oral exposure to gamma-BHC and beta-BHC (ATSDR, 1990).

Mutagenic Effects

No increase in the frequency of chromosomal aberrations were observed in humans exposed to BHC by inhalation in a pesticide production factory. These individuals had been exposed for 8 hours/day for a least six months (Kiraly et al., 1979).

Technical grade BHC did not induce mutations in an Ames test with *S. typhimurium* (Anderson and Styles, 1978).

Carcinogenic Effects

Gamma, alpha and beta BHC have been shown to be liver carcinogens in rats and mice. Ito et al. (1973) reported that mice exposed to beta, gamma or delta BHC alone did not develop hepatocellular carcinoma. However, when these isomers were mixed with alpha BHC, hepatocellular carcinoma was observed. In Wistar rats, oral exposure to gamma BHC for 48 weeks did not result in any identifiable carcinogenic effect (Ito et al., 1975). Mice exposed to 78 mg/kg/day in their diet for 32 weeks did show an increased incidence of malignant hepatomas (ATSDR, 1990).

Ecotoxicity

The available data suggests that the BHC isomers present acute toxicity to freshwater aquatic life at concentration as low as 100 µg/L. For saltwater aquatic life, acute toxicity occurs at concentrations as low as 0.34 µg/L. Acute toxicity tests indicate that the alpha isomer is more toxic than gamma and beta to freshwater algae. Both freshwater and saltwater algae species are more resistant to BHC than invertebrates and fish species (U.S. EPA, 1980).

Standards, Criteria, and Guidelines

alpha

EPA Carcinogenicity Class B2

Oral Slope Factor:	6.3×10^{0} (mg/kg/day)$^{-1}$
Inhal. Slope Factor:	6.30×10^{0} (mg/kg/day)$^{-1}$
Chronic Oral RfD:	NA
Chronic Inhal. RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhal. RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption: .0092 µg/l Fish Consumption: 0.031 µg/l

beta

EPA Carcinogenicity Class C

Oral Slope Factor: 1.80×10^{0} (mg/kg/day)$^{-1}$
Inhal. Slope Factor: 1.86×10^{0} (mg/kg/day)$^{-1}$
Chronic Oral RfD: NA
Chronic Inhal. RfD: NA
Subchronic Oral RfD: NA
Subchronic Inhal. RfD: NA
MCL: NA
AWQC: Water and Fish Consumption: 0.0163 µg/L
Fish Consumption: 0.0547 µg/l

delta

EPA carcinogenicity Class D

Oral Slope Factor: NA
Inhal Slope Factor: NA
Chronic Oral RfD: NA
Chronic Inhal. RfD: NA
Subchronic Oral RfD: NA
Subchronic Inhal. RfD: NA
MCL: NA
AWQC: Water and Fish Consumption: NA
Fish Consumption: NA

gamma

EPA Carcinogenicity Class B2-C

Oral Slope Factor: 1.30×10^{0} (mg/kg/day)$^{-1}$
Inhal Slope Factor: NA
Chronic Oral RfD: 3.00×10^{-4} (mg/kg/day)
Chronic Inhal. RfD: NA
Subchronic Oral RfD: 3.00×10^{-3} (mg/kg/day)
Subchronic Inhal. RfD: NA
MCL: 0.2 µg/L
AWQC: Water and Fish Consumption: 0.0186 µg/l
Fish Consumption: 0.0625 µg/l

BIBLIOGRAPHY

Anderson, D. and Styles, J.A. 1978. An evaluation of six short-term tests for detecting organic chemical carcinogens. Appendix 2. The bacterial mutation test. *Br. J. Cancer.* 37:924-930.

U.S. EPA. 1980. *Ambient Water Quality Criteria for Hexachlorocyclohexane.*

ATSDR, 1990. *Toxicological Profile for Hexachlorocyclohexane.*

Hardie, 1972. (cited in EPA, 1980)

Herbst, M. and Bodenstein, G. 1972. Toxicology of lindane. In: *Lindane*, E. Ulmann, Ed. Verlag K. Shillinger Publ. Freiburg im Breisgaw. p. 23-82. (cited in U.S. EPA, 1980a, 1985a)

ICF, Clement Associates, Inc. Chemical, Physical and Biological Properties of Compounds present at Hazardous Wastes Sites. 1985.

Ito, N., Nagasaki, H., Aoe, H., et al. 1975. Brief communication: Development of hepatocellular carcinomas in rats treated with benzene hexachloride. *J. Natl. Cancer Inst.* 54(3):801-805. (cited in IRIS)

Ito, N., Nagasaki, H., Arai, M., Sugihara, S., and Makiura, S. 1973a. Histological and ultrastructural studies on the hepatocarcinogenicity of benzene hexachloride in mice. *J. Natl. Cancer Inst.* 51(3):817-826. (cited in IRIS)

Ito, N., Nagasaki, H., Arai, M., Makiura, S., Sugihara, S., and Hirao, K. 1973b. Histopathologic studies on liver tumorigenesis induced in mice by technical polychlorinated biphenyls and its promoting effect on liver tumors induced by benzene hexachloride. *J. Natl. Cancer Inst.* 51(5):1637-1646. (cited in IRIS)

Kiraly, J., Szentesi, I., Ruzicska, M., et al. 1979. Chromosome studies in workers producing organophosphate insecticides. *Arch. Environ. Contam. Toxicol.* 8:309-319. (Cited in Toxicological Profile for alpha, beta, gamma, and delta Hexachlorocyclohexane)

Mathur and Saha, 1975. (cited in EPA, 1980)

Shivanandappa, T., Krishnakumari, M.K., and Majumder, S.K. 1982. Inhibition of steroidogenic activity in the adrenal cortex of rats fed benzene hexa chloride BHC. *Experientia (Basel).* 38(1):1251-1253.

Stoven, 1955. (cited in ATSDR, 1990)

U.S. EPA, 1987. *Health Effects and Environmental Profile for Hexachlorocyclohexane (HEEP).*

Welch, R.M. and J.W.A. Findlay. 1981. Excretion of drugs in human breast milk. *Drug Metab. Rev.* 12(2):261-278.

CHLORDANE

Use

Chlordane is currently used in the United States for termite control. Chlordane was previously used as an agricultural, home and garden pesticide and insecticide.

Chemical and Physical Properties

Chemical Formula: $C_{10}H_6Cl_8$

MW: 409.80
SG: 1.56-1.57 at 25°C
Sol (water): Insoluble

BP: 175°C at 2 mm Hg
UP: 1×10^{-5} at 25°C
Sol (organics): Soluble in most organic solvents

Fate and Transport

In outdoor air, chlordane is expected to exist in the vapor phase (Bidleman et al., 1986). In indoor air, chlordane is expected to exist in vapor form and as a constituent of dust particles. Chlordane present in water will adsorb to particles and volatilize. The rate of volatilization will be influenced by the composition of the matrix, temperature, wind and the turbulence of the water. Chlordane will bioconcentrate in both marine and freshwater species (Zaroogiean et al., 1985; Oliver and Niimi, 1985). In soil, chlordane will adsorb to organic matter and volatilize slowly. It will not leach significantly, generally remaining in the top layers of soils for as long as 20 years (Beeman and Matsamura, 1981). Chlordane degrades under natural environmental conditions to photoisomers such as photo-cis-chlordane, which are more toxic to certain species than chlordane and also show higher bioaccumulation (Khan et al., 1970).

Pharmacokinetics

Data obtained from humans exposed to chlordane via inhalation indicates that blood and tissue levels of chlordane increase with exposure duration (Taguchi and Yakushigi, 1988). Ingestion of chlordane by a 59 year old male resulted in deposition in adipose tissue, spleen, brain and liver (Kurz et al., 1983). Other reports reaffirm that this compound is absorbed from the gastrointestinal tract. Chlordane metabolites has been found in human milk (WHO, 1984).

Chlordane is absorbed through the skin and more easily through the lungs. Chlordane is degraded oxidatively to complexes which are excreted in the feces and to a lesser extent, in urine (Patty, 1978).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The fatal dose to adult humans is between 6 and 60 grams. Symptoms occur within 45 minutes to several hours after ingestion, although symptoms have occurred following very small doses either orally or by skin exposure. Symptoms include increased sensitivity to stimuli, tremors, muscular incoordination and convulsions (Gosselin et al., 1984). Princi and Spurbeck reported no illness among workers exposed to chlordane at air concentration of 5 mg/m^3 for one to three years.

Teratogenic and Developmental Effects

Usami et al. (1986) reported no malformations or fetal toxicity in pregnant rats administered chlordane by gavage. However the highest dose resulted in death in four out of eight dams. Evidence exists that suggests chlordane induces neurobehavioral toxic effects on the fetal brain following in utero exposure (Al Hachim and Al-Baker, 1973).

Ambrose (1953) reported reduced fertility in the form of a reduction in the number of mated females that delivered litters, when male and female rats were fed diets that contained chlordane. Balash et al. (1987) reported testicular degeneration in male mice exposed to chlordane for 30 days.

Mutagenic Effects

Sobti et al. reported no DNA repair in human lymphoid cells. However Ahmed et al. reported that chlordane is a genotoxicant to human fibroblasts.

Carcinogenic Effects

The NCI found that chlordane fed in the diets of mice for 80 weeks at concentrations of 30 or 56 mg/kg produced a significant incidence of liver cancer. The increased incidence of hepatocellular carcinomas was found to be dose related. Velsicol (1983) reported an increase in adenomas of the liver in male rats receiving 25 ppm for 130 weeks. Becker and Sell (1979) tested chlordane in a strain of mice known not to develop spontaneous liver tumors. Twenty-seven percent of the surviving treated mice developed hepatocellular carcinomas. None of the controls developed tumors.

Ecotoxicity

The freshwater aquatic life criteria for chlordane is 0.0043 µg/L as a 24-hour average and 2.4 µg/L, not to be exceeded at any time. For saltwater aquatic life, the 24-hour average criterion is 0.0040 µg/L, while the concentration should not exceed 0.09 µg/L at any time (EPA, 1980).

Standards, Criteria, and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	1.3×10^{0} $(mg/kg/day)^{-1}$
Inhal. Slope Factor:	1.3×10^{0} $(mg/kg/day)^{-1}$
Chronic Oral RfD:	6.0×10^{-5} mg/kg/day
Chronic Inhal. RfD:	NA
Subchronic Oral RfD:	6.0×10^{-5} mg/kg/day
Subchronic Inhal. RfD:	NA
MCL:	0.002 mg/l
AWQC:	Water and Fish Consumption:c 0.00046 µg/l Fish Consumption: 0.00048 µg/l

BIBLIOGRAPHY

Ahmed, F.E., Hart, R.W., and Lewis, N.J. 1977. Pesticide induced DNA damage and its repair in cultured human cells. *Mutat. Res.* 42:161-174.

Al-Hachim and Al-Baker, 1973. (cited in ATSDR, 1990)

Ambrose, 1953. (cited in ATSDR, 1990)

ATSDR, 1990. *Toxicological Profile of Chordane.*

Balash et al., 1987. (cited in ATSDR, 1990)

Becker, F.F. and Sell, S. 1979. Fetoprotein levels and hepatic alterations during chemical carcinogenesis in C57BL/6N mice. *Cancer Res.* 39:3491-3494.

Beeman and Matsamura, 1981. (cited in ATSDR, 1990)

Bidleman et al., 1986. (cited in ATSDR, 1990)

Gosselin et al., 1984. (cited in IRIS)

Khan et al., 1970. (cited in IRIS)

Kurz et al., 1983. (cited in ATSDR, 1990)

Oliver and Niimi, 1985. (cited in ATSDR, 1990)

Patty, F.A., 1978. Patty's Industrial Hygiene and Toxicology.

Sobti, R.C., Krishan, A., and Davies, J. 1983. Cytokinetic and cytogenetic effect of agricultural chemicals on human lymphoid cells in vitro. *Arch. Toxicol.* 52:211-231.

Taguchi and Yakushiji, 1988. (cited in ATSDR, 1990)

Usami et al., 1986. (cited in ATSDR, 1990)

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, 1980. *Ambient Water Quality Criteria for Chlordane.*

U.S. EPA, *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA, *Drinking Water Regulations and Health Advisories.*

U.S. EPA, 1991. *Water Quality Criteria Summary.*

Velsicol Chemical Co. 1983b. MRID No. 00144312. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

WHO, 1984. (cited in ATSDR, 1990)

Zaroogiean et al., 1985. (cited in ATSDR, 1990)

DDT/DDD/DDE

Background

DDT is an organic pesticide which is generally a combination of several organochlorinated pesticides. Technical grade DDT is a mixture of DDT (1,1,1 trichloro-2,2-bis(4-chlorophenyl)ethane), DDD (1,1 dichloro-2-(2-chlorophyenyl)-2(4-chlorophenyl)ethane) and DDE (1,1 dichloro-2,2-bis(4-chlorophyenyl)ethene). The latter two are frequent degradation products of DDT so the three are often detected together (ICF, 1985). DDT, DDD, and DDE are structurally similar so their behavior in the environment is expected to be comparable.

Use

DDT has been used as a plant-growth regulator and as a pesticide/herbicide.

Chemical and Physical Properties

Chemical Formulas:	DDT - $C_{14}H_9C1_5$;		
	DDD - $C_{13}H_{10}Cl_4$;		
	DDE - $C_{14}H_8Cl_4$		
MW:	DDT - 354.5;		
	DDD - 320;		
	DDE - 318	BP:	DDT - 260°C
Sol. (water):	DDT - 5.5 µg/L	MP:	DDT - 109°C
	DDD - 20 µg/L		DDD - 112°C
	DDE - 14 µg:		DDE - 90°C
Sol. (organics):	fat and organic solvents	VP:	DDT - 1.9 x 10^{-7} mmHg at 25°C
			DDD - 1.0 x 10^{-6} mmHg at 30°C
			DDE - 6.5 x 10^{-6} mmHg at 20°C

Fate and Transport

DDT is known to be very persistent in soils. It sorbs well to organic matter and is bioaccumulated by plants and animals (ICF, 1985). DDT does not leach rapidly and has a residence time of over 10 years (IARC, 1974).

In aquatic media, DDT isomers are subject primarily to photolysis. Biotransformation to bis(2-chlorophenol)methadone is usually the ultimate fate of DDT (ICF, 1985).

In the atmosphere, DDT is generally subjected to wet and dry deposition and photochemical degradation. The atmospheric half-life of DDT is estimated to be 17 days (ICF, 1985).

Pharmacokinetics

Jensen, et al. (1957) reported that rats absorb 95 percent of an ingested dose of DDT in the gastrointestinal tract and that 35 percent of the absorbed dose is retained in the body for at least 9 days. Absorption of DDT appears to follow the same pathways as fat, in that it is absorbed slowly but completely within 24 hours. DDT is stored in the adipose tissue, liver, brain and muscle tissue of most animals (IARC, 1974). DDT is known to be absorbed by humans in direct proportion to dietary exposure. The clearance half-life of DDT is 10 to 20 years (IRIS, 1990).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

A study by Laug, et al. (1950) in which weanling rats were fed commercial DDT at levels of 0, 1, 5, 10, or 50 ppm for 15 - 27 weeks revealed morphologic liver changes including increasing hepatocellular hypertrophy at dose levels of 5 ppm and above. In an earlier study, chronic oral exposure of rats to levels between 200 and 800 ppm DDT in food also revealed liver alterations. The toxic effects to the liver appear to increase with dosage and the length of exposure.

The effect of chronic exposure to humans is uncertain, based on the available literature. A single dose of 286 mg caused mild sickness in one man (ACGIH, 1984) but, Laws, et al (1967) noted that no adverse effects occurred in workers exposed to 18 mg DDT/day for 19 years (ACGIH, 1984).

Teratogenic and Other Developmental Effects

Exposure to 1 mg/kg bw on days 10, 12, and 17 of gestation caused morphological changes in the gonads of mice. In the same study, it was noted that the fertility of the female offspring was reduced (McLachlan and Dixon, 1972).

DDT is also known to be embryotoxic when administered to mice daily in a dose of 2.5 mg/kg (Schmidt, 1973).

Mutagenic Effects

Chromosomal aberrations were noted in *in vitro* kangaroo-rat and human lymphocyte cells (Palmer, et al., 1972; Lessa, et al, 1976). DDT caused an increase in dominant lethal mutations in *Drosophila melanogaster* (Clark, 1974), Swiss mice (Clark, 1974), and rats (Palmer, et al., 1972)

Carcinogenic Effects

Three studies were located that noted higher tissue concentrations of DDT in humans dying of cancer than in humans dying of other diseases (Casarett, et al., 1968; Dacre and Jennings, 1970,; Wasserman, et al., 1976). The existing human epidemiological studies on DDT are considered inadequate or inconclusive by EPA.

Liver tumors have been linked to DDT exposure in seven studies of various mouse strains and in three rat studies which looked DDT (U.S. EPA, IRIS).

Eight of nine studies performed on laboratory mice indicate that DDT causes hepatocellular adenomas and carcinomas upon ingestion and lung tumors upon inhalation (IRIS, 1990). Walker (1972) reported an increase in liver tumors in mice exposed to DDT in their food for 2 years.

DDD fed to CF-mice at 250 ppm (TWA) for 130 weeks revealed a significant increase in liver tumors (Tomatis et al., 1974). Dose-dependent incidences of hepatocellular carcinomas and hepatomas have been observed in mice and hamsters (NCI, 1978).

Ecotoxicity

DDT is known to be highly toxic to most freshwater and saltwater organisms. Acute toxicity values range from 0.18 to 1800 µg/L, with the final acute value for freshwater species being 1.1 µg/L. The saltwater final acute value for DDT isomers was 0.18 µg/L (ICF, 1985).

DDT isomers are bioaccumulated by most organisms but by fish in particular. Because of this, DDT has caused the death and decreased fecundity in many species of fish-eating birds (ICF, 1985).

Standards, Criteria and Guidelines

EPA Class B2 Carcinogens

Oral Slope Factor:	DDD - 2.4×10^{-1} (mg/kg/day)$^{-1}$
	DDE - 3.4×10^{-1} (mg/kg/day)$^{-1}$
	DDT - 3.4×10^{-1} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	DDD - NA
	DDE - NA
	DDT - 3.4×10^{-1} (mg/kg/day)$^{-1}$
Chronic Oral RfD:	DDD - NA
	DDE - NA
	DDT - 5×10^{-4} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	DDD - NA
	DDE - NA
	DDT - 5×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 2.4×10^{-5} µg/L (DDT)
	Fish Consumption - 2.4×10^{-5} µg/L (DDT)

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists. 1984. *Documentation of the Threshold Limit Values.*

Casarett, L.J., G.C. Fryer, W.L. Yauger, Jr., and H. Klemmer. 1968. Organochlorine Pesticide Residues in Human Tissue. *Arch. Environ. Health.* 17:306-311. (Cited in IRIS).

Clark, J.M. 1974. Mutagenicity of DDT in Mice, *Drosophila melanogaster* and *Neurospora crassa. Aust. J. Biol.* 27:427-440. (Cited in EPA, 1984).

Dacre, J.C. and R.W. Jennings. 1970. Organochlorine Insecticides in Normal and Carcinogenic Human Lung Tissues. *Toxicol. Appl. Pharmacol.* 17:277. (Cited in IRIS).

Jensen, J.A., et al. 1957. DDT Metabolites in Feces and Bile of Rats. *J. Agric. Food Chem.* 5:919. (Cited in EPA, 1984).

ICF, Clement Associates. 1985. Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites.

International Agency for Research on Cancer (IARC). 1975. *IARC Monographs*. Volume 5.

Laug, E.P., A.A. Nelson, O.G. Fitzhugh and F.M. Kunze. 1950. Liver Cell Alteration and DDT Storage in the Fat of the Rat Induced by Dietary Levels of 1-50 ppm DDT. *J. Pharmacol. Exp. Ther.* 98:268-273. (Cited in EPA, 1984).

Lessa, J.M.M., W. Becak, M.N. Rabello, C.A.B. Pereira and M.T. Ungaro. 1976. Cytogenic Study of DDT on Human Lymphocytes *in vitro*. *Mutat. Res.* 40:131-138. (Cited in EPA, 1984).

McLachlan, J.A., and R.L. Dixon. 1972. Gonadel Function in Mice Exposed prenatally to p,p'DDT. *Toxicol. Appl. Pharmacol.* 22:327. (Cited in EPA, 1984).

NCI (National Cancer Institute). 1978. Bioassay of DDT, TDE, and P,P'-DDE for possible carcinogenicity. NCI report No. 131. DHEW Publ. No. (NIH) 78-1386. (Cited in IRIS).

Palmer, K.A., S. Green, and M.S. Legator. 1972. Cytogenic Effects of DDT and Derivatives of DDT in a Cultured Mammalian Cell Line. *Toxicol. Appl. Pharmacol.* 7:562. (Cited in EPA, 1988).

Schmidt, R. 1973. Effect of DDT on Prenatal Development of the Mouse (Under Consideration of Distribution of Tritium-Labelled and Carbon-14-Labelled DDT in Pregnant Mice). (Ger.) *Biol. Rundsch.* 11:316-317. (Cited in EPA, 1984).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

Tomatis, L. and V. Turusov. 1975. *Studies in the Carcinogenicity of DDT.* Gann. 17:219-241. (Cited in IRIS, 1990).

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1984. *Health Effects Assessment for DDT.* EPA 540/1-86/026.

U.S. EPA. *Heatlh Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water* EPA 440/5-86-001.

Walker, A.I.T., E. Thorpe and D. E. Stevenson. 1972. The Toxicology of Dieldrin: Long-Term Oral Toxicity Studies in Mice. *Food Cosmet. Toxicol.* 11:415-432. (Cited in EPA, 1984).

Wasserman, M., D.P., Noguiera, L. Tomatis, et al. 1976. Organochlorine Compounds in Neoplastic and Adjacent Apparently Normal Breast Tissue. *Bull. Environ. Contam. Toxicol.* 15:478-484. (Cited in IRIS).

DIELDRIN

Use

Dieldrin is a manmade cyclodiene insecticide. Cyclodiene insecticides are a subgroup of chlorinated cyclic hydrocarbon insecticides which include aldrin, DDT, BHC and other insecticides. The Shell Chemical Company manufactured dieldrin in the United States until 1974 when the EPA prohibited its manufacture under the Federal Insecticide, Fungicide, and Rodenticide Act. Prior to 1974, the primary use of dieldrin was for the control of corn pests, although they were also used in the citrus industry (Sittig, 1991).

Physical and Chemical Properties

Chemical Formula: $C_{12}H_8Cl_6O$

MW: 380.93 MP: 175-176°C
SG: 1.75 VP: 3.1×10^{-6} mm Hg at 20°C
Sol. (water): 0.186 mg/l at 20°C
Sol. (organics): Soluble in common organic solvents except aliphatic petroleum solvents and methanol.

Fate and Transport

Dieldrin is a common degradation product of the insecticide aldrin in all aerobic and biologically active soils. Dieldrin sorps tightly to soils, and therefore, volatilizes slowly. ATSDR (1989) reports that several studies indicate that microbial degradation in soils is probably a minor fate process for dieldrin. Although several studies in ATSDR (1989) report that movement of dieldrin in waterborne sediments is a major fate process, leaching is minimal.

Glotfelt (1978) reports that evidence supports the view that much of dieldrin used in agriculture reaches the atmosphere. Baldwin et al. (1977), however, states that atmospheric degradation probably prevents significant accumulation of dieldrin in the air.

In aquatic systems, the presence of dieldrin in ground water is rare. In fact, Spalding et al. (1980) and Richard et al. (1975) detected no dieldrin in ground water samples. ATSDR (1989) reports that the lack of leaching of dieldrin from soils may explain its absence from ground water. However, small amounts of dieldrin have been detected in surface waters (Richard et al., 1975; Hindin et al., 1964) suggesting the potential for surface runoff of dieldrin from soils (ATSDR, 1989). Dieldrin is converted to photodieldrin, a stereoisomer, upon exposure to sunlight. It is unlikely, however, that photodieldrin occurs widely in the environment (ATSDR, 1989). Volatilization of dieldrin from water is slow. Mackay and Leinonen (1975) report that evaporation of dieldrin from a 1 m column of 25°C water had a half-life of 539 days.

ATSDR (1989) reports that bioaccumulation and biomagnification are significant fate processes in the environment. In a study by Metcalf et al. (1973), biomagnification of dieldrin was 5,957 in fish and 11, 149 in snails.

Pharmacokinetics

ATSDR (1989) reports that several studies indicate absorption of dieldrin via inhalation, oral, and dermal routes of exposure. A correlation between dieldrin treatment of homes and levels of the insecticide in human breast milk suggests absorption by the inhalation route (Stacey and Tatum, 1985). Hunter and Robinson (1967) and Hunter et al. (1969) found a dose-related increase in dieldrin blood levels following oral administration of the insecticide to human volunteers. Although ATSDR (1989) questions the accuracy, Feldmann and Maibach (1974) found 7.7 ±2.9 percent of a single dose of dieldrin applied dermally to the forearm of human volunteers was absorbed over a five-day period.

Dieldrin appears to be distributed to the blood, brain, liver, and adipose tissue in the human body. Hunter and Robinson (1967) and Hunter et al. (1969) exposed human volunteers to 0, 10, 50, or 211 μg dieldrin/man/day for 24 months. Dieldrin concentrations in the blood and adipose tissue increased in a dose-related manner reaching a steady-state concentration balanced by ingestion and elimination. In mid- and high-dose groups, blood levels increased 4-10 times compared to controls. Dieldrin was bioconcentrated 136 times in the adipose tissue relative to blood levels. Bioconcentration appears to plateau after 9-12 months (Geyer et al., 1986). Following termination of dieldrin administration, its blood level decreased exponentially with a half-life of approximately 369 days (Hunter et al., 1969). In the brain, DeVlieger et al. (1968) reports that the concentration of dieldrin is significantly greater in the white brain matter than in the gray brain matter. Hunter and Robinson (1968) report that the bioconcentration and elimination of dieldrin are related to the lipid mass of the individual. A lean person will attain a higher adipose tissue concentration but the total amount retained will be less than in an individual with increased body fat.

Although information on the metabolites of dieldrin was not available, Ackerman (1980) reports that the concentration of dieldrin in the human body reaches a maximum level at which metabolism and excretion rates equal ingestion and absorption rates. As the concentration of dieldrin in the liver increases, the metabolizing enzyme activity increases and the rate of biodegradation and excretion is enhanced. Jager (1970) and Hunter et al. (1969) report that accumulation occurs at a slower and slower rate until concentrations of dieldrin in blood and tissues reach the maximum limit.

ATSDR (1989) reports that the main route of dieldrin excretion is in the feces primarily via the bile. The insecticide can also be excreted via breast milk. Urine appears to be a minor elimination route.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

In a study by Walker et al. (1969), Carworth Farm "E" rats were exposed to dietary dieldrin concentrations of 0, 0.1, 1.0, and 10.0 ppm for 2 years. These dietary levels are approximately equal to 0, 0.005, 0.05, and 0.5 mg/kg/day. The only effects observed during the study were that the animals became irritable and exhibited tremors and occasional convulsions. At the end of the 2 years, females exposed to the higher doses had increased liver weights and liver-to-body weight ratios. Hepatic lesions characteristic of organochlorine insecticide exposure, including focal proliferation and focal hyperplasia of liver parenchymal cells, were revealed upon histopathological examinations. In a similar experiment with dogs, the same concentrations produced increased liver weights and liver-to-body weight ratios, increased plasma alkaline phosphatase, and decreased serum protein concentration.

Fitzhugh et al. (1964) observed liver enlargement and histopathology in rats exposed to 0.025 mg/kg/day dieldrin for 2 years and weight loss and convulsions in dogs exposed to 0.5 mg/kg/day dieldrin for 25 months. In another study, Walker et al. (1972) reported liver enlargement and histopathology in mice exposed to 0.015 mg/kg/day for 2 years.

Teratogenic and Other Development Effects

Mice exposed to 6.0 mg/kg/day dieldrin during days 7-16 of gestation exhibited decreased maternal weight gain. Fetotoxic effects included decreased number of caudal ossification centers and an increase in supernumerary ribs (Chernoff et al., 1975).

The U.S. EPA (1987) reports that several studies have illustrated reduced fertility and reduced offspring survival following maternal exposure to dieldrin. The lowest dietary dieldrin concentration to cause these reproductive effects was reported by Hodge et al. (1967) to be 2.5 ppm in rats.

Mutagenic Effects

U.S. EPA (IRIS) reports that several studies have indicated that dieldrin causes chromosomal aberrations in mouse cells, human lymphoblastoid cells, forward mutation ion Chinese hamster V79 cells, and unscheduled DNA synthesis in rat and human cells. In addition, Majumdar et al. (1977) reports that dieldrin was mutagenic for *S. typhimurium* with and without metabolic activation. However, U.S. EPA (IRIS) reports that reverse mutation assays with six strains of *S. typhimurium* yielded negative responses with or without metabolic activation. U.S. EPA (IRIS) also reports that several other studies produced negative responses including assays for gene conversion in *S. cerevisiae*, back mutation in *S. marcesans*, forward mutation (Gal R22 in *E. coli*), and forward mutation to streptomycin resistance in *E. coli*.

Carcinogenic Effects

In a long-term carcinogenic bioassay by the Food and Drug Administration, Davis and Fitzhugh (1962) reported a significant increase in the incidence of hepatomas following 2-year exposure of C3HeB/Fe mice to 10 ppm dieldrin. Davis (1965) reported a significant increase in the incidence of benign hepatomas and hepatic carcinomas in C3H mice orally administered 10 ppm dieldrin. Although both of these studies are compromised by poor survival rate, lack of detailed pathology, loss of a large percentage of the animals to the study, and failure to treat the data for males and females separately; a reevaluation of the histological material of both studies by Reuber in 1974 (Epstein, 1975a, b; 1976) revealed that the hepatomas were malignant and that dieldrin was hepatocarcinogenic for male and female C3HeB/Fe and C3H mice.

Numerous other studies in mice reported by the U.S. EPA (IRIS) also reveal the hepatocarcinogenic effects of dieldrin.

Walker et al. (1972) reports significant increases in the incidence of pulmonary adenomas, pulmonary carcinomas, lymphoid tumors, and "other" tumors in female CF1 mice exposed to dieldrin doses as low as 0.1 ppm for 132-140 weeks.

Ecotoxicity

The U.S. EPA (1980) reports that, in freshwater systems, the acute toxicity of dieldrin has been evaluated for species in all major functional and taxonomic classifications. The most resistant fish species is the goldfish with an LC_{50} of 41 μg/l. The most sensitive fish species is the rainbow trout with an LC_{50} of 2.5 μg/l. Invertebrate tolerance limits ranged from an LC_{50} of 39 μg/l for the stone fly to 5 μg/l for the isopod. Chronically, tolerance limits were found to be 0.22, 0.45, and 57 μg/l (LC_{50}) for the rainbow trout, guppy, and fruit fly, respectively. Bioconcentration factors cover a wide range in freshwater systems. Yearling lake trout which may not have reached steady state had a bioconcentration factor of 68,286 and U.S. EPA (1980) reports that this value may be even higher in older, larger lake trout. Freshwater plants, which demonstrated a minimum tolerance limit of 100 μg/l (LC_{50}) for 10 days, are clearly more resistant to dieldrin than freshwater animals.

In saltwater systems, tolerance limits for invertebrates ranged from an LC_{50} of 0.7 μg/l for the pink shrimp to 50 μg/l for the grass shrimp. In fish, tolerance limits ranged from a minimum of 0.9 μg/l for the American eel to 34 μg/l for the northern puffer. Chronically, mysid shrimp were found to have a tolerance limit of 0.49 - 1.1 μg/l. Bioconcentration factors ranged from 400 to 8000 for fish or shellfish.

No data on the ecotoxicity of dieldrin in terrestrial systems were available in the literature reviewed.

Standards, Criteria, and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	1.61×10^{1} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	1.61×10^{-1} (mg/kg/day)$^{-1}$
Chronic Oral RfD:	5×10^{-5} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5×10^{-5} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 7.1×10^{-5} µg/L Fish Consumption - 7.6×10^{-4} µg/L

BIBLIOGRAPHY

Ackerman, L. 1980. Humans: Overview of human exposure to dieldrin residues in the environment and current trends of residue level in tissue. *Pestic. Monit. J.* 14:64-69. (cited in ATSDR).

ATSDR (Agency for Toxic Substances and Disease Registry). 1989. *Toxicological Profile for Aldrin/Dieldrin.*

Baldwin, M.K., et al. 1977. The concentrations of aldrin and dieldrin and their photoisomers in the atmosphere. *Pestic. Sci.* 8:431-445. (cited in ATSDR).

Chernoff, N., et al. 1975. Prenatal effects of dieldrin and photodieldrin in mice and rats. *Toxicol. Appl Pharmacol.* 31:302-308. (cited in IRIS).

Davis, K.J. 1965. Pathology report on mice fed aldrin, dieldrin, heptachlor or heptachlor epoxide for two years. *Internal FED Memorandum to Dr. A.J. Lehman.* (cited in IRIS).

Davis, K.J. and O.G. Fitzhugh. 1962. Tumorigenic potential of aldrin and dieldrin for mice. *Toxicol. Appl. Pharmacol.* 4:187-189. (cited in IRIS).

DeVlieger, M. et al. 1968. The organochlorine insecticide content of human tissue. *Arch. Environ. Health.* 7:140-146 (Suppl. 4) (cited in ATSDR).

Epstein, S.S. 1975a. The carcinogenicity of dieldrin. Part 1. *Sci. Total Environ.* 4:1-52. (cited in IRIS).

Epstein, S.S. 1975b. The carcinogenicity of dieldrin. Part 2. *Sci. Total Environ.* 4:205-217. (cited in IRIS).

Epstein, S.S. 1976. Case study 5: Aldrin and dieldrin suspension based on experimental evidence and evaluation and societal needs. *Ann. N.Y. Acad. Sci.* 271:187-195. (cited in IRIS).

Feldman, R.J. and H.I. Maibach. 1974. Percutaneous penetration of some pesticides and herbicides in man. *Toxicol. Appl. Pharmacol.* 28:126-132. (cited in ATSDR).

Fitzhugh, O.G., et al. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. *Food Cosmet. Toxicol.* 2:551-562. (cited in IRIS).

Geyer, H., et al. 1986. Bioconcentration potential of organic environmental chemicals in humans. *Regul. Toxicol. Pharmacol.* 6:313-347. (cited in ATSDR).

Glotfelt, D.E. 1978. The atmosphere as a site for applied pesticides. *J. Air Pollut. Control Assoc.* 28:917-921. (cited in ATSDR).

Hindin, E., et al. 1964. Collection and analysis of synthetic organic pesticides from surface and ground water. *Residue Rev.* 7:130-156. (cited in ATSDR).

Hodge, H.C., et al. 1967. Toxicology and no-effects of level of aldrin and dieldrin. *Toxicol. Appl. Pharmacol.* 10:613-675. (cited in ATSDR).

Hunter, C.G. and J. Robinson. 1968. Aldrin, dieldrin and man. *Food Cosmet. Toxicol.* 6:253-260. (cited in ATSDR).

Hunter, C.G., et al. 1969. Pharmacodynamics of dieldrin (HEOD) ingestion by human subjects for 18-24 months, and postexposure for 8 months. *Arch. Environ. Health.* 18:12-21. (cited in ATSDR).

Hunter, C.G. and J. Robinson. 1967. Pharmacodynamics of dieldrin (HEOD) I. Ingestion by human subjects for 18 months. *Arch. Environ. Health.* 15:614-626. (cited in ATSDR).

Jager, K.W. 1970. Aldrin, Dieldrin, Endrin, and Telodrin: An Epidemiological and Toxicological Study of Long-Term Occupational Exposure. New York: *Elsevier,* pp. 121-131. (cited in ATSDR).

Mackay, D. and P.J. Leinonen. 1975. Rate of evaporation of low-solubility contaminants from water bodies to atmosphere. *Environ. Sci. Technol.* 9:1178-1180. (cited in ATSDR).

Majumdar, S.K., et al. 1977. Mutagenicity of dieldrin in the Salmonella-microsome test. *J. Hered.* 68:184-185. (cited in ATSDR).

Metcalf, R.L., et al. 1973. Model ecosystem studies of environmental fate of six organochlorine pesticides. *Environ. Health Perspec.* 4:35-44. (cited in ATSDR).

Richard, J.J., et al. 1975. Analysis of various Iowa waters for selected pesticides: atrazine, DDE, and dieldrin - 1974. *Pestic. Monit. J.* 9:117-123. (cited in ATSDR).

Spalding, R.F., et al. 1980. Pesticides in ground water beneath irrigated farmland in Nebraska, August 1978. *Pestic. Monit. J.* 4:560-566. (cited in ATSDR).

Stacey, C.I. and T. Tatum. 1985. House treatment with organochlorine pesticides and their level in milk. *Path, Western Australia Bull. Environ. Contam. Toxicol.* 35:202-208. (citeD in ATSDR).

U.S. EPA. 1980. *Ambient Water Quality Criteria for Aldrin/Dieldrin.*

U.S. EPA. 1987. Dieldrin: *Health Advisory.* Office of Drinking Water, Wash., D.C. (cited in IRIS).

U.S. EPA. 1987. *Health Effects Assessment for Dieldrin (HEA).*

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

Walker, A.J.T., et al. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-year oral exposures of rats and dogs. *Toxicol. Appl. Pharmacol.* 15:345-373. (cited in IRIS).

Walker, A.J.T., et al. 1972. The toxicology of dieldrin (HEOD). I. Long-term oral toxicity studies in mice. *Food Cosmet. Toxicol.* 11:415-432. (cited in IRIS).

ENDRIN/ENDRIN KETONE

Use

Endrin is used primarily as a pesticide agent for field crops. It is also used as a rodenticide and an avicide: Endin Ketone is a biodegradation product of endrin (ATSDR, 1990).

Chemical and Physical Properties

Endrin (no information available on endrin ketone).

Chemical Formula: $C_{12}H_8Cl_6O$

MW: 380.9
SG: 1.65 at 25°C
FP: 27°C
Sol. (water): 250 µg/liter at 25°C
Sol. (organics): Acetone, benzene, carbon tetrachloride, hexane, xylene.

MP: 226° to 230°C (decomposes)
VP: $2.7 \text{ I } 10^{-7}$ mm Hg at 25°C

Fate and Transport

Endrin persists in the environment. Volatilization from soil surfaces and surface waters and phototransformation are important transport mechanisms (Nash, 1983). The physical properties of this chemical suggest that absorption to soils and sediments is another important fate mechanism. Aquatic organisms readily bioconcentrate this chemical. Other important fate mechanisms include biotransformation and biodegradation (ICF, 1985). No information on the fate and transport of endrin ketone was available in the literature reviewed.

Pharmacokinetics

Potential routes of entry include ingestion, skin absorption, eye and skin contact. Endrin is rapidly absorbed through the skin. The main target sites of this chemical are the liver and the central nervous system (Sittig, 1991), but the heart, lungs and liver are also susceptible (ICF, 1985). Symptoms may appear between 20 minutes and 12 hours after exposure (Sittig, 1991). After absorpotion, endrin is partly retained in fatty tissues and partly excreted in urine and feces unchanged and as three metabolites (ACGH, 1980). ATSDR (1990) reports that endrin ketone is a metabolite of endrin that appears to be more toxic in rats than endrin itself. In fact, Hutson et al. (1975) reports that endrin ketone is most likely the toxic entity of endrin. No other toxicological information on endrin ketone was available in the literature reviewed and, based on the findings of Hutson et al. (1975), the toxic effects discussed below in regards to endrin may be attributable to endrin ketone as well.

Human Toxicity

Noncarcinogenic

Systemic Effects

Chronic exposure to low levels of endrin results in damage to the nervous system. Endrin can also produce reproductive disorders following chronic exposure (ICF, 1985).

Rats and mice exposed by inhalation to Endrin concentrations of 0.36 ppm seven hours each day, five days each week for 26 weeks showed no signs of intoxication or growth retardation. Two rabbits of a group of four however failed to survive under the same conditions (Treon et al., 1955).

Acute exposure effects include headache, nausea, vomiting, dizziness, tremors, loss of consciousness, rise in blood pressure, frothing of the mouth, deafness, coma and death by respiratory arrest. Doses as low as 1 mg/kg can cause symptoms. Inhalation may cause irritation of the nose and throat and sudden convulsions (Sittig, 1991).

Teratogenic and Other Development Effects

Endrin has been shown to be a potent teratogen and reproductive toxicant in experimental animals. Teratogenic effects include cleft palate, meningo encephales and fused ribs. Developmental effects include fetal mortality and growth retardation (ICF, 1985).

Mutagenic Effects

Endrin has not been shown to be a mutagenic agent. Microbial systems, with and without metabolic activation, did not produce a mutagenic response when exposed to Endrin (Moriya et al., 1983; Probst et al., 1981; Glatt et al., 1983).

Carcinogenic Effects

Human carcinogenic data is insufficient. Animal carcinogenic data is inconclusive. Reuber (1978) reported carcinogenic effects on male and female Osborne-Mendel rats exposed to 0.1 to 25 ppm Endrin in the diet. Malignant tumors of the liver were found in the 0.1 ppm dose group.

However, Deichmann et al. (1970) reported no evidence of carcinogenicity in Osborne-Mendel rats and Treon et al. (1955) reported no evidence of carcinogenicity in Carworth Farm rats when the animals were exposed to between 1 and 100 ppm Endrin in their diets.

Ecotoxicity

Endrin is toxic to aquatic organisms. Freshwater fish are generally more sensitive than fresh or marine invertebrates. LC_{50} values for saltwater organisms range from 0.037 to 14.2 μg/liter. Abnormal behavior, increased postnatal mortality and increased fetal death have been observed in animals exposed to Endrin. The freshwater acute and chronic toxicity levels are 0.18 μg/liter and 0.0023 μg/liter. For saltwater species, these levels are 0.037 μg/liter for acute toxicity and 0.0023 μg/liter for chronic toxicity (ICF, 1985).

Standards, Criteria, and Guidelines

Endrin (no standards for endrin ketone)

EPA Carcinogenicity Class D (Endrin)

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	3.0×10^{-4} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	3.0×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	2 μg/L
AWQC:	Water and Fish Consumption - 1 μg/L Fish Consumption - NA

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists, Inc. 1980. Documentation of the Threshold Limit Values, Fourth Edition.

ATSDR, 1990. Toxicological Profile for Endrin, Endrin Aldehyde. December 1990.

Dichmann, W.B., W.E. McDonald, E. Blum, et al. 1970. Tumorigenicity of aldrin, dieldrin and endrin in the albino rat. *Ind. Med.* 39:426-434 (cited in IRIS).

Glatt, H., R. Jong and F. Oesch. 1983. Bacterial mutagenicity investigation of epoxides: Drugs, drug metabolites, steroids and pesticides. *Mutat. Res.* 11:99-118 (cited in IRIS).

Hutson, D.H. et al. 1975. Detoxication and bioactivation of endrin in the rat. Xenobiotica 5:697-714. (cited in ATSDR).

ICF, Clement Associates, 1985. Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites.

Moriya, M., T. Ohta, K. Watanabe, T. Miyazwa, K. Kato and Y. Shirasu. 1983. Further mutagenicity studies on pesticides in bacterial reversion assay systems. *Mutat. Res.* 116:185-216 (cited in IRIS).

Nash, R.G. 1983. Comparative volatilization and dissipation rates of several pesticides from soil. *J. Agric. Food Chem.* 31:310-217 (cited in ICF, 1985).

Probst, G.S., K.E. McMahon, L.E. Hill, et al. 1981. chemically induced unscheduled DNA synthesis in primary rate hepatocyte cultures: A comparison with bacterial mutagenicity using 218 compounds. *Environ. Mutagen.* 3:11-32 (cited in IRIS).

Reuber, M.D., 1978. Carcinomas, sarcomas and other lesions in Osborne-Mendel rats ingesting endrin. *Exp. Cell Biol.* 46:129-145 (cited in IRIS).

Sittig, M. 1991. Handbook of Toxic and Hazardous Chemicals and Carcinogens.

Treon, J.F., F.P. Cleveland and J. Cappel. 1955. Toxicity of endrin for laboratory animals. *J. Agric. Food Chem.* 3:842-848 (cited in IRIS and Documentation of the Threshold Limit Values).

U.S. EPA. Integrated Risk Information System (IRIS).

Velsicol Chemical Corporation. 1969. MRID. No. 00030198 (cited in IRIS).

METHOXYCHLOR

Use

Methoxychlor is used as an insecticide. Ayers and Johnson (1976) report that 1.5 million kg of methoxychlor was consumed domestically in 1974. Of this, 0.454 million kg were used agriculturally (alfalfa, deciduous fruit/nuts, soybeans, vegetables), and 1.09 million kg was used non-agriculturally (forests, home and garden, ornamentals, livestock/poultry, commercial, household and industrial establishments). The USDA (1978) reports that in 1976, 0.652 million kg were used agriculturally and 1.4 million kg were used as a livestock insecticide.

Chemical and Physical Properties

Chemical Formula: $C_{16}H_{15}Cl_3O_2$

MW: 345.65
MP: 78-78.2°C or 86-88°C
SG: 1.4 mm Hg at 25°C
Sol. (water): 0.1-0.12 mg/l at 25°C
Sol. (organics): Soluble in acetone, methylene chloride, dichlorobenzene, cyclohexanone, xylene, methylated napthalenes, carbon tetrachloride; slightly soluble in methanol and odorless kerosene.

Fate and Transport

Atmospherically, photolysis and volatilization appear to be important fate processes. Ginsburg (1953) reports that a dry, thin film of methoxychlor, when exposed to sunlight, "readily decomposes." After 12 days, 40 percent of a 10 mg sample and 91.4 percent of a 5 mg sample disappeared. Since the total amount lost from each sample is equivalent and no dark controls were run, volatilization rather than photolysis is the probable cause. Both MacNeil et al. (1972) and Zepp et al. (1976) report the presence of 1,1-bis-(*p*-methoxyphenyl)-2,2-dichloroethylene (DMDE) after the photolysis of methoxychlor.

In aquatic media, hydrolysis may be a significant fate process for methoxychlor. In a study by Wolfe et al. (1977), the hydrolysis half-life for methoxychlor was 367 days at pH 7.0. Anisoin and anisil are the major hydrolysis products. Wolfe et al. (1976) noted, however, that degradation of methoxychlor in the environment was too fast to be accounted for by hydrolysis and another process (probably biodegradation) is, therefore, dominant. Photolysis appears to be a significant fate process for methoxychlor in waters. Seba and Corcoran (1969) report that this fate process is environmentally significant because water-insoluble organics such as methoxychlor are thought to concentrate in organic surface films. The major photoproducts of methoxychlor include MDCO, DMDD, DMDE, and *p*-methoxyphenol. Indirect photolysis may also play an important role in determining the fate of methoxychlor in aquatic media (EPA, 1984). However, EPA (1984) reports that, given the low water solubility of methoxychlor and its photo-products, these compounds would likely sorb to sediments and be unavailable for photolysis. EPA (1984) reports that methoxychlor is expected to volatilize from water.

In soils, the EPA (1984) reports that hydrolysis, photolysis, volatilization, leaching, and microbial degradation will all play a role in the fate of methoxychlor. Photolysis will occur only in the surface soil and, therefore, will probably not be a dominant process (EPA, 1984). The likelihood and extent of hydrolysis will depend on soil moisture (EPA, 1984).

Pharmacokinetics

No data on the absorption of methoxychlor were available in the literature reviewed. EPA (1984) reports that methoxychlor does not accumulate to any appreciable extent in the tissue of animals. For example, Adams et al. (1974) detected no methoxychlor in the liver or adipose tissue of adult rates or in the carcass, brain, or liver of weanling rates of any of four generations maintained on diets containing 0.078-0.116 ppm methoxychlor. EPA (1984) reports that low levels of methoxychlor were detected in the fat, kidney, liver, and brain of dogs fed 20-300 mg methoxychlor/kg bw/day for 1 year. EPA (1984) reports on studies which reveal that methoxychlor is metabolized in the liver. Metabolites include phenolic compounds and organic chlorine. Kapoor et al. (1970) found 98.3 percent of orally-administered methoxychlor (50 mg/kg bw) in the urine and feces (as methoxychlor or its metabolites) within 24 hours. The feces contained 90 percent and the urine, 10 percent, of the excreted materials. Weikel (1957) found approximately 50 percent of administered methoxychlor (3 mg/kg bw) in the feces within 4 days of intravenous injection. Urinary elimination accounted for 5-10 percent of excretion.

Human Toxicity

Noncarcinogenic

Systemic Effects

EPA (1984) reports that, of several studies of subchronic oral exposure of methoxychlor to rats, dogs, swine, monkeys, and humans, the most common observed effect was reduced growth rate. A study by Davison and Cox (1976) with rats revealed that factors, in addition to reduced food consumption, contribute to the reduced growth rate at high dietary levels (10,000 mg/kg diet) while at lower dietary levels, reduced food consumption appears to be the only contributing factor.

Chronically, EPA (1984) reports on studies with rats and mice revealing reduced body weight gain as the only adverse effect. Stein (1968) reports that high daily doses of methoxychlor in dogs resulted in clinical signs of liver damage and behavioral changes. Stein (1968) also reports loss of body weight, some estrogenic effects, and increased BUN in swine receiving high daily doses.

Available information on the toxicity of methoxychlor by inhalation or dermal contact is unclear and complicated by the use of toxic exposure vehicles (i.e., benzene).

Teratogenic and Developmental Effects

Numerous studies have been performed on the teratogenicity of methoxychlor. Kincaid Enterprises (1986) observed excessive loss of litters in young New Zealand white rabbits exposed to 35.5 or 251 mg/kg/day on days 7-19 of gestation. An unusually high incidence of lung agenesis in fetuses was also noted. E.I. duPont de Nemours and Co., Inc. (1978) observed reduced body weight gain, reduced food consumption, increased postimplantation loss, and a decreased number of live fetuses per dam in ChR-CD albino rats exposed to 25 or 62.5 mg/kg/day on days 6-15 of gestation. A significant increase in wavy ribs was also observed in fetuses at these doses. Sprague-Dawley rats exposed on days 6-15 of gestation demonstrated an increased number of resorptions, dead fetuses, and increased post-implantation loss at concentrations above 138.4 mg/kg/day. At 346 mg/kg/day, delayed ossification of skull bones and sternebrae and reduced fetal body weight were observed. Doses as low as 34.6 mg/kg/day resulted in increased incidence of hydroenphrosis, reduced or no ossification of skull bones, sternebrae, and vertebrae as well as wavy ribs (Chemical Formulators, Inc., 1976). In a three-generation study with male and female ChR-CD rates, E.I. duPont de Nemours and Co., Inc. (1966) observed reduced fertility index, reduced litter size, and reduced viability index at 1000 ppm.

Mutagenic Effects

Probst et al. (1981) reports negative results in bacteria, yeast, in assays of methoxychlor-induced DNA damage, or in assays of unscheduled DNA synthesis in mammalian cell cultures with or without activation. Dunkel et al. (1981) observed a weakly positive increase in a transformation study using BALB/3T3 cell line.

Carcinogenic Effects

In seven studies on the carcinogenicity of methoxychlor reviewed by EPA (IRIS), none showed a statistically significant increase in carcinomas with doses as high as 2000 ppm in the diet.

Ecotoxicity

EPA (1984) reports that acute toxicity information is available for at least 27 freshwater invertebrates, 20 freshwater fishes, 6 marine invertebrates and 9 marine fishes. The acute LC_{50} for freshwater invertebrates and freshwater fishes ranged from 0.5-63 µg/l and 7.5-230 µg/l, respectively. In marine animals, LC_{50} values ranged from 3.5-300 µg/l for invertebrates and from 3.5-150 µg/l for fishes. EPA (1984) reports that of the 62 tested species, all but 5 have LC_{50} values less than 100 µg/l. Johnson and Finley (1980) report on the effects of temperature, pH, and water hardness on the acute toxicity of methoxychlor to freshwater animals. No changes were reported other than a slight decrease in toxicity to rainbow trout and bluegills with increased temperature. Sebastien and Lockhart (1981) report that an emulsifiable formulation of methoxychlor was more acutely toxic than a particulate formation to midge larvae, stonefly larvae, and fish. Blackfly larvae, probably related to the fact that they are filter feeders, experienced equal toxicity with either formulation.

Chronically, EPA (1984) reports that exposure for up to 42 days resulted in LC_{50} values ranging from 0.21-1.30 µg/l for 5 freshwater arthropods. Stoneflies and snails, however, were unaffected by 28 days of exposure to 4.2 µg/l. Chronically toxic concentrations ranged from 0.625-23 µg/l for freshwater and marine fishes, and from 0.05-62 µg/l for marine invertebrates. The dungeness crab was the most sensitive species, displaying increased mortality and delayed molting of larvae at 0.05 µg/l.

100 µg/l methoxychlor inhibited the growth of the freshwater algae, *Chlorella pyrenoidosa* (Kricher et al., 1975) and three of five species of marine algae (Sikka and Rice, 1974). Butler (1963) found that 1000 µg/l methoxychlor reduced ^{14}C uptake by mixed algae cultures, but 100 µg/l did not. Wurtsbaugh and Apperson (1978) and Poorman (1973) found methoxychlor to be stimulatory to some algae at concentrations of 20 µg/l and 100 mg/l, respectively.

Standards, Criteria, and Guidelines

EPA Class D carcinogen.

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	5.00×10^{-3} mg/kg-day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5.00×10^{-3} mg/kg-day
Subchronic Inhalation RfD:	NA
MCL:	0.04 mg/l
AWQC:	Water and Fish Consumption: 100 µg/l Fish Consumption Only: NA

BIBLIOGRAPHY

Adams, M., F.B. Coon and C.E. Poling. 1974. Insecticides in the Tissues of Four Generations of Rats Fed Different Dietary Fats Containing a Mixture of Chlorinated Hydrocarbon Insecticides. *J. Agric. Food Chem.* 22(1):69-75 (cited in HEEP).

Ayers, J.H. and O.H. Johnson. 1976. Chemical Economics Handbook. Stanford Research Institute, Menlo Park, CA. pp. 573.3002Q, 573.3007E (cited in HEEP).

Butler, P.A. 1963. Commercial Fisheries Investigations. *Pest. Wildlife Stud.*, Fish Wildlife Serv. Ctr. Co. 167:11-25 (cited in Smith et al., 1981; cited in HEEP).

Davison, K.L. and J.H. Cox. 1976. Methoxychlor Effects on Hepatic Storage of Vitamin A in Rats. *Bull. Environ. Contam. Toxicol.* 16(2):145-148 (cited in HEEP).

EPA. 1984. Health and Environmental Effects Profile for Methoxychlor. September, 1984 (HEEP).

EPA. Health Effects Assessment Summary Tables (HEAST).

EPA. Integrated Risk Information System (IRIS).

Ginsburg, J.M. 1953. Rate of Decomposition of the Newer Insecticides when Exposed Outdoors to Direct Sunlight. *Proc. 40th Annual Meeting of the NJ Mosquito Extermination Assoc.* 40:163-168 (cited in Gardner and Bailey, 1975; cited in HEEP).

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Resource Publ. 137. U.S. Dept. Int., Fish and Wildl. Serv. Washington, DC. p. 98 (cited in HEEP).

Kapoor, I.P., R.L. Metcalf, R.F. Mystrom and G.K. Sangha. 1970. Comparative Metabolism of Methoxychlor, Methiochlor and DDT in Mouse, Insects and in a Model Ecosystem. *J. Agric. Food Chem.* 18:1145-1152 (cited in HEEP).

Kricher, J.C., J.C. Urey and M.L. Hawes. 1975. Effects of Mirex and Methoxychlor on the Growth and Productivity of *Chlorella pyrenoidosa*. *Bull. Environ. Contam. Toxicol.* 14(5):617-620. (CA 84: 55049n, 1975) (cited in Smith et al., 1981; cited in HEEP).

MacNeil, J.D., R.W. Frei, S. Safe, and O. Hutzinger. 1972. Electron-Donor-Acceptor complexity Reagents in the Analysis of Pesticides. V. Analysis of Pesticide Decomposition Products via Electron-Donor-Acceptor Complexes. Photolysis of Methoxychlor. *J. Assoc. Off. Anal. Chem.* 55(6):1270-1275 (cited in HEEP).

Poorman, A.L. 1973. Effects of Pesticides on *Euglena gracilis*. I. Growth Studies. *Bull. Environ. Contam. Toxicol.* 10:25-28 (cited in Butler et al., 1975; cited in HEEP).

Seba, D.B. and E.F. Corcoran. 1969. Surface Slicks as Concentrators of Pesticides in the Marine Environment. *Pestic. Monit. J.* 3:190-193 (cited in HEEP).

Sebastien, R.J. and W.L. Lockhart. 1981. The Influence of Formulation of Toxicity and Availability of a Pesticide (Methoxychlor) to Black Fly Larvae (*Diptera:Simuliidae*), Some Nontarget Aquatic Insects and Fish. *Can. Entomol.* 113(4):281-293 (cited in HEEP).

Sikka, H.C. and C.P. Rice. 1974. Effects, Uptake and Metabolism of Methoxychlor in Marine Algae. *In*: Interactions of Selected Pesticide with Marine Microorganisms. p. 33-47. Research Report to the Office of Naval Research (cited in Smith et al., 1981; cited in HEEP).

Stein, A.A. 1968. Comparative Methoxychlor Toxicity in Dogs, Swine, Rates, Monkey and Man. *Ind. Med. Surg.* 37:540-541 (cited in HEEP).

USDA. (U.S. Department of Agriculture.) 1978. Farmers Use of Pesticides, 1976. USDA, Agricultural Economic Report 418, Washington, DC (cited in HEEP).

Weikel, J.H., Jr., 1957. The Metabolism of Methoxychlor (1,1,1-trichloro-2,2-bis(*p*-methoxyphenyl)ethane. I. The Role of the Liver and Biliary Excretion in the Rat. *Arch. Intern. Pharmacodyn.* 110:423-432 (cited in HEEP).

Wolfe, N.L., R.G. Zepp, G.L. Baughman, R.C. Fincher and J.A. Gordon. 1976. Chemical and Photochemical Transformation of Selected Pesticides in Aquatic Systems. NTIS PB-258846 (cited in HEEP).

Wolfe, N.L., R.G., Zepp, G.L. Baughman, and R.C. Hollis. 1977. Methoxychlor and DDT Degradation in Water: Rates and Products. *Environ. Sci. Technol.* 11:1077-1081 (cited in HEEP).

Wurtsbaugh, W.A. and C.S. Apperson. 1978. Effects of Mosquito Control Insecticides on Nitrogen Fixation and Growth of Blue-Green Algae in Natural Plankton Associations. *Bull. Environ. Contam. Toxicol.* 19(6):641-647 (cited in Smith et al., 1981; cited in HEEP).

Zepp, R.G., N.L. Wolfe, J.A. Gordon and R.C. Fincher. 1976. Light-Induced Transformations of Methoxychlor in Aquatic Systems. *J. Agric. Food Chem.* 24(4):727-733 (cited in HEEP).

INORGANICS

ALUMINUM

Use

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

Chemical and Physical Properties

Chemical Formula: Al

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

Fate and Transport

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

Pharmacokinetics

Little information on aluminum pharmacokinetics was available.

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

Human Toxicity

Noncarcinogenic

Systemic Effects

Fibrotic lung disease and severe and fatal lung damage have been observed in workers exposed to dust of aluminum metal (EPA, 1977). Aluminum is suspected of inducing neurotoxic effects, characterized by gradual loss of motor, speech, and cognitive functions. Another target organ for aluminum toxicity is the bone. Low bone formation or osteomalacia has been linked to aluminum exposure. A form of anemia, which is not related to iron deficiency, has also been linked to aluminum exposure (EPA, 1992). Aluminum particles deposited in the eye may cause necrosis of the cornea (Sittig, 1991).

Teratogenic and Developmental Effects

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

Mutagenic Effects

No information was available in the literature.

Carcinogenic Effects

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

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

Ecotoxicity

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

Standards, Criteria and Guidelines

EPA Class D Carcinogen

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

MCL: NA
AWQC: NA

BIBLIOGRAPHY

Anderson, H., B.E. Dahlberg, K. Magnus and A. Wannag. 1982. Risk of cancer in the Norwegian aluminum industry. *Int. J. Cancer.* 29:295-298 (cited in U.S. EPA, 1992).

Freeman, R.A., and W.H. Everhart. 1971. Toxicity of aluminum hydroxide complexes in neutral and basic media to rainbow trout. *Trans. Am. Fish Soc.* 100(4):644-658 (cited in U.S. EPA, 1980).

Furst, A. 1971. Trace elements related to specific chronic diseases: Cancer. *Geol. Soc. Am. Memoir.* 21:475-480 (cited in U.S. EPA, 1992).

Milham. S. 1979. Mortality in aluminum reduction plant workers. *J. Occupat. Med.* 21:475-480 (cited in U.S. EPA, 1992).

Muller, G., V. Bernuzzi, D. Desor, M-F. Hutin, D. Burnel, and P.R. Lehr. 1990. Developmental alterations in offspring of female rats orally intoxicated by aluminum lactate at different gestation periods. *Teratol.* 38:253-261 (cited in U.S. EPA, 1992).

Ondreika, R. Ginter, E. and Kortus, J. 1966. *Brit. J. Ind. Med.* 23, 305-312 (cited in Toxicology of Metals - Volume II).

Sittig, Marshal. 1991. Handbook of Toxic and Hazardous Chemicals and Carcinogens.

Sorensen, J.R.J., Campbell, I.R., Tepper, L.B. and Ling, R.D. 1974. *Environ. Health Perspect.* 7, 3-95 (cited in Toxicology of Metals - Volume II).

U.S. EPA. 1977. Toxicology of Metals - Volume II. EPA-600/1-77-022.

U.S. EPA. 1980. Chemical Hazard Information Profiles. EPA-560/11-80-011.

U.S. EPA. 1992. Memorandum from J. Dollarhide, Chemical Mixtures Assessment Branch to M. Walters, U.S. EPA Region II, on the toxicity and carcinogenicity of aluminum.

ANTIMONY

Use

Antimony is widely used as an alloy constituent in pewter and white metal, and is used in the manufacture of storage battery plates, solder, and ammunition because of its strength and its resistance to corrosion (Sittig, 1991). It is used as a fire-retardant in textiles and is used to dye steel, aluminum, pewter, and zinc (Sittig, 1991). One compound, antimony potassium tartrate, is used in medicine and as a leather mordant (ACGIH, 1984).

Chemical and Physical Properties

AG:	121.75	BP:	1750°C
SG:	6.684 at 25°C	MP:	630.74°C

Sol.(water): Insoluble
Sol.(organics): Insoluble

Fate and Transport

Antimony is present naturally in water bodies as antimony oxide. Antimony oxide is generally reduced to stibine (SbH_3) in benthic sediments. Stibine is highly volatile and is very soluble in water but, in aerobic environments, it is rapidly oxidized to Sb_2O_3. In anaerobic waters, antimony compounds are quite soluble and, when present in rivers and lakes, they rapidly transport to oceans (ICF, 1985). Antimony is known to sorb to clays and minerals so, in soils, antimony would be expected to remain stable. Particulate antimony compounds are known to transport well in the atmosphere (ICF, 1985).

Pharmacokinetics

No pertinent information was located regarding the pharmacokinetics of antimony. It appears as though antimony primarily effects the lungs upon inhalation. Ingestion of antimony leads to kidney and liver damage (ACGIH, 1984) suggesting absorption occurs in these organs.

Human Toxicity

Noncarcinogenic

Systemic Effects

Schroeder et al. (1970) reported that rats administered 5 ppm potassium antimony tartrate in water exhibited reduced lifespans and altered blood chemistries; no increased incidence in tumors was seen.

One study reported that, of 125 workers employed in the abrasives industry, 6 died suddenly and two died of chronic heart disease. Upon examination of 75 of the workers, 37 exhibited EKG problems, 14 had high blood pressure, and 7 had ulcers (ACGIH, 1984). Ambient air levels were found to range from 3 to 5 mg/m^3. These problems were confirmed to be a result of antimony exposure when rats, rabbits, and dogs were exposed to similar concentrations in the air (3.7 to 5.6 mg/m^3). Cardiac dysfunction and parenchymatous degeneration of the myocardium were noted in all species. Chronic inhalation of antimony trioxide caused severe pneumonitis in guinea pigs (ACGIH, 1984).

Teratogenic and Other Developmental Effects

Human case studies suggest that antimony may cause an increase in spontaneous abortions and several other gynecological disorders (ICF, 1985). Decreased weight gain was observed in babies born to mothers exposed to antimony compounds (ICF, 1985).

Mutagenic Effects

Several bacterial studies indicate that antimony compounds are mutagenic (ICF, 1985).

Carcinogenic Effects

Antimony has been shown to increase lung cancer among exposed workers. An inhalation study performed on rats indicated that antimony trioxide increases the risk of lung and liver tumors (ICF, 1985). The number of studies performed, however, has been inadequate to categorize antimony as a carcinogen. EPA has not evaluated antimony for evidence of human carcinogenic potential.

Ecotoxicity

LC$_{50}$ values for the freshwater species, *Daphnia magna,* and the fathead minnow, range between 9,000 and 21,900 mg/l. No detectable bioconcentration of antimony was noted in bluegill (ICF, 1985).

No data regarding toxicity of antimony to terrestrial species other than laboratory species were located in the available literature.

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor: NA
Inhalation Slope Factor: NA
Chronic Oral RfD: 4 x 10^{-4} mg/kg/day
Chronic Inhalation RfD: NA
Subchronic Oral RfD: 4 x 10^{-4} mg/kg/day
Subchronic Inhalation RfD: NA
MCL: 0.006 mg/l
AWQC: Water and Fish Consumption - .15 mg/l
Fish Consumption - 45 mg/l

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists. 1984. *Documentation of the Threshold Limit Values.*

ICF Clement. 1985. *Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites.*

Schroeder, H.A., M. Mitchner, and A.P. Nasor. 1970. Zirconium, Niobium, Antimony, Vanadium, and Lead in Rats: Life Term Studies. *J. Nutr.* 1000:59-66. (Cited in IRIS).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water* EPA 440/5-86-001.

ARSENIC

Use

Arsenic can be found in the environment in four valence states (-3, 0, +3, +5) and is used industrially in the form of arsenic disulfide, arsenic pentoxide, arsenic trichloride, arsenic trisulfide and lead arsenate, but primarily as arsenic trioxide. Elemental arsenic is a shiny, gray element that possesses both metallic and non-metallic properties. It is present naturally in the environment at low concentrations and is used industrially as arsenic trioxide, in pigment production, glass manufacturing, textile printing, tanning, and in antifouling paints. As arsenic trichloride, it is used in the manufacture of pharmaceuticals (Sittig, 1991).

Metallic arsenic is used as an alloying agent in the smelting of copper, zinc and lead ores.

Chemical and Physical Properties

AW: 74.91	BP: 613°C
SG: 5.72 at 20°C	MP: 817°C
	VP: 1 mmhg at 372°C

Sol. (water): insoluble (except for some salts).

Fate and Transport

Arsenic is generally quite mobile in the environment although, because it occurs in four valence states, it cannot be characterized easily. The most common fate processes of arsenic in the environment are speciation between the +3 and +5 valence states, volatilization, sorption, and biotransformation (EPA, 1984).

In surface waters, arsenic is significantly influenced by the presence of biota. Arsenic is readily bioaccumulated but is often biotransformed to methylated arsenicals, volatile compounds that evaporate from surface waters (EPA, 1985).

In surface soils, arsenic is known to sorb to clays, iron oxides, and particulate matter. The presence of these materials would greatly retard arsenic's leachability (EPA, 1984). In soils with low sorptive capacity, arsenic will leach into ground water, where it would likely be transported readily.

The primary means of removal of atmospheric arsenic are wet and dry precipitation (EPA, 1984).

Pharmacokinetics

Soluble arsenic salts are known to be easily absorbed through the gastrointestinal lining in humans and animals (Coulson, et al., 1935). In humans, peak blood arsenic levels (98 percent of total arsenic ingested) were reached after only 24 hours following the ingestion of

8.25 mg As in three doses (EPA, 1985). Arsenic is distributed, in humans, primarily to the nails, hair, bone and skin, and to a lesser extent, the heart, liver, kidneys and lungs (Kadowski, 1960).

In laboratory animals, arsenic was shown to distribute to the liver, kidneys, lung, spleen, skin, and brain. It is removed rapidly from all organs except for the latter two (EPA, 1985).

Arsenic generally is metabolized to methylated arsenicals such as monomethyl and dimethyl arsenic. Buchet, et al. (1981) reported that 25 percent of arsenic, administered as arsenate to human volunteers, was excreted in the urine as inorganic arsenic, 25 percent as monomethyl and 50 percent as dimethyl arsenic.

Lanz, et al. (1950) noted that, in contrast to a humans metabolic processes, rats retain arsenic in their red blood cells for as long as 180 days. Humans typically remove 90 percent of ingested arsenic within 4 days.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Arsenic is known to be highly toxic to humans. Subchronic exposure of infants to 3 mg/day arsenic in contaminated milk caused several deaths, according to Hamamoto (1955). Oral exposure to 50 to 300 mg of inorganic arsenic was the probable cause of death to several workers, according to Vallee, et al. (1960). From these two case studies, a subacute lethal dose of 0.6 mg/kg/day was estimated for humans (ATSDR, 1989).

Oral exposure of humans to arsenic is known to cause nausea, vomiting, diarrhea, and other gastrointestinal disorders (ATSDR, 1989). Long-term exposure results in paresthesia, weakness, anorexia, bronchitis, and various skin disorders (EPA, 1985). It was reported that children exposed to 0.8 mg/L arsenic in drinking water exhibited evidence of myocardial infarction and arterial thickening (ATSDR, 1989). In Taiwan, chronic exposure to arsenic in drinking water was thought to cause gangrene in the feet and toes in 0.9 percent of the population ("Blackfoot disease"). Concentrations were reported to average 0.5 mg/L arsenic (Tseng, 1977; Tseng et al., 1968).

Exposure to arsenic doses ranging from 2.8 to 5.7 mg/kg/day in newborn Rhesus monkeys caused death in 75 percent of monkeys in the 5.7 mg/kg/day group and death in two of the seven monkeys in the 2.8 mg/kg/day group. Death was attributed to hemorrhaging, edema, and necroses of the brain (EPA, 1985). All of the surviving monkeys had normal cardiovascular and neurological function.

Teratogenic and Other Developmental Effects

Parenteral administration of 10 to 45 mg/kg/day of sodium arsenate to pregnant rats, mice, and hamsters has been reported to increase the frequency of fetal malformations (ATSDR, 1989). Arsenic has also been shown to be teratogenic when administered orally. Hood, et al. (1977) found that a single gavage dose of 29 mgAs^{+5}/kg administered to pregnant mice on day 9, 10 or 11 of gestation resulted in death or resorption of 17-26 percent of the fetuses. Of the live fetuses, 10-16 percent were below average in weight and 1-3 percent were severely malformed.

Mutagenic Effects

Arsenic is known to cause DNA fragmentation and sister chromatid exchange in several cell types in laboratory animals and humans (ATSDR, 1989).

Carcinogenic Effects

Arsenic is classified by EPA as a Class A carcinogen, a known human carcinogen. Oral exposure to elevated levels of arsenic unequivocally increases the risk of skin cancer. Tseng, et al. (1968) and other researchers noted a significant increase in several skin cancer types in populations exposed to elevated arsenic levels in the drinking water (ATSDR, 1989).

Numerous studies of smelter workers have indicated that occupational exposure to arsenic is directly associated with lung cancer (IRIS, 1990). Matanoski, et al. (1981) reported that residents surrounding a pesticide manufacturing plant were at a greater risk of contracting lung cancer than the normal population.

In a supplemental paper, Tseng reported a significant increase in the incidence of bladder, lung, kidney, and colon cancer in a Taiwanese population exposed to elevated arsenic levels in their drinking water.

All evidence from human case studies indicates that chronic exposure to arsenic causes cancer. In laboratory studies, however, attempts to induce cancer in animals have been inconclusive or negative (ATSDR, 1989). Some studies, in which the arsenic retention time has been artificially increased, have shown that arsenic will produce tumors in rats (ATSDR, 1989).

Ecotoxicity

Arsenic compounds are acutely toxic to both freshwater and saltwater species of organisms, with early life stages being the most susceptible (ICF, 1985). Toxicity can occur at levels as low as 40 µg/l in juvenile aquatic species. Saltwater fish species are susceptible to arsenic's toxic effects at levels around 15 mg/l, but some invertebrates are affected at around 508 µg/l (ICF, 1985).

Information pertaining to arsenic's toxicity to terrestrial species (other than laboratory animals) was not located in the available literature.

Standards, Criteria and Guidelines

EPA Class A Carcinogen

Oral Slope Factor:	2.00×10^0 (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	1.51×10^1 (mg/kg/day)$^{-1}$
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	3.0×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.05 mg/L
AWQC:	Water and Fish Consumption - 0.0022 μg/L Fish Consumption - 0.018 μg/L

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists. 1984. *Documentation of the Threshold Limit Values.*

Agency for Toxic Substance and Disease Registry (ATSDR). 1989. *Toxicity Profile for Arsenic.*

Buchet, J.P., R. Lauwerys, and H. Roels. 1981. Comparison of the Urinary Excretion of Arsenic Metabolites After a Single Oral Dose of Sodium Arsenite, Monoethyl Arsonate or Dimethyl Arsenate in Man. *Int. Arch. Occup. Environ. Health.* 48:71-79. (Cited in EPA, 1985).

Coulson, E.J., et al. 1935. Metabolism in the Rat of the Nautrally Occurring Arsenic of Shrimp as Compared with Arsenic Trioxide. *J. Natr.* 10:255. (Cited in EPA, 1985).

Hood, R.D., G.T. Thacker, and B.L. Patterson. 1977. Effects in the Mouse and Rat of Prenatal Exposure to Arsenic. *Environ. Health Perspect.* 19:219-222. (Cited in EPA, 1985).

Enterline, P.E. and G.M. Marsh. 1982. Cancer among workers exposed to arsenic and other substances in a copper smelter. *Am. J. Epidemio.* 116:895-911. (cited in HEAST).

ICF, Clement Associates. 1985. *Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites.*

Kadowski, K. 1960. Studies on the Arsenic Contents of Organ Tissues of the Normal Japanese. *Osaka Med. Jour.* 9:2083. (Cited in EPA, 1985).

Lanz, H. Jr., et al. 1950. The Metabolism of Arsenic in Laboratory Animals Using AS^{74} as a Tracer. *Univ. Calif. Publ. Pharmacal.* 2:263. (Cited in EPA, 1985).

Matanoski, G.E. Landau, J. Tonascia, C. Lazar, E. Elliot, W McEnroe, and K. King. 1981. Cancer Mortality in an Industrial Area of Baltimore. *Environ. Res.* 25:2-8. (Cited in IRIS)

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

Tseng, W.P., et al. 1968. Prevalence of Skin Cancer in an Endemic Are of Chronic Arsenicism in Taiwan. *J. Natl. Cancer Inst.* 40:453-463. (Cited in IRIS).

Tseng, W.P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. *Environ. Health Perspec.* 19:109-119. (cited in IRIS).

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1985. Office of Drinking Water. *Health Advisory for Arsenic.*

U.S. EPA. 1985. *Health Effects Assessment for Arsenic.*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* EPA 440/5-86-001.

BARIUM

Use

Barium, a silver white metal, is produced by reduction of barium oxide. In its metallic state, it is used for the removal of residual gas in vacuum tubes and in alloys with nickel, lead, calcium, magnesium, sodium, and lithium.

Barium compounds are used in the manufacture of a variety of products including lithopone (a white pigment in paints), chlorine, sodium hydroxide, valves, and green flares. They are used in synthetic rubber vulcanization, x-ray diagnostic work, glassmaking, papermaking, beet-sugar purification, and animal and vegetable oil refining. They can be found in use in the brick and tile, pyrotechnics, and electronics industries. These compounds are found in lubricants, pesticides, glazes, textile dyes and finishes, pharmaceuticals, and in saltwater cements. Barium is used as a rodenticide, a flux for magnesium alloys, a stabilizer and mold lubricant in the rubber and plastics industries, an extender in paints, a loader for paper, soap, rubber, and linoleum. It is used as a fire extinguisher for uranium and plutonium fires as well (Sittig, 1991).

Chemical and Physical Properties

AW: 137.3 MP: 725°C
SG.: 3.5 BP: 1640°C
Sol. (water): decomposes, combines with sulfate present in natural waters to form $BaSO_4$, which has a solubility of 1.6 mg/l at 20°C.
Sol. (organics): alcohol, insoluble in benzene.

Fate and Transport

Being extremely reactive, barium decomposes in water, and readily forms insoluble carbonate and sulfate salts. In surface or ground waters it is generally found in solution only in trace amounts. Large amounts will not dissolve because of the sulfate found in most natural water (barium sulfate has a low solubility). In water that contains more than a few ppm sulfate, barium will not dissolve at more than a few ppm. Barium sulfate may become considerably more soluble in the presence of chloride and other anions.

It is rare to find barium in drinking water at concentrations greater than 1 mg/l. Atmospheric transport of barium, in the form of particulates, can occur. Bioaccumulation is insignificant for barium (ICF, 1985).

Because of its formation of water-insoluble salts and its inability to form soluble complexes with humic and fulvic materials, barium is not expected to be very mobile in soils. However, some water insoluble barium compounds may be solubilized under acidic conditions and thereby move back into groundwater (U.S. EPA, 1984).

Pharmacokinetics

Barium and its compounds can affect the heart, lungs, central nervous system, skin, respiratory system, and eyes (Sittig, 1991).

Although quantitative data for the absorption of barium from the GI tract was not found in the literature reviewed, McCauley and Washington (1983) found relative absorption rates for barium salts with barium chloride having greater absorption than barium sulfate which, in turn, had greater absorption than barium carbonate.

Gore and Patrick (1982) reported that barium sulfate administered intratracheally to rats was concentrated in the area immediately beneath the basement membrane within 24 hrs. and remained in this area for at least 7 days. This suggests a degree of absorption from the respiratory tract.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Wones et al. (1990) administered barium (as barium chloride) in the drinking water of eleven healthy male volunteers. Subjects ranged in age from 27-61 years and had no previous history of diabetes, hypertension, or cardiovascular disease. Diets were strictly controlled throughout the 10-week study. Subjects were given 1.5 l/day of distilled, charcoal-filtered water with 0 mg/l barium for weeks 0-2, 5 mg/l for weeks 3-6, and 10 mg/l for weeks 7-10.

No changes in blood pressures or serum chemistry were detected. An increase in serum calcium levels, attributed to a decrease in serum albumin levels, although statistically significant, was not clinically significant. An NOAEL of 0.21 mg/kg/day was identified in this study.

Brenniman and Levy (1984) conducted a retrospective epidemiological study by comparing human mortality and morbidity rates in populations ingesting elevated barium levels (2-10 mg/l) in their drinking water to populations ingesting little or no barium (less than or equal to 0.2 mg/l). Differences in mortality rates from cardiovascular diseases were significantly higher in the communities with elevated barium. However, these differences were largely in the 65 and over age group and did not take population mobility, the use of water softeners, or medications into account. Differences in blood pressure, prevalence of hypertension, stroke, and heart and renal disease were also measured and no significant differences occurred between the populations.

In a variety of animal studies (McCauley, 1985; Perry et al., 1983; Schroeder and Mitchener, 1975a,b; Tardiff et al., 1980) no signs of barium toxicity were found at any dose level. Animals treated with the highest dose of barium, 1000 mg/l in McCauley's study did exhibit ultrastructural changes in the kidney glomeruli and the presence of myelin figures (IRIS).

Taransenko et al. (1977) reported on the effects barium carbonate dust had on rats when inhaled. Male rats were exposed to the dust at levels of 5.2 and 1.15 mg/m^3, 4 hrs/day for 6 months. While the rats in the high dose group experienced what Taransenko called "general toxic effects" (decreased body weight, changes in hematologic parameters), the low dose animals exhibited no toxic effects.

Workers exposed to barium dust have been shown by occupational studies to develop "baritosis." No symptoms are illustrated other than a significantly higher incidence of hypertension (IRIS).

Teratogenic and Other Developmental Effects

Taransenko et al. (1977) reported that male rats exposed to an atmospheric concentration of 22.6 mg $BaCO_3/m^3$ for one cycle of spermatogenesis exhibited decrease number of spermatozoids and a lower percentage of motile sperm forms. Female rats exhibited increased mortality in subsequent litters and a general underdevelopment of newborn pups when exposed to 13.4 mg $BaCO_3/m^3$ for 4 months. An atmospheric concentration of 3.1 mg $BaCO_3/m^3$ produced no systematic effects, although some ovarian follicle atresia was observed. When males exposed to an atmospheric concentration of 5.2 mg $BaCO_3/m^3$, 4 hours/day for 4 months when mated with unexposed females, increased mortality of the fetuses resulted.

Mutagenic Effects

Nishioka (1975) found that repair deficient strains of *Bacillus subtillis* did not exhibit an increased mutation frequency when exposed to barium chloride. Loeb et al. (1978) obtained negative results as well in tests of the induction of errors in viral DNA transcription in vitro.

Carcinogenic Effects

Barium has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential (IRIS).

McCauley et al. (1985) found no carcinogenic effect in a study of the histological and cardiovascular effects of drinking water containing 0,10,100, and 250 mg/l barium for 16,36, and 68 weeks on male Sprague-Dawley rats. Female (rats???) were exposed to 0 or 250 mg/l barium for 46 weeks.

Schroeder and Mitchener (1976a,b) investigated the carcinogenicity of barium acetate in drinking water to both rats and mice. The observed differences in tumor incidence in the rats was insignificant statistically and there was essentially no difference in tumor incidence in the mice.

Ecotoxicity

There is sufficient sulfate or carbonate present in most natural water to precipitate any barium present in the water as a virtually insoluble, non-toxic compound. Therefore, it would require a soluble barium concentration of at least 50 mg/l before toxicity to both fresh and marine aquatic life would be expected (U.S. EPA, 1986). Data pertaining to the toxicity of barium to terrestrial life, domestic or wild, was not found in the literature reviewed.

Standards, Criteria, and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor: NA
Inhalation Slope Factor: NA
Chronic Oral RfD: 7.0 x10^{-2} mg/kg/day
Chronic Inhalation RfD: 1.0 x 10^{-4} mg/kg/day
Subchronic Oral RfD: 7.0 x 10^{-2} mg/kg/day
Subchronic Inhalation RfD: 1.0 x 10^{-3} mg/kg/day
MCL: 2.0 mg/l
AWQC: Water and Fish Consumption: 1 mg/l
Fish Consumption: NA

BIBLIOGRAPHY

Brenniman, G.R. and P.S. Levy. 1984. High Barium Levels in Public Drinking Water and Its Association with Elevated Blood Pressure. Advance in Modern Toxicology IX, E.J. Calabrese, Ed. Princeton Scientific Publications, Princeton, NJ. p. 231-249. (Cited in IRIS).

Gore, D.J. and G. Patrick. 1982. A Quantitative Study of the Penetration of Insoluble Particles into the Tissue of the Conducting Airways. *Ann. Occup. Hyg.* 26(1-4):149-161 (CA98:30673n). (Cited in HEA, 1984).

ICF, Clement Associates. 1985. ***Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites.***

Loeb, L., et al. 1978. Infidelity of DNA Synthesis as Related to Mutagenesis and Carcinogenesis. *Adv. Exp. Med. Biol.* 91:209-217. (Cited in HEA, 1984).

McCauley, P.T., et al. 1985. Investigations into the Effect of Drinking Water Barium on Rats. *Environ. Health Perspect. Vol. IX*, E.J. Calabrese, Ed. Princeton Scientific Publications, Princeton, NJ. p. 197-210. (Cited in IRIS).

McCauley, P.T. and I.S. Washington. 1983. Barium Bioavailability as the Chloride Sulfate or Carbonate Salt in the Rat. *Drug. Chem. Toxicol.* 6(2):209-217 (CA99:17715d). (Cited in HEA, 1984).

Nishioka, H. 1975. Mutagenic Activities of Metal Compounds in Bacteria. *Mutat. Res.* 31:185-189. (Cited in HEA, 1984).

Perry, H.M., et al. 1983. Cardiovascular effects of chronic barium ingestion. *In.:* Proc. 17th Ann. Conf. Trace Substances in Environmental Health, Vol. 17. University of Missouri Press, Columbia, MO. p. 155-164. (Cited in HEAST).

Schroeder, H. and M. Mitchener. 1975a. Life-Term Studies in Rats: Effects of Aluminum, Barium, Beryllium, and Tungsten. *J. Nutr.* 105:421-427. (Cited in HEA, 1984).

Schroeder, H. and M. Mitchener. 1975b. Life-Term Effects of Mercury, Methyl Mercury, and Nine Other Trace Metals on Mice. *J. Nutr.* 105:452-458. (Cited in HEA, 1984).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

Tarasenko, et al. 1987. (full reference not cited in IRIS).

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1984. *Health Effects Assessment for Barium.* EPA/540/1-86/021.

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* EPA 440/5-86-001.

Wones, R.G., et al. 1990. Lack of Effect of Drinking Water Barium on Cardiovascular risk factor. (Cited in IRIS).

BERYLLIUM

Use

Beryllium is a gray metal that is used as an alloy in numerous industries because of its light weight and high tensile strength. In the atomic energy field, it is used as a moderator in fission reactors and as a reflector to reduce leakage from the core (Sittig, 1991). Beryllium is alloyed with numerous other metals. As an alloy with copper, beryllium is used in machinery parts subjected to heavy wear or extreme vibration, in springs, in radar components and in non-sparking tools. As an alloy with nickel, beryllium is used on airplanes, in diamond drill-bit matrices, and in watch-balance wheels. Beryllium-bronze is used in switch parts, watch springs, diaphragms, shims, and bushings (Sittig, 1991)

Exposure to beryllium is generally associated with the milling and alloying processes and not in the mining of the beryl ore.

Chemical and Physical Properties

AW:	9.012	BP:	2970°C
SG:	1.85 at 20°C	MP:	1278°C

Sol. (water): insoluble (except for beryllium salts)
Sol. (organics): dilute acid and alkali.

Fate and Transport

The majority of beryllium releases, most of which occurs as a result of coal combustion and milling processes, are to the atmosphere. Because these are releases of particulate, rather than dissolved beryllium, deposition is the most common fate. Generally, the ultimate destination of atmospheric beryllium is the soil (EPA, 1987). In the soil, beryllium tends to sorb to particulate matter in the relatively insoluble form of beryllium oxide.

When deposed or released to surface waters, the more commonly used beryllium compounds, most of which are water soluble, are hydrolyzed to beryllium hydroxide (ICF, 1985). Because beryllium hydroxide is relatively insoluble in the pH range of most surface waters, it remains stable and sorbs to any particulate matter present (ICF, 1985).

Pharmacokinetics

Beryllium is known to accumulate in the lungs of humans and laboratory animals after atmospheric exposure to elevated concentrations. Reeves, et al. (1967) showed that rats exposed to an atmospheric beryllium concentration of 35 μg/m^3 for 7 hours/day, 5 days/week for 72 weeks accumulated 13.5 μg in the lungs after 36 weeks of exposure. Examinations of human lung tissue revealed that beryllium concentrations in the lungs of occupationally exposed workers reach levels two to ten times as high as those in normal human lung tissues (EPA, 1987).

Ingested beryllium has, in some studies, been shown to be absorbed slightly through the gastrointestinal lining (less than 1 percent). However, Reeves (1965) exposed rats to beryllium in drinking water at an average daily ingestion concentration of either 6.6 or 66.6 µg Be. Sixty to ninety percent of the ingested beryllium was eliminated in the feces, indicating that an appreciable amount was ingested.

Absorbed beryllium accumulates primarily in the skeleton. Of the soft tissues, the liver and kidneys accumulate the most (EPA, 1987).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Acute occupational exposure to atmospheric beryllium is known to cause lung disease. In a study of six fatal cases of beryllium poisoning, Frieman and Hardy (1970) reported that death occurred between 17 and 70 days after exposure. Interstitial pneumonitis was determined to be the cause of the fatalities.

Chronic exposure to beryllium can also result in lung disease. Hardy and Tabershaw (1946) reported that 5 of 17 workers studied in a fluorescent lamp manufacturing plant died from chronic beryllium exposure. The cause of death was noted to be an inflammation of cells within the alveoli.

It has also been noted that chronic exposure to beryllium can cause enlargement of the heart, liver and spleen; cyanosis; and kidney stone development (ICF, 1985).

Teratogenic and Other Developmental Effects

Three major studies were located in the available literature that provide inconclusive evidence as to the teratogenicity of beryllium. It appears as though no reproductive or teratogenic effects are caused by beryllium (EPA, 1987).

Mutagenic Effects

Beryllium has been proven to be mutagenic to cultured mammalian cells. Miyaki, et al. (1979) noted that Chinese hamster V79 cells, induced with beryllium chloride, were six times more likely to mutate than control V79 cells. The same results were noted by Hsie, et al. (1979) in Chinese hamster ovary cells.

Human lymphocyte cells are also known to mutate more frequently when exposed to beryllium compounds. Larramendy, et al. (1981) exposed human lymphocytes to beryllium sulfate in a single dose of 0.25 μg Be/ml. A six-fold increase in chromosomal aberrations was noted during cell division.

Carcinogenic Effects

Carcinogenicity case studies of occupationally exposed workers have been inconclusive. Of the studies performed, external factors were not appropriately taken into account. In most of the studies, the effects of cigarette smoking were not factored in but, when they were, no significant increase in tumors was noted (IRIS).

Studies performed on laboratory animals indicate that beryllium is carcinogenic. Schroeder and Mitchener (1975) reported a slightly significant increase in the incidence of unspecified cancerous growths in Long-Evans rats administered 5 ppm beryllium sulfate in drinking water for a lifetime.

In numerous studies, osteogenic sarcomas were induced in rabbits exposed to beryllium compounds via intravenous injection (IRIS).

Tumors have also been induced in Wistar rats through the intratracheal injection of metallic beryllium, beryllium-aluminum alloys, and beryllium oxide. Adenomas, adenocarcinomas, and malignant lymphomas were all noted in the lungs of the test rats (IRIS, 1990).

Ecotoxicity

Beryllium's toxicity to freshwater aquatic life appears to be affected by the amount of calcium carbonate in the water. Acute toxicity values for the Fathead Minnow changed from 150 μg/l in water with 20 mg/l calcium carbonate, to 20,000 μg/l in water with 400 mg/l calcium carbonate (ICF, 1985). From the limited data available, beryllium is thought to be mildly toxic to saltwater aquatic species.

Changes in skeletal growth were noted in poultry and livestock after soluble beryllium salts were added to their diets. Rachitis, a condition in which the long bones develop improperly, was noted to occur after the induction of 0.125 percent beryllium carbonate into the diet (IRIS).

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	4.3 x 10^{0} (mg/kg/day)$^{-1}$
Inhalation Slope Factor:	8.4 x 10^{0} (mg/kg/day)$^{-1}$
Chronic Oral RfD:	5.0 x 10^{-3} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5.0 x 10^{-3} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	0.001 mg/l
AWQC:	Fish and Water Consumption - 0.0037 µg/L Fish Consumption - 0.0641 µg/L

BIBLIOGRAPHY

Frieman, D.G., and H.L. Hardy. Beryllium Disease: The Relation of Pulmonary Pathology to Clinical Course and Prognosis Based on the Study of 130 Cases from the U.S. Beryllium Case Registry. *Hum. Patho.* 1:25-44. (Cited in EPA, 1985).

Hardy, H.L. and I.R. Tabershaw. 1946. Delayed Chemical Pneumonitis Occurring in Workers Exposed to Beryllium Compounds. *Ind. Hyg. Toxicol.* 28:197-211. (Cited in EPA, 1987).

Hsie, A.W., et al. 1979. Quantitative Mammalian Cell Mutagenesis and a Preliminary Study of the Mutagenic Potential of Metallic Compounds. Karasch, N. Ed. *Trace Metals in Health and Disease*, NY, NY, Raven Press, page 55-69. (Cited in EPA, 1987).

ICF Clement. 1985. *Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites.*

Larramandy, M.L., Popescu, N.C., and N.A. DiPaolo. 1981. Induction by Inorganic Metal Salts of Sister Chromatid Exchanges and Chromosomal Aberrations in Human and Syrian Hamster Cell Strains. *Environ. Mutagen.* 3:597-606. (Cited in EPA, 1987).

Miyaki. M., Akamatsu, N., Ono, T., and H. Koyama. 1979. Mutagenicity of Metal Rations in Cultured Cells from Chinese Hamster. *Mutat. Res.* 68:259-263. (Cited in EPA, 1987).

Reeves, A.L. 1965. The Absorption of Beryllium from the Gastrointestinal Tract. *Arch. Environ. Health.* 11:209-214. (Cited in EPA, 1987).

Reeves, A.L., and A.J. Vorwald. 1967. Beryllium Carcinogenesis: II, Pulmonary Deposition and Clearance of Inhaled Beryllium Sulfate in the Rat. *Cancer Res.* 27:446-451. (Cited in EPA, 1987).

Schroeder, H.A. and M. Mitchener. 1975. Life-Term Studies in Rats: Effects of Aluminum, Barium, Beryllium and Tungsten. *J. Nutr.* 105:421-427. (Cited in IRIS).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1987. *Health Assessment Document for Beryllium.*

U.S. EPA. *Health Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water* EPA 440/5-86-001.

Vorwold, A.J. 1968. Biologic manifestations of toxic inhalants in monkeys. *In:* Use of Nonhuman Primates in Drug Evaluation, H. Vagtborg, Ed. Univ. of Texas Press, Austin, TX. p. 222-228. (cited in IRIS).

CADMIUM

Use

Elemental cadmium is a soft white metal similar to lead and zinc in texture and in other physical properties.

Cadmium is obtained as a byproduct during the production of zinc. Commercially, cadmium is used in the metal plating industry; as a stabilizer in paints, pigments, and plastics; and as an energy storage medium in batteries. It is also used in pesticides, as an alloy additive, and in chemical reagents. Cadmium may escape into the air from zinc, lead, or copper smelters. Naturally occurring levels of cadmium in surface and ground water normally fall in the range of 1-10 g/liter (EPA, 1985).

Physical and Chemical Properties

AW: 112.41 BP: 765°C
SG: 8.642 MP: 321°C
VP: 1 mmHg at 394°C
Sol. (water): metal is insoluble, salts of metal are soluble
Sol. (organics): variable

Fate and Transport

The primary vehicle for cadmium exposure in a non-occupational setting is through the ground water. Cadmium is relatively mobile in aquatic environments and sorbs to organic material found in soils (EPA, 1984). It is thought to be transported slowly by ground water, but no comprehensive studies have been performed in this regard. High cadmium levels are often found in ground water surrounding smelting and plating facilities (Sittig, 1991). Occupationally, workers can be exposed to cadmium in the form of dust or fumes.

Pharmacokinetics

Cadmium is absorbed moderately in the lungs but quite poorly in the gastrointestinal tract (1 to 6 percent in both humans and animals). The primary excretory route for absorbed cadmium is the urine (ATSDR, 1989). Urinary excretion is slow, however, and cadmium has a strong tendency to accumulate in the body (mostly in the liver and renal cortex) over time in exposed humans and in animals (cadmium binds tightly to the protein metallothionein or its cellular components). The half lives of cadmium and its compounds in the body range from 17 to 38 years (ATSDR, 1989). Measurements of alveolar absorption in rats indicate 60 to 70 percent absorption over time. Calculations based on increased body burden in smokers compared to that in nonsmokers suggest that respiratory absorption in humans is probably about 30 to 60 percent (ATSDR, 1989). The absorption of cadmium following oral administration of laboratory animals, and presumably humans, is not a simple process and is modified by many factors including chemical form solubility dose, age, diet, and by the presence of other metals. Small quantities of cadmium may be absorbed through the skin but dermal absorption is not normally significant relative to total cadmium absorption (ATSDR, 1989). In general, soluble compounds such as $CdCl_2$ are better absorbed and are more toxic than highly insoluble compounds such as CdS. (ATSDR, 1989).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

In the case of severe intoxication, sensory disturbances, liver injury, and convulsions may occur. In fatal intoxications, this is followed by shock and/or renal failure and cardiopulmonary depression (EPA, 1985). Exposure to concentrations of 40 to 50 mg/m^3 for 1 hour and 9 mg/m^3 for 5 hours has resulted in fatalities. LD_{50} values in animals exposed to cadmium oxide fumes range from 500 to 15,000 mg/m^3 minute (ATSDR, 1989). Acute oral LD_{50} values in animals for cadmium oxide and common cadmium salts range from 50 to 350 mg/kg (ATSDR, 1989).

Renal effects: The kidney is generally recognized as the most sensitive tissue to low-level cadmium exposure, the major effect being impaired tubular reabsorption. Rats receiving water containing cadmium at 30 or 100 mg/liter developed significant ($p<0.05$) proteinuria after 6 weeks of exposure (EPA, 1985). Various studies indicate that tubular dysfunction does not generally occur in humans until a renal cortical concentration of approximately 200 μg/g wet weight is reached (ATSDR, 1989). Using this figure, it was estimated that a daily oral intake of 352 μg/day over 50 years would not exceed the critical level of cadmium in the renal cortex. A more recent study in which epidemiological studies were reviewed, however, concluded that an average oral exposures of about 200 μg/days will cause tubular proteinuria in about 10 percent of an exposed population by age 45 (ATSDR, 1989). It was also estimated that 10 percent of a working population exposed via inhalation to 50 μg/m^3 would develop proteinuria in 10 years. (ATSDR, 1989).

Hepatic effects: The next highest tissue levels of cadmium are found in the liver. While structural changes were observed following cadmium exposure in food and water to rats and rabbits, clinical tests revealed normal hepatic function. There is little evidence for liver dysfunction in chronically exposed human populations but hepatic levels may serve as a useful index of exposure and a predictor of future renal dysfunction (ATSDR, 1989).

Cardiovascular effects: Certain animal studies have indicated that increases in average systolic blood pressure occur following exposure to cadmium acetate in the drinking water (0.5 mg/kg/day); not all investigations have succeeded in confirming these findings and other factors may confound the effects of cadmium (ATSDR, 1989). The role of cadmium is human hypertension is uncertain (ATSDR, 1989).

Pulmonary effects: Inhalation exposure to high levels of cadmium oxide fumes is intensely irritating to respiratory tissues (ATSDR, 1989).

Gastrointestinal effects: In humans, the symptoms of cadmium toxicity following acute oral exposure include nausea, vomiting, diarrhea, abdominal pain, and salivation (ATSDR, 1989).

Other systemic effects: Weak evidence exists indicating skeletal effects in humans and animals exposed chronically to cadmium. Studies revealed that relatively low doses of cadmium can alter the immune response in animals (at very low renal cadmium concentrations ranging from 0.3 to 6.0 μg/g) (ATSDR, 1989). Parenteral injection of cadmium has been observed to cause severe acute pathological changes in the gonads of animals (ATSDR, 1989). Exposure by injection of male rats with 2.2 mg/kg of $CdCl_2$ resulted in swelling and inflammation of testes, followed by necrosis and atrophy, in several studies. Another common effect in cadmium-exposed animals is anemia (ATSDR, 1989).

Teratogenic and Other Developmental Effects

Sutou, et al. (1980) administered cadmium at 0, 0.1, 1.0, and 10.0 mg/kg/day (as $CdCl_2$) orally to male and female adult rats for 6 weeks. Males and female were mated for 3 weeks, and cadmium was administered during the mating period. Pregnant females were given cadmium during the gestation period. The number of total implants and live fetuses decreased significantly in the 10 mg/kg group, and the number of resorbed fetuses was markedly increased. Fetuses showed decreased body weight, and delayed ossification of the sternebrae and caudal vertebrae. Ahokas, et al. (1980) observed, in a rat drinking-water-study, fetal growth retardation in animals whose dams were exposed to 100 mg cadmium/L but not in those exposed to 0.1 or 10 mg cadmium/L during gestation. The most common finding is the decreased weight of offspring, with ingestion exposure, usually without significant teratogenic or developmental effects (ATSDR, 1989). Cadmium exposure has not been observed to cause teratogenic or other developmental effects in exposed humans (ATSDR, 1989).

Mutagenic Effect

Studies to assess the mutagenic activity of cadmium, in *Salmonella typhimurium, E. coli,* and yeast, have been inconclusive (ATSDR, 1989). Recombination assays in *Bacillus subtilis* have yielded weak positive responses (ATSDR, 1989). Cadmium has been shown to be mutagenic both in the mouse lymphoma assay and in the Chinese hamster cell assay (ATSDR, 1989). Chromosomal aberration studies on human lymphocytes from exposed workers and in human and animal cells treated with cadmium *in vitro* have produced conflicting results (ATSDR, 1989).

Carcinogenic Effects

EPA has evaluated the weight of evidence on the carcinogenicity of cadmium and has concluded that cadmium is a probable human carcinogen (Group B1) by inhalation (ATSDR, 1989/IRIS). An occupational study of smelter workers by Thun, et al. (1985) revealed a two-fold excess risk of lung cancer but confounding factors could not be ruled out. Wistar rats exposed to cadmium chloride developed significant increases in lung tumors (Takenaka, et al., 1983). No sufficient data exists to consider cadmium as carcinogenic by the oral route, nor is there evidence that cadmium, via the dermal route, is carcinogenic to either animals or humans.

Ecotoxicity

The acute LC_{50} values for cadmium exposure in freshwater fish and invertebrates generally range from 100 to 1,000 μg/liter. Salmoids, being very sensitive, would be at the lower end of this range. Saltwater species appear to be, in general, 10-times more tolerant to the acute effects of cadmium than freshwater species (ICF, 1985). Cadmium is strongly accumulated by all organisms (ATSDR, 1989). Bioconcentration factors (BCFs) for cadmium in freshwater range from 164 to 4,190 for invertebrates and from 3 to 2,213 for fish. BCFs for saltwater invertebrates range from 5 to 3,160 (EPA, 1986).

Freshwater acute values for cadmium are available for species in 44 genera and range from 1.0 μg/L for rainbow trout to 28,000 μg/L for mayflies. Chronic tests conducted for cadmium on 12 freshwater fish species and 4 invertebrate species revealed chronic values ranging from 0.15 μg/L for *Daphnia magna* to 156 μg/L for the Atlantic salmon. Acute-chronic ratios, available for eight species, range from 0.9021 for the Chinook salmon to 433.8 for the flagfish (EPA, 1986). Freshwater aquatic plants are affected by cadmium at concentrations ranging from 2 to 7,400 μg/L. The major toxic effect observed in freshwater aquatic plants was growth reduction.

Saltwater acute values for cadmium in five species of fish range from 577 μg/L for Atlantic silverside to 114,000 μg/L for juvenile mummichog. Invertebrate acute values (30 species) range from 15.5 μg/L for a mysid to 135,000 μg/L for an oligochaete worm. Acute toxicity of cadmium usually increases as salinity decreases. Chronic cadmium exposure has been shown to significantly affect the growth of bay scallops at 78 μg/L and the reproduction of certain copepods at 44 μg/L (EPA, 1986).

Standards, Criteria and Guidelines

EPA Class B1 Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	$6.3\text{x}10^{0}$ $(\text{mg/kg/day})^{-1}$
Chronic Oral RfD:	1.0×10^{-3} mg/kg/day (food) 5.0×10^{-4} mg/kg/day (water)
Chronic Inhalation RfD:	Currently under review by EPA
Subchronic Oral RfD:	5.0×10^{-4} mg/kg/day

Subchronic Inhalation RfD:	NA
MCL:	0.005 mg/l
AWQC:	Water and Fish Consumption - 10 μg/l Fish Consumption - NA

BIBLIOGRAPHY

Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, *Toxicological Profile for Cadmium*, ATSDR/TP-88/08, March 1989.

Ahokas, R.A., P.U. Dilts and E. B. LaHage, Cadmium Induced Fetal Growth Retardation: Protective Effect of Excess Dietary Zinc, 1980, *Am. J. Gynecol.* (cited in EPA, 1985).

ICF Clement Associates, *Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites*, September, 1985.

Sittig, Marshall, *Handbook of Toxic and Hazardous Chemicals*, 1991.

Sutou, S., K. Yamamoto, H. Sendota and M. Sugiyama, Toxicity, Fertility, Teratogenicity and Dominant Lethal Tests in Rats Administered Cadmium Subchronically. III, Fertility, Teratogenicity and Dominant Lethal Test, 1980, *Ecotoxicol. Environ. Safety*, 4:51-56, (Cited in EPA, 1985).

Takenaka, S., H. Oldiges. H. Konig, D. Hochrainer and G. Oberdoerster, Carcinogenicity of Cadmium Aerosols in Wistar Rats, 1983, *J. National Cancer Inst.*, 70: 367-373, (Cited in IRIS).

Thun, M.J., T.M. Schnorr, A.B. Smith and W.E. Halperin, Mortality Among a Cohort of U.S. Cadmium Production Workers: An Update, 1985, *J. Natl. Cancer Inst.*, 74(2): 325-333, (Cited in IRIS).

U.S. EPA, *Drinking Water Regulations and Health Advisories.*

U.S. EPA, *Health Advisory for Cadmium*, 1985.

U.S. EPA, Office of Water Regulations and Standards, *Quality Criteria for Water*, 440/5-86-001, May 1, 1986.

U.S. EPA, *Acute Toxicity Handbook of Chemicals to Estuarine Organisms*, EPA/600/8-87/017, April 1987 (Env. Res. Lab/Office of R and D) Gulf Breeze, Florida.

U.S. EPA, *Health Effects Assessment for Cadmium*, EPA 540/1-86/038, 1984.

U.S. EPA, *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

CHROMIUM

Use

Chromium and chromium compounds are used in stainless and alloy steels, refractory products, tanning agents for leather, pigments, electroplating, catalysts, and in corrosion resistant products (ACGIH, 1991).

Chemical and Physical Properties

AW: 51.996 BP: 2672°C
SG: 7.20 at 28°C MP: 1857 ±20°c
Sol. (water): insoluble, some compounds are soluble.

Fate and Transport

Chromium (VI) is soluble in water and is transported easily in ground water. It may exist in aquatic systems as water soluble, complex anions, and may persist for long periods of time (EPA, 1984a). Chromium (VI) may react with organic matter in the soil or surface waters to form trivalent chromium, therefore chromium (VI) may exhibit a shorter lifespan in soils with high organic content (EPA, 1984a).

The primary means by which chromium (III) is transported out of soils and surface waters is through aerosol formation and runoff. It is also hydrolyzed to chromium hydroxide. Leaching does not generally occur with chromium (III) because it is generally present as insoluble chromium trioxide.

Pharmacokinetics

The amount of ingested chromium (VI) and (III) absorbed is estimated to be 5 percent and 3 percent respectively. Donaldson and Barreras (1966) fed $\mathrm{Na_2{}^{51}Cr(VI)O_4}$ to rats and humans and $\mathrm{{}^{51}Cr(III)Cl_3}$ to humans. Based on mean urinary excretion of $\mathrm{{}^{51}Cr(VI)}$, absorption was estimated to be 2.1 percent in humans. In rats, 2 percent of the administered dose was absorbed, based on fecal excretion of $\mathrm{{}^{51}Cr(VI)}$. Based on fecal excretion of $\mathrm{{}^{51}Cr(III)}$, absorption was estimated to be 0.4 percent in humans. However, when $\mathrm{Na_2{}^{51}Cr(VI)O_4}$ was administered introduodenally (in humans) or intrajejunally (in rats), absorption was estimated to be 50 and 25 percent, respectively. When $\mathrm{{}^{51}Cr(III)Cl_3}$ was administered introduodenally, absorption was not appreciably changed. A study by Langard, et al. (1978) indicates that water-soluble chromium (VI) is absorbed rapidly via inhalation. Rats were exposed to zinc chromate dust at a level of 7.35 mg/m^3. After 0, 100, 250, and 350 minutes of exposure, the concentrations of chromium in the blood (µg/ml) were 0.007, 0.024, 0.22, and 0.31, respectively. Chromium (III) is absorbed slowly via inhalation. Baetjer et al. (1959) administered $\mathrm{{}^{51}Cr(III)Cl_3}$ to guinea pigs intratracheally. Only 4 percent of the administered dose was detected in the blood and tissues 10 minutes post-treatment; 69 percent remained in the lungs. 45, 30, and 12 percent of the administered dose was detected in the lungs 1, 30, and 60 days post-treatment, respectively.

Human Toxicity

Noncarcinogenic

Systemic Effects

Bloomfield and Blum (1928) examined 23 men from six chromium plating plants in the U.S. Fourteen of the workers typically spent 2-7 hours/day over vats of chromic acid, which generated airborne hexavalent chromium ranging from 0.12-5.6 mg/m^3. These men experienced nasal tissue damage, including perforated septa, ulcerated septa, chrome holes, nosebleed, and inflamed mucosae. The nine remaining workers not directly exposed to chromium vapors had only inflamed mucosae.

Mackenzie, et al. (1958) exposed groups of rats, both male and female, to potassium dichromate (0-25 ppm of hexavalent chromium) in drinking water for 1 year. No effects were observed at any level of treatment. Pertinent data regarding subchronic exposure of animals to hexavalent chromium via inhalation were not located in the literature (EPA, 1984a).

Ivankovic and Preussman (1975) exposed groups of 60 male and female rats to 0, 1, 2, or 5 percent $Cr(III)_2O_3$ in baked bread, 5 days/week for 600 feedings. The average total amounts of ingested $Cr(III)_2O_3$ were given as 0, 360, 720, and 1800 g/kg bw. No adverse effects were observed at any dose level.

Teratogenic and Other Developmental Effects

The literature available on teratogenic effects resulting from ingestion of chromium is limited. However, several forms of chromium (including chromium (III)), when administered to pregnant rats by stomach intubation in the form of GTF (obtained from yeast), have been found to cross the placental barrier and be recovered by the fetus (EPA, 1985).

Mutagenic Effects

Compounds of both chromium (III) and chromium (VI) increase noncomplementary nucleotide incorporation into DNA with chromium (VI) being effective at lower doses. Exposure of cells from rat liver and kidney to chromium (VI) leads to increased cross-linking in DNA. Positive Ames tests for chromium (VI) have been reported; however chromium (III) exerted no effect at relatively high concentrations (presumably because of its inability to penetrate cells), (EPA, 1985).

Carcinogenic Effects

Data regarding the carcinogenicity of inhaled chromium (VI) is well established for occupational exposure in humans. The effects are observed only in the respiratory passages and in the lungs (EPA, 1985).

Numerous epidemiological studies indicate that various forms of chromium (VI) cause lung cancer as a result of chronic exposure (Machle and Gregorius, 1948). It has been estimated that workers in the chromate pigment industry who had developed lung cancer were exposed to 0.01 to 0.15 mg/m^3 of water soluble chromium and 0.1 to 0.58 mg/m^3 of water insoluble chromium. From subsequent studies, it appears that water insoluble compounds of chromium (VI) resulted in the increase in lung cancer (ACGIH, 1984).

There is inadequate evidence to determine whether or not oral exposure to chromium (III) can lead to cancer. Rats exposed to chromium (III) at 293, 586, or 1,4676 mg/kg/day in the diet (administered as chromium oxide pigments) for 2 years, displayed no increase in the tumor rates over that of the control animals (EPA, 1985).

Ecotoxicity

Chromium is an essential nutrient and is accumulated in a variety of aquatic and marine biota, especially benthic organisms, to levels much higher than in ambient water. Levels in biota, however, are usually lower than levels in the sediments. Passage of chromium through the food chain can be demonstrated (ICF, 1985). The food chain appears to be a more efficient pathway for chromium uptake than direct uptake from seawater (ICF, 1985). Water hardness, temperature, dissolved oxygen, species, and age of the test organism all modify the toxic effects of chromium on aquatic life. Chromium (III) appears to be more acutely toxic to fish than chromium (VI), yet the reverse is true in long-term chronic exposure studies (ICF, 1985). None of the plants normally used as food or animal feed are chromium accumulators. Chromium absorbed by plants tends to remain primarily in the roots and is poorly translocated to the leaves. There is little tendency for chromium to accumulate in food chains in the trivalent inorganic form. Organic chromium compounds, about which little is known, can have significantly different bioaccumulation tendencies (ICF, 1985).

Standards, Criteria and Guidelines

EPA Class A Carcinogen (Hexavalent Chromium)

Oral Slope Factor:	NA
Inhalation Slope Factor:	4.2×10^{1} (mg/kg/day)$^{-1}$ (VI)
Chronic Oral RfD:	5.0×10^{-3} mg/kg/day (VI) 1×10^{0} mg/kg/day (III)
Chronic Inhalation RfD:	NA

Subchronic Oral RfD: 2.0×10^{-2} mg/kg/day (VI)
1.0×10^{1} mg/kg/day (III)
Subchronic Inhalation RfD: NA
MCL: 0.1 mg/l (total)
AWQC: Water and Fish Consumption - 170 mg/L (III)/.050 mg/L (VI))
Fish Consumption - 3433 mg/L (III)

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists (ACGIH). 1991. *Documentation of the Threshold Limitation Values.*

Baetjer, A.M., et al. 1959. The distribution and retention of chromium in men and animals. *Arch. Ind. Health* 20:136-150. (Cited in EPA, 1984b).

Bloomfield, J.J. and W. Blum. 1928. Health Hazards in Chromium Plating. *Public Health Rep.* 43:2330-2351. (Cited in EPA, 1984a).

Donaldson, R.M. and R.F. Barreras. 1966. Intentional Absorption of Trace Quantities of Chromium. *J. Lab Clinic. Med.* 68:484-493. (Cited in EPA, 1984a).

Furst, A., et. al. 1976. Tumorigenic activity of lead chromate. *Cancer Res.* 36:1779-1783. (cited in IRIS).

ICF Clement. 1985. *Chemical, Physical and Biological Properties of Compounds present at Hazardous Waste Sites.*

Ivankovic, S. and R. Preussman. 1975. Absence of toxic and carcinogenic effects after administration of high doses of chromic oxide pigment in subacute and long term feeding experiments in rats. *Food Cosmet. Toxicol.* 13:317-351. (cited in IRIS).

Langard, S., N.J. Gundersen, D.L. Tsalev and B. Glyseth. 1978. Whole Blood Chromium Level and Chromium Excretion in the Rat After Zinc Chromate Inhalation. *Acta. Pharmacal. Toxicol.* 42:142-149. (Cited in EPA, 1984a).

Lindberg, E. and G. Hedenstierna. 1983. Chrome plating: Symptoms, findings in the upper airways, and effects on lung function. *Arch. Environ. Health.* 38:367-374. (cited in HEAST).

Machle, W. and F. Gregorius. 1948. Cancer of the respiratory system in the United States chromate-producing industry. *Public Health Rep.* 63(35):1114-1127. (cited in IRIS).

Machle, W. and F. Gregorius. 1948. Cancer of the Respiratory System in the United States chromate-producing industry. *Public Health Rep.* 63(35):114-1127. (cited in IRIS).

Mackenzie, R.D., R.A. Anwar, R.U. Byerrum, and C.A. Hoppert. 1959. Absorption and Distribution of ^{51}Cr in the Albino Rat. *Arch. Biochem. Biophys.* 79:200-250. (Cited in EPA, 1984a).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1985. *Health Advisory for Chromium, Office of Drinking Water.*

U.S. EPA. 1984a. *Health Effects Assessment for Hexavalent Chromium.* EPA 540/1-86/019.

U.S. EPA. 1990. *Integrated Risk Information System.*

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. 1984b. *Health Effects Assessment for Trivalent Chromium.* 2 EPA 540/1-86/035.

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

COBALT

Use

Cobalt (Co) is a silver-grey, hard, brittle, magnetic metal. It is used in alloy with nickel and aluminum in the manufacture of permanent magnets. Alloys with nickel, aluminum, copper, beryllium, chromium, and molybdenum are used in the electrical, automobile and aircraft industries. Tool steels include Co to improve their cutting qualities. Tungsten carbide tool manufacture utilizes Co as a binder. Various Co compounds are used as pigments in enamels, glazes and paints; as catalysts in afterburners; and in the glass, pottery, photographic, and electroplating industries. Radioactive Co is used in the treatment of cancer. (Sittig, 1991).

Chemical and Physical Properties

Atomic Symbol:	Co
AW: 58.93	BP:2870°C
SG: 8.9 at 20°C	MP:1495°c
Sol.(water):	insoluble; soluble in acid

Fate and Transport

ICF (1985) states that photolysis, volatilization, and bio-transformation are not significant environmental fates for Co. Atmospheric transport of Co can occur, however. In natural aquatic systems, very little Co is present in soluble form, in fact, concentrations greater than 10 μg/liter are rare (ICF, 1985). In aquatic and terrestrial systems, absorption to clay minerals and hydrous oxides of iron, manganese, and aluminum often present in the clay fractions of sediments and soils appears to be the most important control on the mobility of Co (ICF, 1985). Eh, pH, and the concentrations of Co and competing compounds are the principal factors controlling absorption/desorption. Other fate processes include chelation with organic compounds, solubilization by bacteriological activity and slight bioaccumulation (ICF, 1985).

Pharmacokinetics

Co is an essential micronutrient in animals and man. The body, therefore, is capable of metabolizing moderate quantities of Co compounds. Co is an important element in Vitamin B_{12} and certain enzymes, and is associated with the production of erythropoietin, the red cell stimulating factor (Clayton and Clayton, 1981). Schroeder (1967) reports that the normal Co balance in man includes a daily food intake of 140-580 mg/day, a daily water intake of 0-10 mg/day and an inhalation intake <0.1 mg/day. Output includes 120-330 mg/day in urine, 23-60 mg/day in feces, and 6 mg/day in sweat and hair. Forbes et al. (1954) found human tissue concentrations of Co to range form 0.01 ppm for fat, nerve, muscle and the GI tract to 0.06 ppm for liver.

Gastrointestinal absorption of Co and Co compounds is dose-dependent. Smaller doses, on the order of a few mg/kg, are almost completely absorbed while larger doses are less well absorbed (Clayton and Clayton, 1981). For example, Copp and Greenberg (1941) found 30 percent of radioactive Co (^{60}Co) in urine in rats following a 10 mg orally administered dose, and more than 90 percent in urine following a 10 mg injected dose.

Wehner and Craig (1972) studied the distribution of CoO in hamsters. 87 percent of an inhaled 784 mg dose was distributed throughout the body and 11.3 percent of a 5 mg dose administered by gavage. The greatest amounts, 60 percent and 11 percent, respectively, remained in the GI tract. The carcass retained 23 percent and 0.34 percent; the lung, 3.3 percent and <0.06 percent; and the liver and kidneys retained small fractional percentages of the doses.

In man, an intravenously injected dose of 13 mg Co, as $CoCl_2$, resulted in a tenfold increase in urinary output and a seventeen fold increase in fecal excretion during the first week following injection. A total of 3 mg Co were recovered during this week, indicating slow elimination of Co (Kent and McCance, 1941).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The acute toxicity of Co appears to vary according to Co compound. Clayton and Clayton (1981) report on numerous animal studies that demonstrate toxic effects at concentrations ranging from 20 mg/kg for $CoCl_2$ (administered intravenously to rats) to 1700 mg/kg for CoO (administered orally). Toxic effects observed include diarrhea, loss of appetite, paralysis of the hind legs, and lowering of body temperature prior to death. Smaller doses produced albuminuria while larger doses resulted in anuria. Cutaneous vasodilatation occurs almost immediately and blood pressure may fall. Microscopically, organs become congested with small focal hemorrhages on serosal surfaces and large hemorrhages in the liver and adrenals. Degenerative effects may occur in bone, lung, kidney, heart, and pancreas tissue (Clayton and Clayton, 1981). Frederick and Bradley (1946), however, report no toxic effects for Co_2O_3 administered intraperitoneally to rats at 5000 mg Co/kg.

Chronically, Co toxicity appears to have a cumulative effect where elimination cannot keep pace with absorption. Schepers (1955) found repeated dosing of 5 mg Co intratracheally to be lethal to rats while a single 5 mg dose was not. Similarly, Frederick and Bradley (1946) found repeated 30 mg doses of Co were lethal to rats whereas 1500 mg Co was the lethal single dose. Underhill et al. (1931) revealed that dietary components may affect the toxicity of Co.

Rats on a milk diet died at daily doses of 1.0 and 0.5 mg Co after 3.5 months whereas rats on a typical laboratory food diet tolerated 1 mg Co in drinking water for 14 weeks.

However, other studies indicate that a tolerance for Co may be developed if initial doses are sufficiently low to be well tolerated (Clayton and Clayton, 1981). For example, a relatively huge dose of 1g $CoCl_2 \cdot 6H_2O$ was required to be lethal following 13 days of daily subcutaneous injections of 10 mg $CoCl_2 \cdot 6H_2O$ (Seghini, 1940).

Chronic inhalation by animals of a Co-metal blend used in industry containing 6 percent Co resulted in focal fibrotic lesions, hyperplasia of the bronchial epithelium, and developing granulomas in areas of dust deposition after 3 years. The granulomas appear to resemble those reported in industrial workers (Stokinger et al., undated).

Schwartz et al., (1947) has documented a dermatitis of the allergic sensitivity type that appears to be related to the abrasive nature of Co dust. The dermatitis occurred in Co-cemented tungsten carbide workers, Co-alloy workers, and finnish pottery workers using Co-containing clay. Other toxic effects related to occupational exposure to Co dust include a rare "carboloy itch", pneumoconiosis, and sensitization.

Sullivan et al. (1969); McDermott et al., 1966; and Roy et al. (1968) report that Co was unexpectedly found to be the cause of severe lesions in cardiac muscle, hypothyroidism, and thyroid hyperplasia in excessive beer drinkers who drank beer containing $CoSo_4$ as a foam stabilizer. The Co apparently caused acute heart failure that was frequently fatal (50 deaths among 112 beer drinkers). Typically, patients experienced dyspnea, with abdominal pain and edema for 1 to 2 weeks. Extreme cardiomegaly with associated low blood pressure and pulse and peripheral cyanosis was common. Early deaths occurred within 72 hours of hospital admission. Out of 34 survivors in Omaha, Nebraska, 20 regained normal cardiac status and had good exercise tolerance, normal heart size, and minimal EKG changes. Six had recurrent or chronic heart failure. Four patients had neurological and mental deterioration, and 2 died suddenly after leaving the hospital.

Teratogenic Effects

ICF (1985) reports that Co caused craniofacial developmental abnormalities in the offspring of mice exposed by intraperitoneal injection during pregnancy.

No other data on the teratogenic effects of Co were found in the literature reviewed.

Mutagenic Effects

ICF (1985) reports that there is limited data indicating that $CoCl_2$ has mutagenic activity in a variety of test systems. No other data on the mutagenic effects of Co were found in the literature reviewed.

Carcinogenic Effects

Gilman (1962) and Heath (1960) report that Co and Co cause injection site sarcomas in rats. However, ICF (1985) reports that this type of response, by itself, is not generally considered adequate evidence of carcinogenicity. ICF (1985) states that "the absence of positive carcinogenic responses in other studies with experimental animals and the lack of epidemiologic evidence suggest that cobalt and its compounds are unlikely to pose a carcinogenic risk to humans." Other data on the carcinogenicity of Co were not found in the literature reviewed.

Ecotoxicity

ICF (1985) reports that data on the ecotoxicity of Co is limited. 50 ppm per day (3 mg/kg body weight) in the diet was acutely toxic to chickens. In sheep acute toxicity occurred at 6 mg/kg body weight. 3 mg/kg body weight, 1000 times the normal daily intake of Co, did not produce harmful effects in sheep, even after several weeks.

Standards, Criteria, and Guidelines

Unclassified by EPA as to carcinogenicity

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

BIBLIOGRAPHY

Clayton G.D. and F.E. Clayton. 1981. *Patty's Industrial Hygiene and Toxicology, 3rd revised edition.* Vol. 2A: Toxicology.

Copp, D.H. and D.M. Greenberg. 1941. *Proc. National Academy Science U.S.* 27:153. (cited in Patty's, 1981).

Forbes, R.M., et al. 1954. *J. Biol. Chemical.* 209:857. (cited in Patty's, 1981).

Frederick, W. and W. Bradley. 1946. *7th Ann. Mtg. Am. Ind. Hyg. Assoc.* Chicago. (cited in Patty's, 1981).

Gilman, J.P.W. 1960. Metal carcinogenesis of malignant tumors induced by cobalt in the rat. *Br. J. Cancer.* 14:478-482. (cited in ICF, 1985).

ICF, Clement Associates. 1985. *Chemical, Physical, and Biological Properties of Chemicals Present at Hazardous Waste Sites.*

Kent, N.L. and R.L. McCance. 1941. *Biochem. J.* 35:877. (cited in Patty's, 1981).

McDermott, P.H., et al. 1966. *J. Am. Med. Assoc.* 198:253. (cited in Patty's, 1981).

Roy, P.E., et al. 1968. *Am J. Clin. Pathol.* 50:234. (cited in Patty's 1981).

Schepers, G.W. 1955. *Arch. Ind. Health.* 12:127. (cited in Patty's, 1981).

Schroeder, H.A., et. al. 1967. *J. Chron. Dis.* 20:869. (cited in Patty's 1981).

Schwartz, L., et al. 1943. *Occupational Diseases of the Skin.* 2nd ed. (cited in Patty's 1981).

Seghini, C. 1940. *Clin. Med. Ital.* 71:355. (cited in Patty's 1981).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

Stokinger, H.E., et al. Unpublished results. (cited in Patty's 1981).

Sullivan, J.F., et al. 1969. *Ann Intern. Med.* 70:277. (cited in Patty's, 1981).

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

Wehner, A.P. and D.K. Craig. 1972. *Am. Ind. Hyg. Assoc. J.* 33:146. (cited in Patty's 1981).

COPPER

Use

Copper is widely used in the electrical industry because of its high conductivity. It is used in the casting industry and as an important alloying material. Common alloys which contain copper include brass, bronze, bell metal, and German silver (Sittig, 1991). Copper compounds are used in insecticides, fungicides, molluscicides, and paints (Sittig, 1991).

Chemical and Physical Properties

AW: 63.546	BP: 2567°C
SG: 8.92	MP: 1083°C
Sol.(Water): some copper salts.	VP:1 mmhg at 1628°C
Sol.(Organics): insoluble	

Fate and Transport

Copper is present in the atmosphere primarily as dust and fumes from copper smelting plants. Although the atmospheric fate of copper has not been widely studied, it is thought that any chemical reactions would probably result in speciation (EPA, 1984). The principle atmospheric removal mechanisms are probably wet and dry deposition.

In surface waters, chemical speciation and sorption are the two dominant fate processes of released copper (EPA, 1984). In acidic waters, copper probably exists as Cu^{+2}. In alkaline waters, it probably exists as the carbonate complex. In organically rich waters, copper sorbs and forms complexes with organic material (EPA, 1985).

In the soils, the environmental fate of copper appears to depend on the Ph. In acidic soils, copper mobility would increase and leaching would occur more readily. In highly organic soils, copper would form complexes and would not leach (EPA, 1984).

Pharmacokinetics

In an extensive study using radioactive copper, Weber, et al. (1969) reported that the absorption in humans is diphasic. Primary absorption occurs within one hour of ingestion in the stomach and duodenum. The second phase occurs greater than 3.5 hours after ingestion in the small intestine. The average net absorption of ingested copper was 60 percent. Absorbed copper is stored primarily in the liver, heart, brain, kidneys, and muscles. Other studies indicate that mammals absorb copper in the upper gastrointestinal tract only.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Chattani, et al. (1965) evaluated data on the intentional ingestion of copper (as copper sulfate) by 53 suicide patients. Ingestion amounts ranged between 0.25 - 7.6g copper. Five patients died and those that survived were subject to nausea, vomiting, and epigastric pain.

Inhalation exposure to copper can cause the influenza-like symptoms of "metal fume fever". Symptoms include fever, chills, aching muscles, dryness of mouth and throat, and headaches (ICF, 1985). Chronic toxic effects of copper ingestion can include gastritis, hepatic neurosis, gastrointestinal bleeding, hypotension, and death (ICF, 1985). Copper salts act as skin irritants and can even cause conjunctivitis and corneal ulcerations when in direct contact with the eyes (ICF, 1985).

The chronic effects of copper poisoning can best be illustrated by the effects of Wilson's disease, a disease which inhibits the metabolism of copper in the body. Individuals with this disease accumulate approximately 20 times the normal amount of copper. These elevated concentrations effect the central nervous system, eyes, brain, and kidneys. It is characterized by tremors, drooling, seizures, jaundice, and eventually death (EPA, 1984).

Teratogenic and Other Developmental Effects

Copper compounds are known to be teratogenic to hamster and mice. Lecyk (1980) noted that low doses of copper stimulated embryonic development but higher doses (3000 - 4000 ppm) caused an increase in fetal mortality and embryonic malformations.

Mutagenic Effects

Copper appears to increase the number of mutagenic incidences in bacteria but does not seem to effect humans or animals in the same way (ICF, 1985).

Carcinogenic Effects

Copper and its compounds were not found to be carcinogenic to laboratory animals (EPA, 1985). Data regarding human carcinogenicity were not located in the available literature.

Ecotoxicity

The toxicity of copper to aquatic organisms appears to decrease with alkalinity, hardness, and total organic content (ICF, 1985). Acute toxicity values range between 7.2 mg/l for *Daphnia pulicaria* and 10,200 mg/l for the bluegill. Bioconcentration of copper appears to occur readily in freshwater and saltwater species (ICF, 1985).

Copper is known to be highly toxic to sheep. A dose of 200 mg/kg will generally kill a sheep. Ingestion of 1.5g/day for 30 days is also fatal to many breeds of sheep (ICF, 1985). It appears as though sheep have a reduced ability to excrete or metabolize copper (EPA, 1985).

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCLG:	1.3 mg/l
AWQC:	Water and Fish Consumption - NA Fish Consumption - NA

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists. 1984. *Documentation of the Threshold Limit Values.*

Chattani, H.K., P.S. Gupter, S. Gailati and D.N. Gupta. 1965. Acute Copper Sulphate Poisoning. *Am. J. Med.* (Cited in EPA, 1984).

Lecyk, M. 1980. Toxicity of Cupric Sulfate in Mice Embryonic Development. *Zool. Pol.*, 28(2):101-105. (Cited in EPA, 1984).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

U.S. EPA. 1987. *Drinking Water Criteria Document for Copper,* Office of Health and Environmental Assessment, ECAO. Cincinnati, OH for the ODW, Wash. D.C. External Review Draft. (Cited in HEAST).

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1984. *Health Effects Assessment for Copper,* EPA 540/1-86/025.

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* ·August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* EPA 440/5-86-001.

Weber, P.M., S. O'Reilly, M. Pollycove, and L. Shipley. 1969. Gastrointestinal Absorption of Copper: Studies with ^{64}Cu, ^{95}Zr, a Whole Body Counter and the Scintillation Camera. *J. Nucl. Med.* 10:591. (Cited in EPA, 1984).

IRON

Use

Iron is primarily used as an alloy with carbon to produce steel. Occupational exposure generally occurs during mining, transport, and ore preparation (Sittig, 1991).

Iron is present naturally as the fourth most abundant element in the Earth's crust. It is commonly present as elemental iron, iron oxide, and iron pentacarbonyl (Sittig, 1991).

Chemical and Physical Properties

AW: 55.847	BP: 2,750°C
SG: 7.86	MP: 1,535°C
Sol.(water): Insoluble	
Sol.(organics): Alcohol, ether	

Fate and Transport

Iron can be present in the atmosphere as particulate matter or in compounds that are susceptible to chemical and photochemical reactions (EPA, 1984). The predominant sources of iron in the atmosphere are natural processes such as volcanic activity and wind erosion. The principle man-made sources of iron in the atmosphere are industrial emissions and the burning of fossil fuels (EPA, 1984). Iron is removed from the atmosphere by wet and dry deposition and, to a lesser extent, by photochemical reactions (EPA, 1984).

In aquatic systems, iron is susceptible to precipitation, speciation, oxidation-reduction, and photochemical reactions. The particular reaction depends on the Ph of the body of water and the concentration of microorganisms. In more acidic waters, iron remains in solution and, as a result, is more mobile. Iron is expected to be present in the form of suspended particulates and, to a lesser extent, ions and organic complexes (EPA, 1984). The residence time of iron in aquatic media is expected to be greater than 140 years.

In soils, iron is present primarily as Fe (III). In most soils, iron is not mobile because of its high sorptive qualities. Small amounts may be transported in the form of colloidal ferric oxyhydroxides. The mobility of iron increases in more acidic soils (EPA, 1985).

Pharmacokinetics

Iron is absorbed by humans as heme iron from meats and as non-heme iron from grain and vegetables. Bjorn-Rasmussen, et al. (1974) reported that heme iron is absorbed at a rate of 37 percent whereas non-heme iron is absorbed at a rate of five percent. Iron is absorbed in the mucosal cells of the proximal duodenum and in the small intestine. Absorption is regulated by the amount of available iron already present in the body.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

In children, as little as 0.3g iron has been associated with severely toxic effects (Greenblatt, et al., 1976). Certain iron compounds, such as ferrous succinate and ferrous sulfate are severely toxic to humans when acutely exposed. Doses as small as two grams have caused fatalities. Majumder et al. (1975) reported that ferrous sulfate is much more toxic to rats and guinea pigs whose diets lack sufficient amounts of Vitamin C. Vitamin C deficient guinea pigs treated with 5 mg/day suffered severely toxic effects and mortality.

Chronic exposure to iron can result in irritation to the respiratory system and skin (ICF, 1985). Chronic ingestion is known to cause hemosiderosis and hemochromatosis. Chronic inhalation studies of steel workers have not revealed an association between iron fumes and chronic bronchitis and emphysema (EPA, 1985)

Teratogenic and Other Developmental Effects

Increased iron intake by pregnant women has resulted in only beneficial effects (EPA, 1984). No information regarding the teratogenic effects of iron were located in the available literature.

Mutagenic Effects

Demerec, et al. (1951) reported that high concentrations of ferrous or ferric chloride caused point mutations in *E.coli*. Castro, et al. (1979) reported that ferrous sulfate inhibited the transformation of Syrian hamster embryo cells.

Carcinogenic Effects

Several studies have suggested that iron oxide dust may promote the induction of cancer by known carcinogens. Iron oxide may be co-carcinogenic because of its ability to cause hyperplasia. No conclusive evidence was located in the available literature that suggests that iron compounds are carcinogenic. Some studies have indicated that iron-carbohydrate complexes such as ferric dextran may cause local tumors but, the evidence is not definitive (EPA, 1984).

Ecotoxicity

It is unlikely that iron causes any toxic effects to wildlife, however, the available data are inadequate to draw any conclusions (ICF, 1985)

Standards, Criteria and Guidelines

EPA Class D Carcinogen (elemental iron)
EPA Class C Carcinogen (iron compounds only)

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	5.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	5.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 0.3 mg/l Fish Consumption - NA

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists. 1984. *Documentation of the Threshold Limit Values.*

Bjorn-Rasmussen, E., L. Hallberg, B. Isacksson and B. Arvidsson. 1974. Food Iron Absorption in Man: Applications of the Two-Pool Extrinsic Tag Method to Measure Heme and Non-Heme Iron Absorption for the Whole Diet. *J. Clin. Invest.* 53:247-255. (Cited in EPA, 1984).

Castro, B.C., J. Meyers and J. A. DiPaolo. 1979. Enhancement of Viral Transformation for evaluating Carcinogenic or Mutagenic Potential of Inorganic Metal Salts. *Cancer Res.* 39:193-198. (Cited in EPA, 1984).

Demerec, M., G. Bertani and J. Flint. 1951. A Survey of Chemicals for Mutagenic Action In E.coli. *Am. Natur.* 85:119-136. (Cited in EPA, 1984).

Greenblatt, D.J., M.D. Allen, and J. Koch-Weser. 1976. Accidental Iron Poisoning in Childhood: Six Cases Including one Fatality. *Clin. Pediatr.* 15:835-838. (Cited in EPA, 1984).

ICF Clement Associates. 1985. *Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites.*

Majumder, A.K., B.K. Nandi, N. Subramanian, and I.B. Chatterjee. 1975. Nutrient Interrelationship of Ascorbic Acid and Iron in Rats and Guinea Pigs Fed Cereal Diets. *J. Nutr.* 105(2):240-244. (Cited in EPA, 1984).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1985. *Health Advisory for Iron.*

U.S. EPA. 1984. *Health Effects Assessment for Iron and Compounds.* EPA 540/1-86/054.

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* EPA 440/5-86-001.

LEAD

Use

Lead is a heavy metal that exists in three oxidation states (0, +2, and +4). In addition to their natural occurrence, lead and its compounds may enter and contaminate the global environment at any stage during mining, smelting, processing, and use. The annual increase in lead consumption in the United States during the 10-year period from 1962-1971 averaged 2.9 percent, largely due to increased demands for electro-chemical batteries and gasoline additives (EPA, 1984). Nonindustrial sources that may contribute to the possibility of ingestion of lead by man include the indoor use of lead-bearing paints and plasters, improperly glazed earthenware, lead fumes or ashes produced in burning lead battery casings, and exhaust from internal combustion engines (EPA, 1984).

Chemical and Physical Properties

AW: 207.19 BP: 1,704°C
SG: 11.35 at 20°C MP: 327.5°C
Sol. (Water): Insoluble
Sol. (Inorganics): HNO_3, hot H_2SO_4

Fate and Transport

Lead is artificially introduced into the environment primarily through the combustion of lead-containing fossil fuels and from lead mining operations (EPA, 1984). Lead fumes undergo decomposition when exposed to light. As a result, fumes that are present around gas stations and in heavily travelled areas are not a significant avenue of contamination (EPA, 1989). Particulate lead, carried in the atmosphere, is removed by either wet or dry deposition. Rainfall is not as significant in the deposition of lead particles as would be expected (EPA, 1984).

The transport of lead in ground water and surface water is highly variable based on its oxidation state. In polluted waters, organic complexation of lead is the primary factor in the determination of toxicity. Lead is adsorbed strongly to organic materials in soils but is not easily absorbed by living plants (EPA, 1984).

Pharmacokinetics

It has been estimated that, in man, approximately 8 percent of the lead ingested daily is absorbed. Absorption of lead consumed by humans after a 6-hour fast was increased up to 8-fold when compared with lead consumed with food. Similar effects were observed in dietary studies of mice given a dose of 3 µg Pb/kg-bw, but not at much higher doses (2,000 µg Pb/kg bw) (EPA, 1984). Numerous dietary factors influence the absorption of lead from the gastrointestinal tract. Lead absorption has been demonstrated to be enhanced by low dietary calcium or iron, high dietary fat, or low or high protein. Four baboons exposed to lead aerosols (Pb_3O_4) of varying particle size for 4 weeks showed that absorption was faster for 1.6 μm particles than for more fine particles (0.8 μm) (EPA, 1984).

In humans, it appears as though hemoglobin and hemo-proteins are affected by lead more so than any other organ or system (EPA, 1984). At levels of 0.4 μg Pb/ml blood in adults, the amount of hemoglobin and hemo-proteins produced is decreased.

Human Toxicity

Noncarcinogenic

Systemic Effects

The majority of the studies concerned with the effects of lead exposure in humans are based on blood lead levels, not ambient lead levels (EPA, 1984). Decreased hemoglobin production is seen at low blood lead levels of 0.5 μg/ml blood in children.

Chronic exposure of rats to lead acetate produced slight effects on conduction tissue excitability, systolic blood pressure, and cardiac ATP concentrations. This study was performed over a period of 20 weeks on rats given 5 mg Pb/L water in their drinking water (EPA, 1984).

Teratogenic and Other Developmental Effects

Postnatal developmental delays have been reported in pups from rats that received 50-250 mg Pb/liter in drinking water throughout gestation (EPA, 1984). Effects on reproductive parameters were noted in rats and mice in a three-generation study with 25 ppm lead (from an unspecified soluble lead salt) in drinking water. In this study, environmental concentrations of other metals were minimized (EPA, 1984). In high doses, lead compounds have been used to induce abortions. Oliver (1911) noted that the miscarriage rate among British women occupationally exposed to lead was elevated. Several other studies have reported that increases in spontaneous abortions, premature delivery, and early membrane rupture have been associated with lead exposure.

In one study, groups of 60-90, 21-day-old female CD rats were administered a semipurified, nutritionally adequate, virtually lead-free diet. Lead acetate was administered in deionized drinking water at concentrations of 0, 0.5, 5, 50, or 250 mg Pb/liter of water. The treated females were mated with untreated males after 6-7 weeks and were continued on treatment throughout gestation and lactation. There were no treatment-related differences in food or water consumption between the various treatment groups; however, body weights of offspring were depressed at the two highest doses. Sexual maturation, as measured by the time of vaginal opening, was delayed in a dose-dependent manner, with effects observed at concentrations 25 mg Pb/liter or greater (EPA, 1984).

Mutagenic Effects

DiPaolo, et al. (1978) noted that lead acetate induces cell transformation in Syrian hamster embryo cells and increases the incidence of simian adenovirus induction.

Grandjean, et al. (1983) discovered a relationship between sister-chromatid-exchange and lead exposure in workers.

Carcinogenic Effects

An increase in the incidence of renal tumors was observed in rats exposed to 1000 ppm and 2000 ppm in the diet for 2 years (Azar et al., 1973).

Similar results were observed when Kasprzak, et al. (1985) orally administered a dose of 8500 ppm Pb, as lead subacetate, per day to Sprague-Dawley rats for 79 weeks. Forty-four percent of the treated rats developed renal tumors; four of twenty-nine rats developed adenocarcinomas and the remaining nine developed adenomas. In a similar study, Koller, et al. (1986) administered 2600 ppm Pb, as lead acetate, in drinking water to Sprague-Dawley rats for 76 weeks. Eighty-one percent developed renal tubular carcinoma.

Dietary lead acetate administered in doses of 3-4 mg/day, 500-2000 mg/kg diet or 1 percent in the diet have produced renal tumors in Wistar rats (EPA, 1984). In a separate study, it was shown that a lead acetate produced renal carcinomas or adenomas in Swiss mice and several other rodents.

From available studies, it appears as though inorganic leads are the cause of any carcinogenic effects seen in humans or animals.

Ecotoxicity

Chronic toxicity studies of lead in *Daphnia magna* indicate that water hardness effects lead toxicity. The daphnids were nearly 11 times more sensitive to lead in soft water than in hard water. The chronic toxicity value of lead nitrate in water with a hardness of 52 mg/liter as $CaC0_3$ is 12.26 µg/liter. An early life stage test was conducted on the highly sensitive rainbow trout (*Salmo gairdneri*). For trout raised in water with a hardness of 28 mg/l $CaCO_3$, a chronic toxicity value of 18.80 mg/l was generated. The only chronic study located concerning saltwater species was conducted on mysid shrimp (*Mysidopsis bahia)*. The results indicate that this small crustacean is highly sensitive to lead nitrate, yielding a chronic toxicity value of 25.08 μg/liter. The aforementioned chronic values are decisive in showing that lead nitrate is highly toxic to freshwater and saltwater aquatic life (EPA, 1984).

Standards, Criteria and Guidelines

EPA Class B2 Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	NA
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	NA
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	Water and Fish Consumption - 50 μg/l Fish Consumption - NA

BIBLIOGRAPHY

Azar, A., et al. 1973. Review of lead studies in animals carried out at Haskell Laboratory-Two year feeding study and response to hemorrhage study. In: Barth, D. et al. *Environmental Health Aspects of Lead.* Proceedings International Symposium; October 1972; Amersterdam, The Netherlands. Commission of the European Communities, Luxemberg p. 199-208. (cited in IRIS).

DiPaolo, J.A., R.L. Nelson, and B.C. Casto. 1978. *In Vitro* Neoplastic Transformation of Syrian Hamster Cells by Lead Acetate and its Relevance to Environmental Carcinogenesis. *Br. J. Cancer.* 38: 452-455. (Cited in IRIS).

Grandjean, P., H.C. Wulf, and E. Neibuhr. 1983. Sister-Chromatid-Exchange in Response to Variations in Occupational Lead Exposure. *Environ. Res.* 32: 199-204. (Cited in IRIS).

Kasprzak, K.S., K.L. Hoover, and L.A. Poirier. 1985. Effects of Dietary Calcium Acetate on Lead Subacetate Carcinogenicity in Kidneys of Male Sprague-Dawley Rats. *Carcinogenesis.* 6(2): 279-282, (Cited in IRIS).

Koller, L.D., N. I. Kerkvliet, and J.H. Exon. 1986. Neoplasia Induced in Male Rats Fed Lead, 1990 Acetate, Ethyl Urea and Sodium Nitrate. *Toxicol. Pathol.* 13: 50-57. (Cited in IRIS).

Oliver, T. 1911. Lead Poisoning and the Race. *Br. Med. J.* 1: 1096-1098. (Cited in EPA, 1984.

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

U.S. EPA. 1986. *Air Quality Criteria for Lead,* June 1986 and Addendum. EPA 600/8-83-028F. (Cited in HEAST).

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1984. *Health Effects Assessment for Lead,* EPA 540/1-86/055.

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* EPA 440/5-86-001.

MANGANESE

Use

Manganese is used primarily as an alloy in steel and iron manufacturing. Manganese compounds are used in the manufacture of dry cell batteries, paints, varnishes, dyes, inks, fireworks, fertilizers, and disinfectants. Organic manganese compounds have been tested as potential supplemental anti-knock agents in gasolines (Sittig, 1991).

Chemical and Physical Properties

AW: 54.938	BP: 1962°C
SG: 7.20	MP: 1244°C
Sol. (water): decomposes	VP: 1 mmHg at 1292°C
Sol. (organics): insoluble	

Fate and Transport

Manganese occurs most often in the +2, +4 and +7 valence states. Elemental manganese, as well as manganese compounds, are present in the atmosphere as a result of natural processes.

In the atmosphere, manganese can be present in particulate form and, as such, it is susceptible to photo-chemical and thermal reactions (EPA, 1984). Manganese reacts with SO_2 and NO_2 and is removed from the atmosphere most effectively through wet and dry deposition (EPA, 1984).

In aquatic media, the fate of manganese is effected primarily by the amount of dissolved oxygen present and by the acidity of the water. In aerobic waters, manganese forms MnO_2 and Mn_3O_4 which either remain suspended or deposit to the sediments. The residence time of insoluble manganese compounds is known to be as much as 300 years (EPA, 1984).

In soils, the solubility of manganese is increased with low Ph and with high concentrations of chlorides, nitrates, or sulfates. Under these conditions, manganese is transported readily and is absorbed rapidly by plants (ICF, 1985).

Pharmacokinetics

Absorption of manganese occurs primarily in the gastrointestinal tract and is controlled homeostatically by the amount of manganese already present in the body. Under normal conditions, approximately 3 percent of ingested manganese is absorbed. Anemia victims appear to absorb more than twice that amount (EPA, 1984). Manganese absorption appears to be competitive with iron absorption.

Inhalation studies indicate that small manganese particles are absorbed in the lungs by the alveoli and are excreted within 4 days. Approximately 40-70 percent of absorbed manganese is excreted in the feces (EPA, 1984).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Orally administered manganese appears to cause minimal toxic effects in humans (ICF, 1985). The World Health Organization (WHO, 1973) reviewed several investigations which studied the effects of average daily consumption of concentrations of manganese ranging from 2 to 8.8 mg/Mn/day in adult diets. Levels from 8 to 9 mg/day were determined to be "perfectly safe". Reference doses (RfDs) were based on these studies, with a NOAEL of 0.14 mg/kg/day.

In one chronic ingestion study, Kawamura, et al. (1941) reported that 14.3 mg Mn/L drinking water causes lethargy, spasms, tremors, and mental disturbances. Both chronic inhalation and ingestion of manganese appear to effect the central nervous system most predominantly.

Teratogenic and Other Developmental Effects

Chronic manganese poisoning has been shown to cause depressed reproductive function in male and female laboratory animals. Penalver (1955) reported that oral exposure to manganese causes impotency in humans. Mandzgaladze (1967) reported that manganese exposure causes an increase in still births and spontaneous abortions in humans.

Mutagenic Effects

Manganese has been reported to be mutagenic to *Salmonella* strains and *E. coli*. Casto, et al. (1979) reported that manganese was moderately effective in enhancing viral transformation in Syrian hamster embryo cells.

Carcinogenic Effects

Manganese compounds, such as manganese chloride, manganese acetylacetonate, and manganese dioxide, caused an increased incidence of injection site tumors in rats but, EPA has determined that these results cannot be extrapolated to include elemental manganese (IRIS). No increase in lymphosarcomas and fibrosarcomas were noted by Furst (1978) in rats orally exposed to manganese powder.

Ecotoxicity

Data regarding the toxicity of manganese to aquatic organisms were not located in the available literature.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	5.0×10^{-3} mg/kg/day (water)/1.4×10^{-1} mg/kg/day (food)
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	1.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	1.14×10^{-4} mg/kg/day
MCL:	NA
AWQC:	Water and Fish Consumption - 50 µg/l Fish Consumption - 100 µg/l

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists. 1984. *Documentation of Threshold Limit Values.*

Casto, B.C., J. Meyers, and J.A. DiPaolo. 1979. Enhancement of Viral Transformation for Evaluation of the Carcinogenic or Mutagenic Potential of Inorganic Metal Salts. *Cancer Res.* 39: 193-198. (Cited in EPA, 1984).

Furst A. 1978. Tumorigenic Effects of Organomanganese Compounds on F344 Rats and Albino Mice: Brief Communications. *J. Natl. Cancer Inst.* 60: 1171-1173. (Cited in EPA, 1984).

ICF, Clement Associates. 1985. *Chemical, Physical, and Biological Properties of Compounds present at Hazardous Waste Sites.*

Kawamura, R., H. Ikuta, S. Fukuzumi, et al. 1941. Intoxication by Manganese in Well Water. Kitasato, *Arch. Exp. Med.* 18: 145-169. (Cited in EPA, 1984).

Mandzgaladze, R.N. 1967. Some Clinical and Experimental Data on the Effects of Manganese Compounds on Sexual Function. *Vopr. Gig. Tr. Prof.* 11: 126-130. (Cited in EPA, 1984).

NRC (National Research Council). 1989. *Recommended Dietary Allowances, 10th ed.* Food and Nutrition Board. National Research Council National Academy Press, Wash., D.C. p. 230-255. (Cited in HEAST).

Penalver, R. 1955. Manganese Poisoning; the 1955 Ramazzani Orations. *Med. Surg.* 24: 1-7. (Cited in EPA, 1984)

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1984. *Health Effects Assessment for Manganese and Compounds*, EPA 540/1-86/057.

U.S. EPA. *Health Effectts Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* EPA 440/5-86-001.

WHO (World Health Organization). 1973. *Trace elements in human nutrition: Manganese.* Report of a WHO Expert Committee. Technical Report Service, 532, WHO, Geneva, Switzerland p. 34-36. (Cited in IRIS).

MERCURY

Use

Mercury is used commonly in its elemental, organic, and inorganic forms.

Organic mercury, seen in both alkyl and aryl forms, is used to treat seeds for airborne diseases and is used as an additive in wood preservatives and disinfectants (Sittig, 1991). Aryl mercury compounds are also used to denature ethyl alcohol, germicides, and bactericides.

Inorganic mercury, commonly found as mercuric or mercurous salts, is utilized in gold, silver, bronze, and tin plating processes. It is also used in tanning, dyeing, felt-making, taxidermy textile manufacturing, and photography (Sittig, 1991).

In its elemental form, mercury is used as a liquid cathode in the production of chlorine and caustics. It can also be found in lamps, batteries, thermometers, and switches (Sittig, 1991).

Chemical and Physical Properties

AW: 200.59	BP: 356.8°C
SG: 13.594 at 20°C	MP: -38.87°C
Sol. (water): 81.3 μg/l at 30°C	VP: 0.0012 mmHg at 20°C

Fate and Transport

Mercury is expected to be present in the atmosphere primarily as Hg(O) from electrical industries and from the burning of fossil fuels. Elemental mercury, several inorganic species and dimethyl mercury can volatilize to the atmosphere when released to surface waters and soils (ICF, 1985). Once released to the atmosphere, mercury is removed primarily by precipitation (EPA, 1984), but certain compounds can also be photolyzed (ICF, 1985).

In aquatic environments, mercury readily adsorbs to organic matter. In waters with high organic content, sedimentation and subsequent bioaccumulation are likely to occur (ICF, 1985).

Mercury binds strongly to soils with high organic matter and, as a result, remains relatively immobile (EPA, 1984). Mercury does not transport well in ground water except when combined with leachate from municipal landfills (EPA, 1984).

Pharmacokinetics

Elemental and inorganic mercury appear to be poorly absorbed through the gastrointestinal tract lining in humans. Less than 15 percent absorption was observed by Suzuki and Tonaka

(1971) in a case study performed on individuals who accidentally ingested several grams of metallic mercury. Conversely, organic mercury, in the form of methyl mercury, is almost completely absorbed (EPA, 1984).

When inhaled by humans, approximately 80 percent of a dose of inorganic mercury is absorbed by either the alveoli or the bronchioles. Morrow, et al. (1964) reported that 40 percent of a dose of mercury, administered to dogs in the form of an aerosol, was absorbed.

When absorbed by humans, mercury is known to accumulate in the kidneys. It is excreted in both the urine and the feces (EPA, 1985).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Chronic exposure to organic mercury results mainly in adverse effects to the central nervous system in humans (EPA, 1984). The cortex, in particular the cortical neuron of the occipital lobe, is the region of the brain that is most heavily affected. Clinical symptoms of mercury poisoning include paraesthesia, sensory depression in the extremities, ataxia, and vision and hearing impairment (EPA, 1984). These data were obtained primarily through studies performed on two populations, one in Japan and one in Iraq, that accidently ingested large quantities of mercury. The procedures by which mercury concentrations were measured have been determined to be inadequate for both studies, so the doses received are inaccurate.

Fitzugh, et al. (1950) reported a 10 percent reduction in body weight in male rats after being fed diets of 160 ppm mercuric acetate for 2 years. Female rats appeared to be unaffected, and rats of both sexes appeared to be unaffected by chronic doses of less than 160 ppm. Slight damage was done, to varying degrees, to the proximal convoluted tubules in the kidneys.

Smith, et al. (1970) performed a study of 500 workers exposed to atmospheric mercury in chloroalkali plants. At low exposure concentrations (0.06-0.1 mg Hg/m^3), loss of appetite and weight loss were noted. At exposure concentrations greater than 0.1 mg Hg/m^3, tremors were observed.

In a separate study, the effects of chronic mercury inhalation in workers in a felt hat factory were observed. Mercury vapor, mercuric nitrate, and particulate elemental mercury were all found to be present in the air. Of workers exposed to concentrations greater than 0.24 mg Hg/m^3 for 20 years, 54 percent displayed observable tremors, the classic symptom of mercury poisoning (EPA, 1985).

Teratogenic and Other Developmental Effects

Prenatal exposure to methyl mercury is known to cause brain damage in humans. Numerous case studies of children, accidentally exposed to methyl/mercury through ingestion of contaminated fish, have shown a significant increase in psychomotor retardation (EPA, 1984).

Baranski and Szymczyk (1973) noted that pups of rats exposed to high concentrations of inorganic mercury vapors just before or during gestation, showed a significant increase in fatalities within 6 days after birth.

Mutagenic Effects

Five mg/liter of methyl mercury hydroxide administered to *Drosophila melanogoster* in the diet induced chromosomal aberrations. Methyl and phenyl mercury produced small increases in the rate of point mutations (Ramel, 1972).

Carcinogenic Effects

No form of mercury, either elemental, organic or inorganic, has been shown to cause cancer in humans or laboratory animals or to induce changes in cultured cells (EPA, 1985).

Ecotoxicity

Methylmercury appears to be more toxic to aquatic organisms then mercuric salts, although more testing has been done on the latter (ICF, 1985). LC_{50} values for mercuric salts range between 0.02 µg/l to 2,000 µg/l for freshwater aquatic species. In rainbow trout, methylmercuric salts were found to be approximately 10 times more toxic than mercuric salts. LC_{50} values for saltwater species range from 3.5 to 1,680 µg inorganic mercury/l. Molluscs and crustaceans, both filter feeders, appear to be more sensitive to the toxic effects of inorganic mercury than do planktonic species.

Standards, Criteria and Guidelines

EPA Class D carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	3.0×10^{-4} mg/kg/day
Chronic Inhalation RfD:	8.58×10^{-5} mg/kg/day

Subchronic Oral RfD:	3.0×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	8.58×10^{-5} mg/kg/day
MCL:	0.002 mg/l
AWQC:	Water and Fish Consumption - 0.14 µg/l Fish Consumption - 0.15 µg/l

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists. 1984. *Documentation of the Threshold Limit Values.*

Baranski, B. and I. Szymcyk. 1973. Effects of Mercury Vapors Upon Reproductive Function of the Female White Rat. *Med. Pracy.* 24(3): 249-261. (Cited in EPA, 1984).

Druet, P., et al. 1978. Immune type glomerulonephritis induced by $HgCl_2$ in the Brown Norway Rat. *Ann. Immunol.* 129c:777-792. (cited as principal oral RfD study in HEAST).

Fawer, R.F., et al. 1983. Measurement of hand tremor induced by industrial exposure to metallic mercury. *J. Ind. Med.* 40: 204-208. (cited in HEAST).

Fitzhugh, O.G., A.A. Nelson, E.P. Laug, and F.M. Kunze. 1950. Chronic Oral Toxicities of Mercuri-phenyl and Mercuric Salts. *Arch. Ind. Hyg. Occup. Med.* 2: 433-441. (Cited in EPA, 1984.

ICF, Clement Associates. 1985. *Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites.*

Morrow, P.E., F.R. Gibb and L. Johnson. 1964. Clearance of Insoluble Dust from the Lower Respiratory Tract. *Health Phys.* 10: 543-555. (Cited in EPA, 1984).

Ramel, C. 1972. Genetic Effects. In: *Mercury in the Environment - An Epidemiological and Tosicological Appraisal.* L. Frieburg and J. Vostal, Ed. CRC Press, Cleveland, OH. p. 169-181. (Cited in IRIS).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

Smith, R.G., A.J. Vorwald, L.S. Patil and T.F. Mooney, Jr. 1970. Effects of Exposure to Mercury in the Manufacture of Chlorine. *Am. Ind. Hyg. Assoc. J.* 31: 687-700. (Cited in EPA, 1984).

Suzuki, T. and A. Tonaka. 1971. Methylmercury in Maternal and Fetal Red Blood Cells. *Ind. Med.* 13: 52. (Cited in EPA, 1984).

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1985. *Health Advisory*, Office of Drinking Water.

U.S. EPA. 1984. *Health Effects Assessment for Mercury*, EPA 540/1-86/042.

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* EPA 440/5-86-001.

NICKEL

Use

Elemental nickel is used in electroplating, casting, batteries, and coinage. It is used in the manufacture of acid-resisting alloys, magnetic tapes, surgical, and dental instruments and colored ceramics and glass (Sittig, 1991). Elemental nickels most common use is as an alloy in the production of stainless steel because of its excellent corrosion resistant properties.

Nickel carbonyl, a common nickel compound, is formed in the extraction of pure nickel from ore.

Chemical and Physical Properties

AW: 58.71	BP: 2,732°C
SG: 8.90 at 25°C	MP: 1,453°C
Sol. (water): insoluble	VP: 1mm Hg at 1,810°C
Sol. (organics): variable	

Fate and Transport

Nickel is most often released to the atmosphere as dusts and fumes from smelting and processing facilities, coal burning, and diesel oil combustion (EPA, 1985). The principal removal pathways of nickel from the atmosphere are wet and dry deposition. Chemical interactions of nickel in the atmosphere generally result in elemental nickels conversion to nickel oxide (EPA, 1984).

In aquatic environments, nickel generally exists in solution as hydroxide, carbonate, sulfate, and organic complexes. The environmental fate of nickel in aquatic media appears to be dependent on the extent of pollution. In highly polluted waters, nickel is more apt to remain dissolved (EPA, 1984).

In soils, the amount of organic matter, iron oxides, and manganese oxides, may determine the fate of nickel. In soils with high iron and manganese oxide content, nickel would sorb and remain stable but, in soils with high organic content, nickel would complex and become more mobile (EPA, 1984).

Pharmacokinetics

Nickel is absorbed by humans and animals through ingestion, inhalation and, to a lesser extent, percutaneous exposure. Horak and Sunderman (1973) reported that, of the 160 to 500 μg nickel ingested daily by the average man, 1 to 10 percent is absorbed.

Absorbed nickel appears to be distributed throughout the pancreas, testes, and bones in calves and throughout the kidneys, liver, heart, and testes in rats (EPA, 1985). Nickel is transported through the body's sera primarily by serum albumin in man, rabbits, rats, and bovine (EPA, 1985). In man, it is excreted in the urine and is deposited in hair follicles. Ingested metal that is not absorbed is excreted in the feces (EPA, 1985).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Ambrose et al. (1976) exposed rats to nickel sulfate hexahydrate in concentrations of 0, 100, 1000, or 2500 ppm as Nickel in the diet for 2 years. High-dose rats exhibited decreased body weights, increased heart-to-body weight ratios and decreased liver-to-body weight ratios. Inhalation studies indicate that chronic exposure to high concentrations of nickel fumes can cause severe toxic effects, including pathological respiratory changes and death in humans. Less severe effects, including dermatitis, sinusitis, and nasal mucosal injury have been reported by workers occupationally exposed to various nickel compounds (ICF, 1985).

Teratogenic and Other Developmental Effects

Inhalation of nickel carbonyl vapors by dams caused a highly significant increase in eye malformation in newborns. The teratogenic effects of nickel carbonyl were found to be dose related (EPA, 1984).

Mutagenic Effects

Nickel carbonyl has been found to bind to liver and kidney DNA (Hui and Sunderman, 1980). Numerous studies cited in IRIS reveal that nickel subsulfide induces morphologic transformation in Syrian hamster embryos, and baby hamster kidney (BHK-21) cell cultures, sister chromatid exchange in human lymphocytes, and DNA strand breaks. As cited in IRIS, Sunderman (1984) observed nickel subsulfide to concentrate in the cell nucleus in *in vitro* assays.

Carcinogenic Effects

There have been numerous case studies performed on nickel smelting workers indicating that exposure to nickel fumes increases the chance of lung and nasal cavity tumors. Pedersen, et al. (1973) and Doll, et al. (1977) reported that nickel refinery workers exposed to 20 to 26 mg Ni/m^3 on a chronic basis developed a significantly higher number of tumors than would be expected in a normal population. In Pedersen's studies, the risk of lung cancer increased 3.75 fold and the risk of nasal cancer increased 27 fold. More recent refinery methods and more stringent occupational exposure regulations have greatly reduced the carcinogenic potential to workers.

There is not sufficient evidence concerning oral exposure to nickel to draw any conclusions.

Nickel subsulfide, nickel carbonyl, nickel oxides, and nickel sulfate are all thought to indure tumors in laboratory animals (ICF, 1985).

Ecotoxicity

Nickel tends to be more toxic to aquatic life when there are lower concentrations of iron and manganese in the water (decreased hardness). Nickel salt concentrations between 510 and 46,200 µg/L were determined to be acutely toxic to freshwater species (ICF, 1985). Saltwater algae have shown stunted growth in nickel concentrations as low as 1,000 µg/L.

Standards, Criteria and Guidelines

EPA Class A Carcinogen (refinery dust, subsulfide)
EPA Class B2 Carcinogen (carbonyl)

Oral Slope Factor:	NA
Inhalation Slope Factor:	8.4×10^{-1} (mg/kg/day)$^{-1}$ (refinery dust, subsulfide)
Chronic Oral RfD:	2.0×10^{-2} mg/kg/day (soluble salts)
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	2.0×10^{-2} mg/kg/day (soluble salts)
Subchronic Inhalation RfD:	NA
MCL:	0.1 mg/l
AWQC:	Water and Fish Consumption - 13.4 µg/L Fish Consumption - 100 µg/L

BIBLIOGRAPHY

Ambrose, A.M., et al. 1976. Long-term to toxicologic assessment of nickel in rats and dogs. *J. Food Sci. technol.* 13:181-187. (cited in IRIS).

Colborne, Roberts, et al. 1983. (full reference not given in IRIS).

Doll, R., J.D. Mathews and L.G. Morgan. 1977. Cancers of the Long and Nasal Sinuses in Nickel Workers: A Reassessment of the Period of Risk. *Br. J. Ind. Med.* 34: 102-105. (Cited in EPA, 1985).

Gilman, J.P.W. .and G.M. Ruckerbauer. 1962. Metal carcinogenisis. I. Observations on the carcinogenicity of a refinery dust, cobalt oxide, and colloidal thorium dioxide. *Cancer Res.* 22: 152-157. (cited in IRIS).

Horak, E. and F. W. Sunderman, Jr. 1973. Fecal Nickel Excretion by Healthy Adults. *Clin. Chem.* 19: 429-430. (Cited in EPA, 1985).

Hui, G. and F.W. Sunderman. 1980. Effects of nickel compounds on incorporation of thymidine-34 into DNA in rat liver and kidney. *Carcinogenesis.* 1:297-304. (Cited in IRIS).

ICF, Clement Associates. 1985. *Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites.*

Pederson, E., A. Andersen and H. Hogetveit. 1973. Cancer of Respiratory Organs Among Workers at a Nickel Refinery in Norway. *Int. J. Cancer.* 12: 32-41. (Cited in EPA, 1985).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. *Health Advisory for Nickel, Office of Drinking Water.*

U.S. EPA. *Health Effects Assessment for Nickel.* EPA 540/1-86/018.

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* 2 EPA 440/5-86-001.

THALLIUM

Use

Thallium and its compounds are used as catalysts in certain organic reactions, in phosphor activators, in bromoiodide crystals for lenses, plates, and prisms in infrared optical instruments, in photoelectric cells, in mineralogical analysis; in alloys with mercury in low-temperature thermometers, switches and closures; in high-density liquids, dyes and pigments; and in the manufacture of optical lenses, fireworks, and imitation precious jewelry. It forms a stainless alloy with silver and a corrosion resistant alloy with lead. Its medicinal use for epilation is almost discontinued (Sittig, 1991). Prior to 1972, thallium and its compounds were used as rodenticides, fungicides, and insecticides (Sittig, 1991; Stokinger, 1981).

Physical and Chemical Properties

AW:	204.37	MP:	303.5°C
SG:	11.85 g/mc^3	BP:	1457°C
VP:	10 mm Hg at 1000°C		
Sol. (water):	insoluble		

Fate and Transport

Cement factories, coal burning power plants, and metal smelters are the principal sources of thallium in the environment (Sharma et al., 1986; Brockhaus et al., 1980, 1981). Atmospherically, thallium may be present in its elemental form, as oxides of Tl, as Tl_2S, or Tl_2So_4. Tl_2S is likely to be speciated to Tl_2SO_4 and Tl_2O will be rapidly hydrolyzed to TlOH by the moisture in the atmosphere (U.S. EPA, 1988).

Both TlOH and Tl_2SO_4 are most likely removed from the atmosphere by wet deposition given their water solubilities. Tl_2O_3, however, may persist in the atmosphere longer because it is insoluble in water and, therefore, will be removed by dry deposition (U.S. EPA, 1988).

In aquatic systems, insoluble forms of thallium will accumulate in the sediment (Mathis and Kevern, 1975). Kempton et al. (1987a,b) reports that thallium may be removed from the water by sorption onto suspended solids in water. Most of the soluble thallium that enters aquatic systems will remain in the soluble state due to its formation of soluble complexes with inorganic and organic ligands (Stephenson and Lester, 1987a,b). These complexes are even more stable at higher pHs (O'Shea and Mancy, 1978). Wallwork-Barber et al. 91985) report that thallium in water may be transported to fish and vegetation. The bioconcentration factor of thallium in whole aquatic organisms ranges from 12-34 (Zitko and Carson, 1975; Barrows et al., 1980).

In soils, leaching of thallium, particularly from sandy soils, appears to be likely given its transport in water (U.S. EPA, 1988). Cataldo and Wildung (1983) report that up to 10 percent of the thallium absorbed in plant roots from soil may be transported from the root to the shoot of the plant.

Pharmacokinetics

U.S. EPA (1988) reports that numerous studies reveal that absorption of soluble thallium by any route of exposure is rapid and virtually complete, although dermal absorption is not likely to be significant in environmental exposure. Several studies indicate that distribution of thallium from the blood is rapid and widespread; with highest levels detected in the kidney, heart, and liver; and lowest levels detected in the nervous system and body fat. Lie et al. (1960) reports that the relative concentrations in different tissues appear to be independent of the route of administration and the time after administration. In addition, Sabbioni et al. (1980) and Gregus and Klaasen (1986) found no correlation between tissue concentrations and the valance of thallium administered or the dosage, respectively. The U.S. EPA (1988) reports that several studies indicate that thallium translocates to the placenta and fetus, but levels in the fetus are substantially lower than those in maternal tissues. Sabbioni et al. (1980) hypothesizes that thallium *in vivo* is transformed to one oxidation state. Barclay et al. (1953) and Richelmi et al. (1980) report that, in humans, excretion of thallium occurs predominantly in the urine. A range of estimated excretion half-lives have been reported with Talas et al. (1983) reporting 2.15 days for tracer doses in ambulatory heart patients and Barclay et al. (1953) and U.S. EPA (1980) reporting 21.7 days in a terminal cancer patient.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

U.S. EPA (1988) reports that thallium salts are potent poisons that cause acute toxicity in humans. Accidental ingestion of thallium salt rodenticides and insecticides, internal and topical use of thallium as a depilatory agent have all resulted in human poisoning (Gettler and Weiss, 1943; Moeschlin, 1980). In children, Bedford (1928) reports that acute toxicity appears to be approximately 6 mg thallium/kg/day. Moeschlin (1980) reports approximately 8-12 mg thallium/kg as the average lethal dose for adults. Independent of the species or the type of thallium salt administered, U.S. EPA (1988) reports that the acute oral LD_{50} values in rats and mice range from 16 to 35 mg thallium/kg.

Chronic oral exposure of a population living in the vicinity of a cement factory that discharged large quantities of thallium into the atmosphere through the ingestion of fruits and vegetables grown in the area appears to have resulted in an increased incidence of neurological and subjective symptoms (Brockhaus et al., 1980, 1981; Dolgner et al., 1983). Subchronic oral exposure of laboratory animals to concentrations greater than 0.25 mg/kg/day resulted in neurological and skeletal muscle effects (Mazo et al., 1983, Deshimaru et al., 1977), hair loss, elevated kidney weights, body weight loss, and mortality (Downs et al., 1960).

U.S. EPA (1979) exposed rats intermittently to thallium (III) oxide via inhalation at 0.5 - 2.0 mg/m$_3$. Deteriorating health and increased mortality were observed. However, no adverse health effects were observed in workers occupationally exposed to thallium in a magnesium seawater battery plant (Marcus, 1985) or in cement production (Schaller et al., 1980); Ludolph et al., 1986).

Teratogenic and Other Developmental Effects

U.S. EPA (1988) reports on numerous studies which indicate that thallium results in achondroplastic malformations when injected into developing chicken eggs, or tested in mammalian whole embryo cultures or limb bud cultures. Gibson and Becker (1970) observed reduced fetal body weight, hydronephrosis, and the absence of vertebral bodies following parenteral administration of greater than 2 mg thallium/kg/day to pregnant rats. A slight increase in fetal loss was observed following oral administration of thallium to rats (≥2 mg/kg/day) and mice (≥ 4 mg/kg/day) (Roll and Matthiaschk, 1981). Reduced survival at weaning in both species and reduced growth rate in mice were observed in the offspring of rats and mice allowed to deliver, as well. Bornhausen and Hagen (1984) report that adult offspring of dams treated with thallium during gestation had significant learning deficits in a lever-pressing behavior conditioning test.

U.S. EPA (1988) reports that adult male rats exposed to 0.74 mg thallium/kg/day in the drinking water had decreased sperm motility, inhibition of ß-glucoronidase activity, and histopathological alterations of the testes after 60 days of exposure but not after 30 days.

Mutagenic Effects

Data on the mutagenicity of thallium is mixed. Negative results have been obtained in reverse mutation tests (Kanematsu et al., 1980; Singh, 1983) and in tests for effects on cell division (Loveless et al., 1954). Positive results were obtained in a rec assay (Kanematsu et al., 1980) and in several mammalian test systems, including a dominant lethal test in male rats (Zasukhina et al., 1983).

Carcinogenic Effects

Data regarding the carcinogenicity of thallium were not available in the literature reviewed.

Ecotoxicity

In freshwater aquatic systems, U.S. EPA (1980) reports that acute sensitivity of *Daphnia magna* and the fathead minnow to thallium were similar, with LC_{50} values in the range of 910 to 2180 µg/l. LC_{50} values for the bluegill were approximately two orders of magnitude higher. *Daphnia magna* and the fathead minnow also had similar chronic values; 130 and 57 µg Tl/l, respectively. Exposure of an alga to 110 and 100 µg Tl/l resulted in a 50 percent reduction in chlorophyll *a* and cell numbers, respectively. Atlantic salmon had the highest bioconcentration factor for fishes with a value of 130 for muscle tissue. This species appears to be particularly sensitive to thallium; concentrations as low as 20 µg/l resulted in partial mortality after about 100 days exposure.

In saltwater systems, the mysid shrimp had the greatest acute sensitivity with an LC_{50} of 2130 µg thallium/l. The sheepshead minnow and tidewater silverside had exhibited similar sensitivity to thallium with 96-hour LC_{50} values of 20,900 µg/l and 24,000 µg/l, respectively. 8,400 µg/l produced chronic effects in the sheepshead minnow. 4,080 µg/l resulted in a 50 percent reduction in photosynthesis in a saltwater algal species. Bioconcentration factors less than 20 were observed in two bivalve species exposed for 40 to 88 days.

No data on the ecotoxicity of thallium in terrestrial systems were available in the literature reviewed.

Standards, Criteria and Guidelines

Unclassified by EPA as to carcinogenicity

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	7.0×10^{-5} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic OraL RfD:	7.0×10^{-4} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL	0.002
AWQC:	Water and Fish Consumption: 13 µg/l Fish Consumption: 48 µg/l

BIBLIOGRAPHY

Barclay, R.K., et al. 1953. Distribution and excretion of radioactive thallium in the chick embryo, rat and man. *J. Pharmacol. Exp. Ther.* 107: 178-187. (cited in HEED).

Barrows, M.E., et al. 1980. Bioconcentration and elimination of selected water pollutants by blue gill sunfish (*Lepomis macrochirus*). In: *Symp. Dyn. Exposure Hazard Assess. Toxic Chem.*, 1978. Ann Arbor Science, Ann Arbor, MI, p. 379-392. (cited in HEED).

Bornhausen, M. and U. Hagen. 1984. Operant behavior performance changes in rats after prenatal and postnatal exposure to heavy metals. *IRCS Med. Sci.* 12: 805-806. (cited in HEED).

Brockhaus, A., et al. 1980. Excessive thallium absorption among a population living near a thallium emitting cement plant. In: *Mechanisms of Toxicity and Hazard Evaluation.* B. Holmstedt, R. Lauwerys, M. Mercier and M. Roberfroid, Ed. Elsevier/North-Holland Biomedical Press, The Netherlands. p. 565-568. (cited in HEED).

Brockhaus, A., et al. 1981. Intake and health effects of thallium among a population living in the vicinity of a cement plant emitting thallium containing dust. *Int. Arch. Occup. Environ. Health.* 48: 375-389. (cited in HEED).

Cataldo, D. and R. Wildung. 1983. The role of soil and plant metabolic processes in controlling trace element behavior and bioavailability to animals. *Sci. Total Environ.* 28: 159-168. (cited in HEED).

Deshimaru, M., et al. 1977. Electron microscopic study of experimental thallotoxicosis. *Folia Psych. Neurol. Jpn.* 31: 269-275. (cited in HEED).

Dolgner, R., et al. 1983. Repeated surveillance of exposure to thallium in a population living in the vicinity of a cement plant emitting dust containing thallium. *Int. Arch. Occup. Environ. Health.* 52: 79-94. (cited in HEED).

Downs, W.L., et al. 1960. Acute and subacute toxicity studies of thallium compounds. *Am. Ind. Hyg. Assoc.* 21: 399-406. (cited in HEED).

Gettler, A.O. and L. Weiss. 1943. Thallium poisoning. III. Clinical toxicology of thallium. *Am. J. Clin. Pathol.* 13: 422-429. (cited in HEED).

Gibson, J.E. and B.A. Becker. 1970. Placental transfer, embrytoxicity and teratogenicity of thallium sulfate in normal and potassium-deficient rats. *Toxicol. Appl. Pharmacol.* 16: 120-132. (cited in HEED).

Gregus, Z. and C.D. Klaassen. 1986. Disposition of metals in rats: A comparative study of fecal, urinary and biliary excretion and tissue distribution of eighteen metals. *Toxicol. Appl. Pharmacol.* 85: 24-38. (cited in HEED).

Kanematsu, N., et al. 1980. Rec assay and mutagenicity studies on metal compounds. 77: 109-116. (cited in HEED).

Kempton, S., et al. 1987a. Heavy metal removal in primary sedimentation. I. The influence of metal solubility. *Sci. Total Environ.* 63: 247-258. (cited in HEED).

Kempton, S., et al. 1987b. Heavy metal removal in primary sedimentation. II. The influence of metal speciation and particle size distribution. *Sci. Total Environ.* 63:. 231-246. (cited in HEED).

Lie, R., et al. 1960. The distribution and excretion of thallium-204 in the rat with suggested MPC's and a bioassay procedure. *Health Phys.* 2: 334-340. (cited in HEED).

Loveless, L.E., et al. 1954. Survey of effects of chemical on division and growth of yeast and *Escherichia coli. J. Bacteriol.* 68: 637-644. (cited in HEED).

Ludolph, A., et al. 1986. Chronic thallium exposure in cement plant workers: Clinical and electrophysiological data. *Trace Elem. Med.* 3(3): 121-125. (cited in HEED).

Manzo, L., et al. 1983. *Environmental toxicology research on thallium: Metabolic and toxicological studies in the rat as carried out by nuclear and radioanalytical methods.* NTIS PB 83-133249. (cited in HEED).

Mathis, B.J. and N.R. Kevern. 1975. Distribution of mercury, cadmium, lead, and thallium in a eutrophic lake. *Hydrobiologia.* 46(2-3): 207-222. (cited in HEED).

MRI (Midwest Research Institute). 1986. *Subchronic (90-day) toxicity study of thallium sulfate in Sprague-Dawley rats.* Office of Solid Waste, U.S. EPA, Wash., D.C. (cited in HEAST).

Moeschlin, S. 1980. Thallium poisoning. *Clin. Toxicol.* 17: 133-146. (cited in HEED).

O'Shea, T.A. and K.H. Mancy. 1978. The effect of pH and hardness of metal ions on the competitive interaction between trace metal ions and inorganic and organic complexing agents found in natural waters. *Water Res.* 12(9): 703-711. 1978 (cited in HEED).

Richelmi, P., et al. 1980. Salivary levels of thallium in acute human poisoning. *Arch. Toxicol.* 43: 321-325. (cited in HEED).

Roll, R. and G. Matthiaschk. 1981. Investigations on embryotoxic effects of thallium chloride and thallium acetate in mice and rats. *Teratology.* 24(2): 46A-47A. (cited in HEED).

Sabbioni, E., et al. 1980. Metabolic fate of different inorganic and organic species of thallium in the rat. *Sci. Total Environ.* 15(2): 123-135. (cited in HEED).

Schaller, K.H., et al. 1980. Investigations of thallim-exposed workers in cement factories. *Int. Arch. Occup. Environ. Health.* 47(3): 223-321. (cited in HEED).

Sharma, J., et al. 1986. Hazards and analysis of thallium - A review. *Toxicol. Environ. Chem.* 11: 93-116. (cited in HEED).

Singh, I. 1983. Induction of reverse mutation and mitotic gene conversion by some metal compounds in *Saccharomyces cerevisiae. Mutat. Res.* 117: 149-152. (cited in HEED).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

Stephenson, T. and J.N. Lester. 1987a. Heavy metal behavior during the activated sludge process. I. Extent of soluble and insoluble metal removal. *Sci. Total Environ.* 63: 199-214. (cited in HEED).

Stephenson, T. and J.N. Lester. 1987b. Heavy metal behavior during the activated sludge process. II. Insoluble metal removal mechanisms. *Sci. Total Environ.* 63: 215-231. (cited in HEED).

Stokinger, H.E. 1981. The metals: 32 thallium, Tl. In: *Patty's Industrial Hygiene and Toxicology, Vol. 2A, 3rd Ed.*, G.C. Clayton and F.E. Clayton, Ed. John Wiley and Sons, Inc., New York. p. 1914-1931. (cited in HEED).

Talas, A. and H.H. Wellhoener. 1983. Dose-dependency of thallium (1+) kinetics as studied in rabbits. *Arch. Toxicol.* 53(1): 9-16. (cited in HEED).

U.S. EPA. 1979. Study of Carcinogenicity and Toxicity of Inhaled Antimony Trioxide, Antimony Ore Concentration and Thallic Oxide in Rats. MRI Project No. 4466-B. Contract No. 210-77-0156. OTS Fiche No. 0511065. (cited in HEED).

U.S. EPA. 1980. *Ambient Water Quality Criteria Document for Thallium.*

U.S. EPA. 1988. *Health and Environmental Effects Document (HEED).*

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* EPA 440/5-86-001.

Wallwork-Barber, M.K., et al. 1985. Thallium movement in a simple aquatic ecosystem. *J. Environ. Sci. Health, Part A.* 20: 689-700. (cited in HEED).

Zasukhina, G.D., et al. 1983. Mutagenic effect of thallium and mercury salts on rodent cells with different repair activities. *Mutat. Res.* 124(2): 163-173. (cited in HEED).

Zitko, V. and W.V. Carson. 1975. Accumulation of thallium in clams and mussels. *Bull. Environ. Contam. Toxicol.* 14(5): 530-533. (cited in HEED).

VANADIUM

Use

Vanadium pentoxide is used as a catalyst in the production of several industrial chemicals. It is also used as a photographic developer, as a coating for welding electrodes, and as an alloying agent (ACGIH, 1984).

Chemical and Physical Properties

AW: 50.9 BP: 3,380°C
SG: 5.96 at 20°C MP: 1,890°C
Sol. (water): at 20°C insoluble
Sol. (organics): insoluble

Fate and Transport

The environmental fate of vanadium varies with each compound. Some compounds are volatile so, atmospheric transport would be a legitimate fate process (ICF, 1985). Vanadium appears to become more water soluble in acidic soils, thus becoming more leachable, and is known to bioaccumulate slightly.

Pharmacokinetics

Vanadium is thought to be stored primarily in fat and blood serum but has been detected in the lungs and intestines in humans (U.S. HEW, 1969).

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

The principle systemic effects of chronic exposure to vanadium are the irritation of the skin and eyes. Oral exposure to vanadium is known to cause gastrointestinal disturbances. Inhalation exposure to vanadium is known to cause irritation of the lungs and after repeated exposures difficulty in breathing and bronchitis are known to occur (NIOSH, 1977). Vanadium's toxicity seems to increase with the increase in valence number (ICF, 1985).

Teratogenic and Other Developmental Effects/Mutagenic Effects

Vanadium and its compounds have not displayed mutagenic, teratogenic, or developmental effects in several studies performed on laboratory animals (ICF, 1985).

Carcinogenic Effects

Vanadium is not classified as to human carcinogenicity because of insufficient human or animal data. In one study, researchers exposed Swiss mice to vanadyl sulfate at concentrations of 19.8 mg/kg bw for their lifetime. There was no evidence that vanadyl sulfate caused tumors in the mice (NIOSH, 1977). Numerous other studies have resulted with similar conclusions (NIOSH, 1977).

Ecotoxicity

Freshwater organisms have LC_{50} values ranging between 5,000 and 100,000 µg/L. The average LC_{50} value for freshwater organisms is around 10,000 µg/L (ICF, 1985).

No further data regarding the ecotoxicity to wildlife were located.

Standards, Criteria and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhaltion Slope Factor:	NA
Chronic Oral RfD:	7.0×10^{-3} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	7.0×10^{-3} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists. 1984. *Documentation of the Threshold Limitation Values.*

ICF, Clement Associates. 1985. *Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites.*

NIOSH. 1977. *Criteria for a Recommended Standard for The Occupational Exposure to Vanadium.*

Schroeder, J.A., et al. 1970. Zirconium, niobium, antium, antimony, Vanadium, and lead in rats: Life-term studies. *J. Nutr.* 100(1): 59-68. (cited as principal oral RfD study in HEAST).

Sittig, M. 1991. *Handbook of Toxic and Hazardous Chemicals.*

U.S. Dept. of Health, Education and Welfare (U.S. HEW). 1969. *Preliminary Air Pollution Survey of Vanadium and Its Compounds.*

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

ZINC

Use

Zinc is a bluish-white, lustrous metal with a distorted hexagonal close-packed structure.

It is used in a variety of manners including, galvanizing sheet iron; as an ingredient of such alloys as bronze, brass, Babbitt metal, German silver, and special alloys for die-casting. It is used as a protective coating for other metals to prevent corrosion; for electrical apparatus, especially dry cell batteries; household utensils; castings; printing plates; building materials; railroad car linings; and automotive equipment. Additionally, zinc is utilized as a reducing agent in organic chemistry; for deoxidizing bronze; extracting gold by the cyanide process; purifying fats for soap; bleaching bone glue; manufacturing sodium hydrosulfite; insulin zinc salts, and as a reagent in analytical chemistry (Windholz, 1983).

Physical and Chemical Properties

AW:	65.38	BP:	907°C
SG:	7.133 at 25°C	MP:	419.58°C

Sol. (water): insoluble in water, some salts are soluble
Sol. (organics): soluble in acid and alkali

Fate and Transport

Zinc is likely to be present in the atmosphere as dust and fumes from zinc production facilities, lead smelts, brass works, automobile emissions, fuel combustion, incineration and soil erosion (Lloyd and Showak, 1984). The U.S. EPA (1984) reports that conversion of zinc into a stable species such as zinc oxide, and not removal through decomposition, may be the fate of atmospheric zinc. Fishbein (1981) reports that atmospheric interactions are minimal for particulates with large aerodynamic diameters because of their short air residence time. Zinc, however, is found at the highest concentrations in particles with an aerodynamic diameter less than 3μm (Fishbein, 1981) and zinc oxide emitted from high-temperature processes (e.g., brass foundries, galvanizing, smelting and welding processes) may have particle sizes in the range of 0.01-0.4μm (NIOSH, 1975). These smaller particles may have a long residence time making speciation (conversion) more likely.

Callahan et al. (1979) reports that sorption is probably the dominant fate of zinc in the aquatic environment. The U.S. EPA (1984) reports that zinc introduced into the aquatic environment is partitioned into sediments through sorption onto hydrous iron and manganese oxides, clay minerals and organic material; and that a small part may be partitioned into the aquatic phase through speciation into soluble zinc compounds. They also report that precipitation of zinc sulfide is an important control on the mobility of zinc in reducing environments; and precipitation of hydroxides, carbonate, or basic sulfate may occur at high zinc concentration. Although they have a tendency to be absorbed more strongly on the sediments, organic and inorganic ligand complexes may increase the mobility of zinc in aquatic media (U.S. EPA, 1984).

The U.S. EPA (1984) states that zinc is likely to be strongly sorbed onto soil and, if not sorbed, leaching may occur. pH and salinity affect sorption with decreasing pH and increasing salinity factoring desorption (U.S. EPA, 1980). Page (1981) detected zinc in 100 percent of ground water samples from New Jersey indicating that leaching is prevalent.

Pharmacokinetics

The U.S. EPA (1980) states that gastrointestinal absorption of zinc is dependent in part upon the zinc status of the organism. In reference to this statement, the U.S. EPA (1984) states:

> "This is a reasonable conjecture, in that zinc levels in the body are rigidly controlled by various homeostatic mechanisms. Also, it appears that dietary levels of other nutrients may influence the kinetics of zinc absorption. The fact that zinc is excreted, in part, through the gastrointestinal tract complicates quantitation of zinc uptake. It is also likely that the anion associated with zinc, chelation or other complexing moieties may influence gastrointestinal absorption."

^{65}Zn, as the chloride, was shown to be rapidly absorbed in human volunteers by Spencer et al. (1965). Peak plasma values were achieved within 4 hours and average absorption was 50 percent with values ranging from 20-80 percent NRC (1978) also reported varying degrees of absorption. Contrary to this, Stokinger (1981) only found small amounts of zinc being absorbed by laboratory animals.

It appears that dietary protein uptake may enhance zinc uptake (NCR, 1978), while high dietary levels of phytate, a complex organic phosphorus-containing compound in cereal products, may inhibit zinc uptake (U.S. EPA, 1984). However, Arvidsson et al. (1978) found that phytate had little or no influence on zinc uptake when ^{65}Zn was added to bread during baking that was then fed to 11 human subjects. Sandstead et al. (1978) suggests that dietary fiber content may influence the uptake of zinc.

Richards and Cousins (1977) speculate that metallothionen, a low-molecular-weight metal-binding protein in the intestinal mucosa, may bind with zinc and facilitate absorption.

Human Toxicity

Noncarcinogenic Effects

Systemic Effects

Brown et al. (1964) found that high zinc levels in foods stored in galvanized containers led to severe diarrhea, abdominal cramping, nausea and vomiting upon consumption. Murphy (1970) reported lethargy in a 16-yr. old boy administered 12g of zinc in peanut butter over a 2-day period (in a belief that it would accelerate wound healing). Anemia was observed in three children exposed to zinc from toy cars made of zinc alloy. The children played with the cars in the bath and probably ingested some of the bath water. The children were excreting greater than 1 mg/liter zinc in urine.

Ten young men given 150 mg zinc sulfate for 43-61 days to accelerate wound healing complained of gastric discomfort but no other adverse effects were observed, wound healing was accelerated (Pories et al., 1967). Greaves and Skillen (1977), as well, found no adverse effects resulting from administration of 150 mg zinc sulfate to 18 patients.

Prasad et al. (1978) found that prolonged zinc therapy for sickle-cell anemia reduced ceruloplasmin levels to 50 percent of what they had been before therapy. Ceruloplasmin levels were returned to normal by discontinuation of the therapy.

Teratogenic and Other Development Effects

Cox et al. (1969) and Ketcheson et al. (1969) reported reduced copper content in fetal livers (and other tissues) as the only effect resulting from administering 4000 or 5000 ppm zinc to pregnant rats during gestation.

Of a "small group" of women supplementing their diet with 100 mg zinc sulfate during the third trimester of pregnancy, 3 experienced premature deliveries and 1 delivered a still born infant (Kumar, 1976). Kumar (1976) supplemented rats with "100 ppm zinc orally" and found a "significant increase" in the number of fetal resorptions.

Mutagenic Effects

No data regarding the mutagenicity of zinc were found in the literature reviewed.

Carcinogenic Effects

Wallenius et al. (1979) exposed female rats to diets containing 15, 50, or 200 ppm zinc. The palatal mucosa was then painted with 4-nitro-quinoline-n-oxide 3 times/week to induce cancer. After cancer of the palate became grossly visible the animals were killed. Animals exposed to 200 ppm dietary zinc developed macroscopically detectable cancer earlier than rats exposed to the two lower doses. However, in an identical study, Mathur et al. (1979) exposed rats to dietary zinc concentration of 5.9, 50, and 260 ppm. Palatal mucosa was sampled at 3, 9, 13, and

23 weeks after exposure, at which time all rats were killed and examined. Animals on the zinc-deficient diet showed the most advanced histologic changes after 3 weeks. After 20 weeks, cancers were found in both the zinc-deficient and zinc-supplemented groups while the rats on the adequate (50 ppm zinc) diet evidenced only moderate dysplasia.

Ecotoxicity

Acute toxicity of zinc to freshwater aquatic life is dependent on the hardness of the water. The concentration (μg/liter) should not exceed the numerical value given by $e^{(0.83 [\ln (\text{hardness})] + 1.95)}$. The U.S. EPA (1986) gives the following examples: "at hardnesses of 50, 100, and 200 mg/liter $CaCO_3$, the concentration of total recoverable zinc should not exceed 180, 320, and 570 μg/liter at any time". The 24-hour average concentration should not exceed 47 μg/liter (U.S. EPA, 1986).

In saltwater systems, the 24-hour average concentration should not exceed 58 μg/liter and the concentration at any time should not exceed 170 μg/liter (U.S. EPA, 1986).

ICF (1985) reports that zinc poisoning has occurred in cattle. Poisoning was caused by an accidental contamination of food in one outbreak with a zinc concentration of 20 g/kg. It was estimated that the cows had a daily intake of 140 g for about 2 days. The exposed cows exhibited severe enteritis, some died and some had to be slaughtered. Severe pulmonary emphysema with changes in the myocardium, kidneys, and liver with extremely high concentrations of zinc in the liver were detected in post-mortem studies.

Standards, Criteria, and Guidelines

EPA Class D Carcinogen

Oral Slope Factor:	NA
Inhalation Slope Factor:	NA
Chronic Oral RfD:	3.0×10^{-1} mg/kg/day
Chronic Inhalation RfD:	NA
Subchronic Oral RfD:	3.0×10^{-1} mg/kg/day
Subchronic Inhalation RfD:	NA
MCL:	NA
AWQC:	NA

BIBLIOGRAPHY

Arvidsson, B., et al. 1978. A radionuclide technique for studies of zinc absorption in man. *Int. J. Nucl. Med. Biol.* 5:104. (cited in HEA, 1984).

Brown, M.A. et al. 1964. Food poisoning involving zinc contamination. *Arch. Environ. Health.* 8:657. (cited in HEA, 1984).

Callahan, M.A., et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants, Vol. I.* U.S. EPA, Office of Water Planning and Standards, Office of Water and Waste Management, Washington, DC EPA/440/4-79-029a. (cited in HEA, 1984).

Cox, D.H., et al. 1969. Excess dietary zinc for maternal rat and zinc, iron, copper, calcium and magnesium content and enzyme activity in maternal and fetal tissues. *J. Nutr.* 98:459. (cited in HEA, 1984).

Fishebein, L. 1981. Sources, transport, and alternations of metal compounds: an overview. I. arsenic, beryllium, cadmium, copper, chromium, and nickel. *Environ. Health Perspec.* 40:43-64. 1981 (cited in HEA, 1984).

Geaves, M.W. and A.W. Skillen. 1970. Effects of long-continued ingestion of zinc sulphate in patients with venous leg ulceration. *Lancet.* p. 889-891. 1970 (cited in HEA, 1984).

ICF Clement Assoc. 1985. *Chemical Physical, and Biological Properties of Chemical Present at Hazardous Waste Sites.*

Ketcheson, M.R., et al. 1969. Relationships of maternal dietary zinc during gestation and lactation to development and zinc, iron, and copper content of the postnatal rat. *J. Nutr.* 98:303. (cited in HEA, 1984).

Kumar, S. 1976. Effects of zinc supplementation on rats during pregnancy. *Nutr. Rep. Int.* 13:33. 1976 (cited in HEA, 1984).

Lloyd, T.B and W. Showak. 1984. Zinc, and zinc alloys. *Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed.* Vol. 24, M. Grayson and D. Ecktroth, ed. J. Wiley and Sons, NY p. 807-851. (cited in HEA, 1984).

Mathur, A., et al. 1979. Influence of zinc on onset and progression of oral carcinogenesis in rats. *Acta Odontological Scandinavia* 37:3777. (cited in HEA, 1984).

NIOSH (National Institute for Occupational Safety and Health). 1975. *Criteria for a Recommended Standard...Occupational Exposure to Zinc Oxide* U.S. DHEW, PHS, CDC, NIOSH; Cincinnati, OH. NIOSH No. 76-104. (cited in HEA, 1984).

NRC (National Research Council). 1978. *Zinc* University Park Press, Baltimore, MD. (cited in HEA, 1984).

Page, W.G. 1981. Comparison of ground water and surface water for patterns and levels of contamination by toxic substances. *Environ. Sci. Technol.* 15:1475-1481. (cited in HEA, 1984).

Pories, W.J., et al. 1967. Acceleration of wound healing in man with zinc sulfate given by mouth. *Lancet* 1:121-124. (cited in HEA, 1984).

Prasad, A.S., et al. 1975. Zinc deficiency in sickle cell disease. *Clin. Chem.* 21:582-587. (cited in HEA, 1984).

Richards, M.P. and R.J. Cousins. 1977. Isolation of an intestinal metallothionen indiced by parenteral zinc. *Biochem. Biophys. Res. Common.* (cited in HEA, 1984).

Sandstead, H.H., et al. 1978. Influence of dietary fiber on trace element balance. *Am. J. Clin. Nutr.* 31:5180. (cited in HEA, 1984).

Spencer, H., et al. 1965. Metabolism of zin-65 in man. *Radiat. Res.* 24:432. (cited in HEA, 1984).

Stokinger, H.E. 1981. The Metals. In: *Patty's Industrial Hygiene and Toxicology, 3rd ed.*, Vol. 11A, G.D. Clayton and F.E. Clayton, Ed. J. Wiley and Sons, Inc. N.Y. p. 2033-2049. (cited in HEA, 1984).

U.S. EPA. *Drinking Water Regulations and Health Advisories.*

U.S. EPA. 1986. *Health Effects Assessment for Zinc.* EPA 540/1-86/048.

U.S. EPA. *Health Effects Assessment Summary Tables (HEAST).*

U.S. EPA. *Integrated Risk Information System (IRIS).*

U.S. EPA, OERR. *CERCLA Compliance With Other Laws Manual Interim Final.* August, 1988.

U.S. EPA. 1986. *Quality Criteria for Water.* EPA 440/5-86-001.

Wallenius, K., et al. 1979. Effect of different levels of dietary zinc on development of chemically inducted oral cancer in rats. *Int. J. Oral. Sur.* 8:56. (cited in HEA, 1984).

Windholz, M. Ed. 1983. *The Merck Index, 10th ed.*

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APPENDIX D

SHOWER INHALATION MODEL

Supplemental Equations for Estimating Volatile Emissions from Showering
(From: Foster and Chrostowski, 1987)

1. Gas-film mass transfer coefficient (cm/hr), termed "Kv", of a given VOC

$$Kv_{VOC} = Kv_{water}\,(18/MW_{VOC})^{0.5}$$

where: Kv_{water} = 3,000 cm/hr
MW_{VOC} = molecular weight (g/mole)

2. Liquid-film mass transfer coefficient (cm/hr), termed "Kl"

$$Kl_{VOC} = Kl_{CO2}\,(44/MW_{VOC})^{0.5}$$

where: Kl_{CO2} = 20 cm/hr

3. Adjusted overall mass transfer coefficient (cm/hr), termed "Ka"

$$Ka = \left[\frac{1}{Kl} + \frac{RT}{HKv}\right]^{-1}\left[\frac{Tl}{Ts}\,\frac{\mu s}{\mu l}\right]^{-0.5}$$

where:
Kl = liquid-film mass transfer coefficient (cm/hr)
R = gas constant (8.2×10^{-5} atm-m^3/mole-K)
T = calibration water temperature (293 K)
H = Henry's Law constant (atm-m^3/mole)
Kv = gas-film mass transfer coefficient (cm/hr)
Tl = calibration water temperature (293 K)
μs = water viscosity at Ts (0.5996 cp)
Ts = shower water temperature (318 K)
μl = water viscosity at Tl (1.0 cp)

4. Concentration emitted from shower droplet, termed "Cwd"

$$Cwd = Cwo\,(1-\exp[-Ka\ ts/60\ d])$$

where:
Cwd = VOC conc. emitted from shower droplet (mg/l)
Cwo = VOC conc. in shower water (mg/l)
Ka = adjusted overall mass transfer coefficient (cm/hr)
ts = shower droplet drop time (2 sec)
d = diameter of shower droplet (1 mm)
1/60 = conversion factor (hr/3600 sec + 10 mm/cm) with specific interfacial area of 6/d

5. VOC generation rate, termed "S"

S = Cwd FR/SV

where: S = VOC generation rate (mg/m^3-min)
Cwd = VOC conc. emitted from shower droplet (mg/l)
FR = shower water flow rate (10 l/min)
SV = shower room air volume (6 m^3)

6. Single Dose (mg/kg/shower) =

$$\frac{(Ir)\ (S)}{(BW)\ (Ar)} \left[Ds + \frac{(\exp\ (-Ar\ \ x\ \ Dt))}{Ar} - \frac{\exp\ (Ar\ (Ds\ -\ Dt))}{Ar} \right]$$

where: Ir = inhalation rate (0.01 m^3/min [0.6m^3/hr])
S = VOC generation rate (mg/m^{-3} -min)
BW = body weight (70 kg)
Ar = air exchange rate for "tight homes" (2.5 x 10^{-2} min^{-1})
Ds = shower duration (12 min)
Dt = total duration in the bathroom (20 min)

7. Chronic Dose = Single Dose x EF x ED x (AT)$^{-1}$

where: EF = exposure frequency (350 days/year)
ED = exposure duration (30 years)
AT = averaging time (25,550 days/carcinogenic risk)
(10,950 days/noncarcinogenic risk)

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Anson Environmental

Environmental Audits
Hazardous Waste
Asbestos Management
Groundwater Remediation
Storage Tank Management
Impact Statements
Wetland Investigations

256 Main Street
Northport, NY 11768
516-757-7090
(fax) 516-757-1229

January 28, 1992

Dorothy Allen, Project Manager
United States Environmental Protection Agency, Region II
26 Federal Plaza
New York, NY 10278

Re: Administrative Order Index No. II
CERCLA-90208
Anchor Chemical Site
500 West John Street
Hicksville, NY

Dear Mrs. Allen:

During our January 15, 1992 meeting in your offices, we agreed to provide written documentation demonstrating that field conditions require the modification of the Project Operations Plan (POP) so that the installation of deep wells would be to a depth of 110 to 120 feet rather than to the Magothy aquifer. The screens for these wells will be set at this depth. In addition, we requested authorization to gravity feed the sand/gravel pack and bentonite pellets.

The following information is organized in the order specified in James Doyle's, Esq. letter to Richard Leland, Esq. dated January 21, 1992.

a. Marine Pollution Control (MPC) has been performing environmental projects for over twenty-five years. They have been performing spill remediation and ground water monitoring well installation projects for NYS Department of Environmental Conservation for the past twelve years. For one project, MPC installed more than 155 ground water monitoring wells to depths in excess of 130 feet. The purpose of these wells was to define the Northville gasoline plume in Setauket, Long Island.

The soil in Setauket consisted of clays, silts and significant gravel layers. The wells at this site were only placed ten feet into ground water. Because of the soil conditions, 20 wells were "lost" because of the binding of the soils and "heaving sands". These 20 wells were in addition to the 155 wells that were successfully installed. Where the wells were successfully installed there was less "heaving sands" and the depth of the saturated zone was only ten feet compared to the Anchor Chemical site where this zone is approximately sixty feet thick.

MPC also drilled one 140 foot well in Jamaica, Queens. The depth to water in this well was 10 feet below land surface, therefore, 130 feet of drilling took place in the saturated zone. The soils at the Jamaica site were medium to coarse sands with no silts present. This differed significantly from the Anchor Chemical site as described in Item b. In addition, there were no "heaving sands" at Jamaica.

The boring logs for the above described wells, along with, nine other logs for deep wells are enclosed. The information in these boring logs is summarized in the attached table.

All of these wells were installed using either the CT-350 or CME 75 drill rig. Both of these rigs have been used to drill wells on the Anchor Chemical site.

b. The soil conditions at the Anchor Chemical site are described in the attached boring log prepared by BB&L for MW-1D. To summarize, fine sand and some silt were interspersed with coarser sands and gravel. Ground water was encountered at 59.75 feet below land surface. In addition, "heaving sands" were present (up to three feet thick) and required manual removal from inside the augers using a sand bailer prior to split spoon sample collection.

"Heaving sands" are loosely compacted saturated fine sands that rise into the interior of hollow stem augers during drilling. Hydrostatic or other pressures present in the geological formation cause water and loose sediments to rise inside the augers.

These soil conditions, that is, the amount of sand and silt and the thickness of the saturated zone, make reaching depths in excess of 120 feet difficult because of the "binding effects" created by the silts, fine sand and ground water. Sand bailing of "heaving sands", in some instances, took more than one hour to accomplish. During this time the augers were idle and binding by the soils occurred. At these depths and in these geological conditions, time is of the essence in successfully installing ground water monitoring wells.

c. The names of representatives of drilling companies contacted to determine whether the "F-10" or similar rig is capable of drilling deeper than 122 feet in the type of soils present at the Anchor Chemical site is enclosed. Five of the six drillers contacted committed to depths of 80 to 100 feet in the type of geological condition at Anchor Chemical site. The sixth driller, Empire Soil Investigation, who owns six F-10 WT rigs, is willing to commit to only 120 feet with 6.25 inches I.D. augers.

d. The boring log for MW-1D is attached. Mr. James Schafer, of BB&L, prepared this boring log. The names, training and experience of individuals who examined the soil sample from 122 feet are attached along with the memorandum from Mr. James DeMartinis, of BB&L, describing the soil sample collected from 122 feet.

It is Mr. DeMartinis' opinion that this soil between the surface and 120 feet are not representative of the Magothy aquifer because their sedimentological and mineralogical characteristics are not typical of the Magothy aquifer. The minerals in the sample at 122 feet were too immature to be of a geological age representative of the Magothy.

e. There are two acceptable methods for introducing the filter pack-tremie and gravity feed. We are requesting approval of gravity feed as the method of filter packing the wells at Anchor Chemical site.

The concern with gravity feed is segregation of the gravel which can take place during installation of this filter pack. This results in a well that consistently produces sediment-laden water samples. However, based on Handbook of Suggested Practices for the Design and Installation of Ground-Water Monitoring Wells, page 210, "segregation may not be a problem when placing truly uniform filter pack where the uniformity coefficient is less than 2.5, but placement by free fall is not recommended in any other situations" (Driscoll, 1986).

At Anchor Chemical, the issue of sediment-laden water samples will be addressed during well installation by use of a filter pack material with a uniformity coefficient of less than 2.5. This will be accomplished by certified testing written documentation from the filter pack supplier.

Secondly, the wells will be developed until the development water has a turbidity of less than 50 NTUs as specified in the POP.

Empire Soils Investigation of Highland Park, New Jersey and Environmental Drilling Inc. of West Creek, New Jersey both stated conditions on the Anchor Chemical site, e.g. the thickness of the saturated zone, the soil conditions and "heaving sands", would not allow them to use the tremie method to install gravel pack and bentonite pellets because the annulus between the augers and casing is too small for any tremie pipe to be used at depths in excess of 80 feet.

The gravity feed technique has been used for installing the filter pack and bentonite at several NPL sites on Long Island. The depth of the water columns on these sites have resembled that at Anchor Chemical.

These sites are:

- -Goldisc Recording
- -Bethpage State Park
- -Tronic Plating
- -Islip Municipal Sanitary Landfil

Roux Associates, under the direction of Stanley Sucharski, installed wells at Goldisc Recordings in Holbrook, Long Island. Some of these wells had water columns that resembled the column at Anchor Chemical site. The filter packs for these wells were installed using gravity feed.

At Bethpage State Park, Geraghty & Miller installed wells using gravity feed. These wells were approximately 190 feet deep. The water column was approximately 80 feet thick.

The shallow and deep wells at the Tronic Plating site in Farmingdale, Long Island had the filter pack gravity fed. The water column in the deep wells was approximately 100 feet thick.

Islip Municipal Sanitary Landfill has approximately 30 wells installed which ranged from 130 to 500 feet deep. All filter packs were gravity fed.

Bachar El Sabeh, while employed by Delta Well Drilling, Inc., coordinated the installation of the wells at the Bethpage, Tronic Plating and Islip sites. Mr. El Sabeh is currently employed by BB&L and his resume is enclosed.

Also enclosed are copies of validated and unvalidated laboratory data from soil samples collected on site. The laboratory analysis of samples collected from the four indoor borings do not indicate the presence of elevated levels of volatile organic compounds. (The laboratory analysis is not completed for the other two indoor borings.) The same is true of the soil samples collected from drywells 1, 6 and 7 where borings were installed to ground water. In fact, sediment samples from drywells #2, 3, 4, 6 and 8 contained one volatile organic compound and that was toluene. Other compounds present were ethylbenzene and xylenes in drywell #2 only. These are some of the constituents of gasoline which would be expected in a drywell that drained a parking lot. The presence of 1,1,1-trichloroethane and 1,1-dichloroethane were also noted in drywell #2.

We trust that the enclosed information is sufficient to address the concerns of your January 21, 1992 letter and for you to approve our request to set the screens for deep wells at 110 to 120 feet below land surface and to allow gravity feeding of the gravel pack and bentonite.

We look forward to a favorable response to our requests.

Very truly yours,

Stanley Sucharski

Dean Anson II
Co-Facility Coordinator

Stanley Sucharski
Co-Facility Coordinator

cc: James Doyle, Esq.,
James F. O'Brien, Esq.,
Richard G. Leland, Esq.
Mr. Arthur D. Sanders

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Boring Logs for MW-1D

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    Telephone Conversation Logs

Validated Laboratory Data

Unvalidated Laboratory Data

Marine Pollution Control

Well Drilling Experience

Chart and Boring Logs

MARINE POLLUTION CONTROL
WELL CONSTRUCTION EXPERIENCE

Date	Location	Driller	Rig	Depth of Well	Depth To Water	Amount of * Saturated Zone
4/6/89	Heinz Landfill Port Jefferson	D. Klaus	CT 350	160'	150'	10
3/24/89	Heinz Landfill Port Jefferson	D. Klaus	CT 350	157'	150'	7
3/28/89	Heinz Landfill Port Jefferson	D. Klaus	CT 350	154'	150	4
3/23/89	Heinz Landfill Port Jefferson	D. Klaus	CT 350	154'	150'	10
12/12/89	180 N. Country Rd., Mt. Sinai	J. Meaney	CT 350	145'	135'	10
2/28	Setauket	Joe K.	CME 75	145'	125'	20
5/90	UPS - Jamaica	Klaus	CT 350	140'	10'	130
4/27/90	Riverhead Terminal	D. Klaus	CT 350	130'	115'	15
	Setauket	Joe K.	CME 75	130'	110'	20
3/22	Setauket	Joe K.	CME 75	130'	110'	20
1/10/90	Port Jefferson Power Station	Don Klaus	CT 350	111'	100'	11
1/11/90	Port Jefferson Power Station	Don Klaus	CT 350	110'	100'	10
	Setauket	Joe K.	CME 75	110'	100'	10
11/29/89	Texaco 160 Manetto Hill Rd., Plainview	Don Klaus	CT 350	107'	97'	10
1/8/90	Port Jefferson Power Station	Don Klaus	CT 350	103'	90'	13
11/30/89	Brookhaven Comb. Turb. Fac. Shoreham	D. Klaus	CT 350	100'	90'	10
11/30/89	Brookhaven Comb. Turb. Fac. Shoreham	D. Klaus	CT 350	100'	90'	10
11/30/89	Brookhaven Comb. Turb. Fac. Shoreham	D. Klaus	CT 350	100'	90'	10
11/30/89	Brookhaven Comb. Turb. Fac. Shoreham	D. Klaus	CT 350	100'	90'	10

* Amount of Saturated Zone is amount of drilling in groundwater

CONTRACTORS PERFORMANCE DATA SHEET

PROJECT: Heinz Landfill
LOCATION: port Jefferson, N.Y.
CLIENT: gibbs and hill

CONTRACTOR: Marine Pollution Control
NYSDEC ID NO. Site I.D. #152055

WORK PERFORMED AND ACCEPTED

PERIOD: 3·22 TO 3·24 1989

ITEM NUMBER	DESCRIPTION	UNIT	UNIT PRICE	MONDAY	TUESDAY	WED	THRU	FRIDAY	TOT. THIS WEEK	TOT. TO DATE
	MOB. AND DEMOBILIZATION	LS				/ ct350 | cmc	ct350 | cmc	ct350 | cmc	.	
	AUGER DRILLING	FT				80' | 55'	65' | 60'	45 | 0	305	
	STEAM CLEANING	Hr				1 | 1	1 | 1	1 | 0	5 hrs.	
	SPLIT SPOON SAMPLES	Ea				10 | 12	13 | 12	6 | 0	59	
	PERMEABILITY TESTS	Hr								
	PVC WELL/RISER PIPE INSTALLED	Ft					154'	157'	311	
	WELL DEVELOPMENT	Hr								
	PROTECTIVE CASING/LOCKING CAP	LS								
	STANDBY TIME	Hr								

CONTRACTOR PERSONNEL	M	T	W	T	F
DRILLER Jon Klaus - Ct350			✓	✓	✓
HELPER Joe Kaplan - CMC			✓	✓	

COMMENTS 2 Rigs on Site.

INSPECTORS NAME ____________

CONTRACTORS PERFORMANCE DATA SHEET

PROJECT: Heins landfill
LOCATION: port Jefferson, N.Y.
CLIENT: gibbs and hill

CONTRACTOR: Marine Pollution Control
NYSDEC ID NO. Site I.D. # 152095

ITEM NUMBER	DESCRIPTION	UNIT	UNIT PRICE	WORK PERFORMED AND ACCEPTED PERIOD: 3-27 TO 3-29 1989 27 MONDAY		28 TUESDAY		29 WED		THRU	FRIDAY	TOT. THIS WEEK	TOT. TO DATE
	MOB. AND DEMOBILIZATION	LS		ct350	cmc	ct350	cmc	ct350	cmc				
	AUGER DRILLING	FT		77	0	70	0	110	0			257'	
	STEAM CLEANING	Hr		1	1	1	1	1	2?			7 hrs	
	SPLIT SPOON SAMPLES	Ea		17	0	13	0	21	0			51	
	PERMEABILITY TESTS	Hr											
	PVC WELL/RISER PIPE INSTALLED	Ft				154'						154	
	WELL DEVELOPMENT	Hr											
	PROTECTIVE CASING/LOCKING CAP	LS		1		1		1				3	
	STANDBY TIME	Hr											

CONTRACTOR PERSONNEL	M	T	W	T	F
DRILLER					
HELPER					

COMMENTS

INSPECTORS NAME

CONTRACTORS PERFORMANCE DATA SHEET

PROJECT: Heins landfill 07705
LOCATION: Port Jefferson N.Y.
CLIENT: Grossman Gull

CONTRACTOR: Marine Pollution Control
NYSDEC ID NO. Site I.D.# 152095

ITEM NUMBER	DESCRIPTION	UNIT	UNIT PRICE	WORK PERFORMED AND ACCEPTED PERIOD: 4.5 TO 4.7 1989 MONDAY	TUESDAY	5 WED	6 THRU	7 FRIDAY	TOT. THIS WEEK	TOT. TO DATE Total for Job!!!
	MOB. AND DEMOBILIZATION	LS				ob36 | [illegible]		ob30 | [illegible]		
	AUGER DRILLING	FT				55 | 0		|	55	617
	STEAM CLEANING	Hr				1 | 0		0 | 1	1 hrs	13 hrs
	SPLIT SPOON SAMPLES	Ea				9 | 0		|	9	119
	PERMEABILITY TESTS	Hr								
	PVC WELL/RISER PIPE INSTALLED	Ft					160		160	625'
	WELL DEVELOPMENT	Hr								
	PROTECTIVE CASING/LOCKING CAP	LS						1	1	4
	STANDBY TIME	Hr								

CONTRACTOR PERSONNEL	M	T	W	T	F
DRILLER					
HELPER					

COMMENTS

INSPECTORS NAME

CT 350

DRILLING / WELL LOG

Boring/Well #1 CLIENT N.Y.S.D.E.C Page 1 of 4

Location [illegible] N. COUNTRY RD., MT. SINAI Date [illegible]

Total Depth Drilled 145' Type of Sample AUGER Interval 5'

Casing Installed 122' PVC Screen Installed 20' [illegible] 0.020"

Drilling Method H.S.A Drilling Fluid Used —

Drilling Contractor MPC Driller J. [illegible]

Prepared By GEORGE VASSILEV

Sample Depth From	To	OVA Reading (ppm)	Sample Description
0	5'		FINE-MED., REDDISH/BROWN, SILTY SAND,
			POORLY SORTED, PROBABLY FILL MATERIAL.
5'	10'		FINE-MED., REDDISH/BROWN, SILTY SAND,
			POORLY SORTED, PROBABLY FILL MATERIAL.
10'	15'		FINE-MED., LT. BROWN/REDDISH, SILTY SAND,
			MOIST, 10-15% MED-COARSE GRAVEL, POORLY
			SORTED, HYDROCARBON ODORS.
15'	20'		FINE-MED., LT. BROWN/REDDISH, SILTY SAND,
			MOIST, 10-15% MED.-COARSE GRAVEL, POORLY
			SORTED, ODORS.
20'	25'		FINE-MED., LT. BROWN/REDDISH, SILTY SAND,
			MOIST, 10-15% MED.-COARSE GRAVEL,
			POORLY SORTED, ODORS.
25'	30'		GRAYISH (STAINED?), SANDY SILT, BALLS
			UP OFF AUGERS, STRONG ODORS.
30'	35'		GRAYISH, SANDY SILT, 15-20% COARSE
			GRAVEL, SLIGHT ODORS, COBBLES, POORLY SORTED.
35'	40'		FINE-MED., GRAYISH, SILTY SAND, 15-20%
			COARSE GRAVEL, COBBLES, POORLY SORTED ODORS.

DRILLING / WELL LOG

Boring/Well #1 CLIENT N.Y.S.D.E.C. Page 2 of 4

Location 180 N. COUNTRY RD., MT. SINAI Date 12/12/89

Total Depth Drilled 145' Type of Sample AUGER Interval 5'

Casing Installed 122' PVC Screen Installed 20' PVC 0.020"

Drilling Method H.S.A Drilling Fluid Used ——

Drilling Contractor MPC Driller J. MEANEY

Prepared By GEORGE VASSILEV

Sample From	Depth To	OVA Reading (ppm)	Sample Description
40'	45'		FINE-MED, GRAYISH, SILTY SAND, 15-20% COARSE-V. COARSE GRAVEL, COBBLES, POORLY SORTED, ODORS.
45'	50'		FINE-MED., GRAYISH, SILTY SAND, 15-20% COARSE-V. COARSE GRAVEL, COBBLES, POORLY SORTED, ODORS.
50'	55'		FINE-MED., GRAYISH, SILTY SAND, 15-20% COARSE-V. COARSE GRAVEL, COBBLES, POORLY SORTED, ODORS
55'	60'		FINE-MED., LT. BROWN/GRAY, SILTY SAND, 10-15% MED.-COARSE GRAVEL, POORLY SORTED, ODORS.
60'	65'		FINE-MED., LT. BROWN/REDDISH, SILTY SAND, 10-15% MED.-COARSE GRAVEL, POORLY SORTED, ODORS.
65'	70'		FINE-MED, LT. BROWN/REDDISH, SILTY SAND, 10-15% MED.-COARSE GRAVEL, POORLY SORTED, ODORS.
70'	75'		FINE-MED, LT. BROWN/REDDISH, SILTY SAND, 5-10% MED-COARSE GRAVEL, POORLY SORTED, ODORS.
75'	80'		FINE-MED, LT. BROWN/YELLOW SAND, < 5% GRAVEL, MODERATELY SORTED, ODORS(?).
80'	85'		FINE-MED., YELLOW/ORANGE SAND, 5-10% FINE GRAVEL, MODERATELY SORTED, ODORS(?).

DRILLING / WELL LOG

Boring/Well #1 CLIENT N.Y.S.D.E.C. Page 3 of 4

Location 180 N. COUNTRY RD., MT. SINAI Date 12/12/89

Total Depth Drilled 145' Type of Sample AUGER Interval 5'

Casing Installed 122' PVC Screen Installed 20' PVC 0.020'

Drilling Method H.S.A. Drilling Fluid Used ——

Drilling Contractor MPC Driller J. MEANEY

Prepared By GEORGE VASSILEV

Sample Depth From	Sample Depth To	OVA Reading (ppm)	Sample Description
85'	90'		FINE-MED, YELLOW/ORANGE SAND, 5-10% FINE GRAVEL, MODERATELY SORTED.
90'	95'		MEDIUM YELLOW/ORANGE SAND, 5-10% FINE-MEDIUM GRAVEL, TRACE SMALL PEBBLES, POORLY SORTED.
95'	100'		MEDIUM, YELLOW/ORANGE SAND, 5-10% FINE-MED. GRAVEL, TRACE SM. PEBBLES, POORLY SORTED.
100'	105'		MEDIUM, YELLOW/ORANGE SAND, 5-10% FINE-MED. GRAVEL, TRACE SM. PEBBLES, POORLY SORTED.
105'	110'		MEDIUM, YELLOW/ORANGE SAND, 5-10% FINE-MED. GRAVEL, TRACE SM. PEBBLES, POORLY SORTED.
110'	115'		MEDIUM, YELLOW/ORANGE SAND, 5-10% FINE-MED. GRAVEL, TRACE SM. PEBBLES, POORLY SORTED.
115'	120'		MEDIUM, YELLOW/ORANGE SAND, 5-10% FINE-MED GRAVEL, TRACE SM. PEBBLES, POORLY SORTED.
120'	125'		MEDIUM, YELLOW/ORANGE SAND, 5-10% FINE-MED. GRAVEL, TRACE SM. PEBBLES, POORLY SORTED.
125'	130'		MEDIUM YELLOW/ORANGE SAND, 5-10% FINE-MED GRAVEL, TRACE SM. PEBBLES, POORLY SORTED.
130'	135'		MEDIUM, YELLOW/ORANGE SAND, 5-10% FINE-MED

DRILLING / WELL LOG

Boring/Well #1 CLIENT N.Y.S.D.E.C. Page 4 of 4

Location 180 N. COUNTRY RD., MT SINAI Date 12/12/89

Total Depth Drilled 145' Type of Sample AUGER Interval 5'

Casing Installed 122' PVC Screen Installed 20' PVC 0.020"

Drilling Method H.S.A Drilling Fluid Used —

Drilling Contractor MPC Driller J. MEANEY

Prepared By GEORGE VASSILEV

Sample From	Depth To	OVA Reading (ppm)	Sample Description
			GRAVEL, TRACE SM. PEBBLES, POORLY SORTED
135'	140'		MEDIUM, YELLOW/ORANGE SAND, 5-10% FINE -MED.
			GRAVEL, TRACE SM. PEBBLES, POORLY SORTED.
140'	145'		MEDIUM, YELLOW/ORANGE SAND, 5-10% FINE -
			MED. GRAVEL, TRACE SM. PEBBLES, POORLY SORTED.
			WELL CONSTRUCTION
			142'-122' CAVE IN MATERIAL
			122'-120' BENTONITE PELLETS
			120'-2' CLEAN BACKFILL
			2' - 1' BENTONITE PELLETS
			1' - 0' CLEAN BACKFILL
			FINISHED TO GRADE WITH
			MANHOLE AND LOCKING CAP
			WITH LOCK.

TABLE 1

TRC WORK PLAN
UPS SITE JAMAICA, NEW YORK
MONITORING WELL PARAMETERS

Well ID	Depth (approx)	Work Required	
(2-inch diameter)			
MW-2	28'	Replace	
MW-3	28'	Replace	
MW-5	28'	Replace	
MW-6	28'	Replace	
MW-7	28'	Replace	
(4-inch diameter)			
MW-5I	60'	Replace	
MW-7I	60'	Replace	Installed May 90 w/CT350
MW-9M	140'	Replace	
MW-10	28'	Repair: Install protective cover and locking cap.	
MW-13	28'	Repair: Replace flush mount protective cover.	
MW-14D	100'	Repair: Replace locking well cap.	
MW-14I	60'	Replace	
MW-14M	140'	Replace	
MW-15	28'	Repair: Replace locking cap.	
MW-16	28'	Replace	
MW-17M	130'	Repair: Replace locking cap.	

In repairing the wells that require replacement of the protective cover, a new protective cover or curb box of appropriate size will be placed so as to be approximately flush with grade. If the well is located in a paved area, the disturbed area will be covered with cold patch according to manufacturer's directions. Replacement of asphalt or concrete paving is not within the scope of this work plan. Any well caps that are installed will be of the locking variety and will include a lock.

Included in the work activities under this task is a search for MW-9M, which was apparently buried during landscaping operations. Hand tools and/or a rubber tired backhoe will be used to explore the near surface soil in the area of MW-9S in an attempt to locate this well. If the well is found, steps will be taken as necessary to restore it to a useable condition.

3.0 MONITORING WELL INSTALLATION

TRC will implement the replacement of up to eleven monitoring wells on the site. The drilling method will be hollow stem auger. The approximate location of the monitoring wells is shown in Figure 1. The exact location of each well will be marked by TRC personnel prior to drilling. Details of well construction are shown schematically in Figure 2.

Each well shall have a 10 foot long screen section. The approximate depth of the wells shall be as follows:

a) shallow: MW-2, MW-3, MW-5, MW-6, MW-7, MW-16, MW-14S - screen setting 18-28 feet

b) intermediate: MW-5I, MW-7I, MW-14I - screen setting 50-60 feet

c) magothy: MW-9M, MW-14M - screen setting 130-140 feet

DTW
20'

SAMPLE/CORE LOG

Boring/Well 155 C Project/No. NIC Page 1 of 1

Site Location SETAUKET Drilling Started 2-25 Drilling Completed 3-1

Total Depth Drilled 145 feet Hole Diameter ______ inches Type of Sample/ Coring Device ______

Length and Diameter of Coring Device ______ Sampling Interval ______ feet

Land-Surface Elev. ______ feet ☐ Surveyed ☐ Estimated Datum ______

Drilling Fluid Used ______ Drilling Method ______

Drilling Contractor ______ Driller ______ Helper ______

Prepared By ______ Hammer Weight ______ Hammer Drop ______ inches

Sample/Core Depth (feet below land surface)		Core Recovery (feet)	Time/Hydraulic Pressure or Blows per 6 inches	Sample/Core Description
From	To			
0		10		CLAY SAND
10		45		SAND
45		90		CLAY
90		145		SAND CLAY

CT350

DRILLING / WELL LOG

Boring/Well #16 CLIENT NORTHVILLE INDUSTRIES Page 1 of 3
Location RIVERHEAD TERMINAL Date 4/27/90
Total Depth Drilled 130' Type of Sample AUGER Interval 5'
Casing Installed 110' Screen Installed 20'
Drilling Method H.S.A. Drilling Fluid Used NONE
Drilling Contractor MPC ENVIRONMENTAL SERV. Driller D. KLAUS
Prepared By GEORGE VASSILEV - HYDROGEOLOGIST

Sample From	Depth To	OVA Reading (ppm)	Sample Description
0'	5'		VERY FINE, BROWN SILTY SAND, 5-10% FINE GRAVEL, POORLY SORTED.
5'	10'		VERY FINE, LT. GRAY SANDY SILT, <5% FINE GRAVEL, POORLY SORTED.
10'	15'		VERY FINE, LT. GRAY SANDY SILT, <5% FINE GRAVEL, POORLY SORTED.
15'	20'		VERY FINE, LT. GRAY SANDY SILT, <5% FINE GRAVEL, POORLY SORTED.
20'	25'		VERY FINE, LT. BROWN SANDY SILT, DAMP, <5% FINE GRAVEL, POORLY SORTED.
25'	30'		VERY FINE, LT. BROWN SANDY SILT, DAMP, <5% FINE GRAVEL, POORLY SORTED.
30'	35'		VERY FINE, LT. BROWN SANDY SILT, DAMP, <5% FINE GRAVEL, POORLY SORTED.
35'	40'		SILT, BROWN, LOOSE, DAMP, <5% FINE GRAVEL, POORLY SORTED.
40'	45'		VERY FINE, LT. BROWN SILTY SAND, DAMP, <5% FINE GRAVEL, POORLY SORTED.
45'	50'		FINE-MEDIUM, TAN SAND, NO GRAVEL

DRILLING / WELL LOG

Boring/Well #16 CLIENT NORTHVILLE INDUSTRIES Page 2 of 3
Location RIVERHEAD TERMINAL Date 4/27/90
Total Depth Drilled 130' Type of Sample AUGER Interval 5'
Casing Installed 110' Screen Installed 20'
Drilling Method H.S.A. Drilling Fluid Used NONE
Drilling Contractor MPC ENVIRONMENTAL SERV. Driller D. KLAUS
Prepared By GEORGE VASSILEV - HYDROGEOLOGIST

Sample From	Depth To	OVA Reading (ppm)	Sample Description
			WELL SORTED.
50'	55'		FINE-MEDIUM, TAN SAND, NO GRAVEL, WELL SORTED.
55'	60'		FINE-MEDIUM, TAN SAND, NO GRAVEL, WELL SORTED.
60'	65'		FINE-MEDIUM, TAN SAND, NO GRAVEL WELL SORTED.
65'	70'		FINE-MEDIUM, TAN SAND, NO GRAVEL, WELL SORTED.
70'	75'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
75'	80'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
80'	85'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
85'	90'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
90'	95'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.

DRILLING / WELL LOG

Boring/Well #16 CLIENT NORTHVILLE INDUSTRIES Page 3 of 3

Location RIVERHEAD TERMINAL Date 4/27/90

Total Depth Drilled 130' Type of Sample AUGER Interval 5'

Casing Installed 110' Screen Installed 20'

Drilling Method H.S.A. Drilling Fluid Used NONE

Drilling Contractor MPC ENVIRONMENTAL SERV. Driller D. KLAUS

Prepared By GEORGE VASSILEV - HYDROGEOLOGIST

Sample From	Depth To	OVA Reading (ppm)	Sample Description
95'	100'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
100'	105'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
105'	110'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
110'	115'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
115'	120'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
120'	125'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
125'	130'		FINE-MEDIUM, TAN SAND, <5% FINE GRAVEL, POORLY SORTED.
			NO ODORS OR CONTAMINATION DETECTED DURING DRILLING
			TOTAL DEPTH ~130'
			DEPTH TO WATER ~ 115'

14" GAS IN WELL

SAMPLE/CORE LOG

Boring/Well 45 Project/No. North Well Page 1 of 1

Site Location Skiawkki Drilling Started [illegible] Drilling Completed ____

Total Depth Drilled 130 feet Hole Diameter ____ inches Type of Sample/ Coring Device ____

Length and Diameter of Coring Device ____ Sampling Interval ____ feet

Land-Surface Elev. ____ feet ☐ Surveyed ☐ Estimated Datum ____

Drilling Fluid Used ____ Drilling Method ____

Drilling Contractor ____ Driller Joe K Helper Dan K Ed C

Prepared By Joe K Hammer Weight ____ Hammer Drop ____ inches

Sample/Core Depth (feet below land surface) From	To	Core Recovery (feet)	Time/Hydraulic Pressure or Blows per 6 inches	Sample/Core Description
0		30'		SAND
30		50		SAND CLAY
50		85		CLAY SAND WET
85		130		SAND CLAY WET

SAMPLE/CORE LOG

Boring/Well 97 Project/No. NIC Page 1 of 1

Site Location STEWART Drilling Started 3/92 Drilling Completed

Total Depth Drilled 130 feet Hole Diameter inches Type of Sample/ Coring Device

Length and Diameter of Coring Device Sampling Interval feet

Land-Surface Elev. feet ☐ Surveyed ☐ Estimated Datum

Drilling Fluid Used Drilling Method

Drilling Contractor Driller Helper

Prepared By JOE K Hammer Weight Hammer Drop inches

12 BAG GRAVEL

GRAVEL TO 105

Sample/Core Depth (feet below land surface) From	To	Core Recovery (feet)	Time/Hydraulic Pressure or Blows per 6 inches	Sample/Core Description
0		40		SAND GRAVEL
40		100'		CLAY SAND WET
100		104		ROCK
104		130		SAND CLAY

CT 350

DRILLING / WELL LOG

Boring/Well #2 CLIENT LILCO Page 1 of 3

Location Port Jefferson Power Plant Date 1/10/90

Total Depth Drilled 111' Type of Sample S.S. Interval 5'

Casing Installed 91' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv. Driller Don Klaus

Prepared By George Vassilev - Hydrogeologist

Sample Depth From	Sample Depth To	OVA Reading (ppm)	Sample Description
0	5		Yel/or to brown fine-med sand, fine-med gravel, hand dug
5	7		upper 3" a white fine-med sand, fine gravel, USCS SP
			lower 7" a brown fine-med silty sand, fine gravel, USCS SM, BC 7,11,18,21
10	12		Brown fine-med silty sand, med gravel USCS SM, BC 33,24,26,27 18" recovery
15	17		upper 12" a yel/or to gray fine-med silty sand, fine gravel, USCS SM
			lower 6" a gray fine silty sand, USCS SM blow counts 10,22,25,22
20	22		upper 8" a dk. brown fine-med silty sand, USCS SM
			mid 4" a lt. brown fine silty sand, USCS SM
			lower 2" a white fine-med sand, USCS SP BC 16,22,22,25 refusal, move rig
25	27		upper 12" a brown fine-med silty sand, fine gravel, USCS SM
			lower 4" a brown to gray fine silty sand, fine gravel, compacted, USCS SM,
			blow counts 11,13,12,12
30	32		upper 8" a brown fine-med silty sand, fine gravel, USCS SM
			lower 2" a crushed granite cobble blow counts 13,14,12,13
35	37		upper 1" a crushed granite cobble lower 11" a lt br/yel fine-med sand,
			USCS SP, BC 10,12,12,14
40	42		upper 4" a br. fine-med silty sand, fine gravel

DRILLING / WELL LOG

Boring/Well #2 CLIENT LILCO Page 2 of 3

Location Port Jefferson Power Station Date 1/10/90

Total Depth Drilled 111' Type of Sample S.S. Interval 5'

Casing Installed 91' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv. Driller Don Klaus

Prepared By George Vassilev - Hydrogeologist

Sample Depth From	To	OVA Reading (ppm)	Sample Description
40	42		lower 8" a yel/or fine-med sand, fine gravel,USCS SP, BC 10,7,11,14
45	47		Yel/or to white fine-med sand, fine gravel,USCS SP, BC 11,14,16,17 18" rec
50	52		upper 10" a yel/or fine-med sand,fine gravel, USCS SP
			mid 7" a white coarse sand,fine-med gravel, USCS SP
			lower 3" a red/br v fine-fine sand, fine gravel,USCS SM, BC 10,9,9,15
55	57		White fine-med sand,fine-med gravel, USCS SP, BC 9,10,13,17 10" recovery
60	62		White fine-med sand, fine-med gravel, USCS SP, BC 16,17,19,18 16" recovery
65	67		upper 8" a br. fine-med sand,med-coarse gravel, USCS SP
			mid 3" a yel/or fine-med sand,USCS SW lower 5" a yel/wh coarse-v coarse sand,
			med gravel,USCS SP, BC 27,19,26,30
70	72		upper 8" a br. fine-med silty sand,fine gravel, USCS SP
			lower 4" a white fine-med sand,USCS SM blow counts 20,13,15,16
75	77		upper 8" a br to yel/or fine-med silty sand,compacted,fine gravel,USCS SM
			lower 2" a crushed quartz cobble, blow counts 18,15,19,20
80	82		upper 8" a br fine-med sand,fine gravel USCS SP
			lower 4" a yel/or v fine-fine sand, USCS SM, blow counts 12,13,11,12
85	87		Yel/or fine-med sand,fine gravel,USCS SP, blow counts 9,11,16,17

DRILLING / WELL LOG

Boring/Well #2 CLIENT LILCO Page 3 of 3

Location Port Jefferson Power Plant Date 1/10/90

Total Depth Drilled 111' Type of Sample S.S. Interval 5'

Casing Installed 91' Screen Installed 20'

Drilling Method H.S.A. Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv. Driller Don Klaus

Prepared By George Vassilev - Hydrogeologist

Sample Depth From	To	OVA Reading (ppm)	Sample Description
90	92		Yel/or to white fine-med sand,fine gravel,USCS SP, BC 9,11,10,15 13" rec.
95	97		Yel/wh fine-med sand,fine-med gravel, USCS SP, BC 8,8,10,12 10" recovery
100	102		Brown fine-med sand,fine-med gravel,wet USCS SP, BC 8,9,8,14 10" recovery
110	115		Dk. brown clay on last 5' of augers
			Well Construction
			DTW approx. 100' TD of well 111'
			20' of 0.020" slotted screen 91' of PVC SCH 40 pipe
			111' to 87' #2 morie sand pack
			87' to 85' bentonite pellets 85' to 4' clean backfill
			4' to 3' bentonite pellets Finished to grade with manhole

CT 350

DRILLING / WELL LOG

Boring/Well #3 CLIENT LILCO Page 1 of 3

Location Port Jefferson Power Station Date 1/11/90

Total Depth Drilled 110' Type of Sample S.S. Interval 5'

Casing Installed 90' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv. Driller Don Klaus

Prepared By George Vassilev - Hydrogeologist

Sample Depth From	To	OVA Reading (ppm)	Sample Description
0	5		Brown fine silty sand, fine-med gravel, hand dug
5	7		Yel/or fine-med sand, fine gravel, USCS SP, BC 6,8,8,6 14" recovery
10	12		Brown v fine-fine silty sand, USCS SM, blow counts 7,10,10,12 22" recovery
15	17		upper 8" a br fine-med silty sand, USCS SM,
			lower 10" a lt br med-coarse sand, med gravel, USCS SP, BC 7,10,19,15
20	22		Tan/white med sand, fine-med gravel, USCS SP, BC 9,8,8,10 16" recovery
25	27		upper 10" a tan/wh fine sand, USCS SP, lower 2" a brown compacted silt, damp,
			USCS SM, BC 7,7,7,8
30	32		upper 12" a brown damp silt, USCS ML lower 12" a yel/or fine-med sand, fine
			gravel, USCS SP, BC 7,6,7,7
35	37		upper 4" a br. fine-med silty sand, damp USCS SM
			lower 11" a yel/or fine-med sand, USCS SP, blow counts 7,7,9,11
40	42		Yel/or fine-med sand, fine-med gravel, USCS SP, BC 5,6,9,11 18" recovery
45	47		upper 5" a yel/or fine-med sand, USCS SP lower 4" a yel/wh coarse-v coarse sand,
			med-coarse gravel, USCS SP, blow counts 5,4,9,21
50	52		upper 6" a br fine silty sand, USCS SM lower 6" a yel/or fine sand, laminated,
			USCS SP, BC 8,9,10,21

DRILLING / WELL LOG

Boring/Well #3 CLIENT LILCO Page 2 of 3

Location Port Jefferson Power Station Date 1/11/90

Total Depth Drilled 110' Type of Sample S.S. Interval 5'

Casing Installed 90' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv. Driller Don Klaus

Prepared By George Vassilev - Hydrogeologist

Sample Depth From	To	OVA Reading (ppm)	Sample Description
55	57		Yel/or fine sand, fine gravel, USCS SP, blow counts 7,10,9,25 10" recovery
60	62		upper 2" a yel/or fine-med sand, fine gravel, USCS SP
			lower 10" a yel/or coarse-v coarse sand med-coarse gravel, USCS SP, blow counts
			14,12,14,20
65	67		upper 3" a yel/or coarse-v coarse sand, med-coarse gravel, USCS SP
			lower 9" a white fine sand, fine gravel, USCS SP, BC 15,26,23,25
70	72		First sample, no recovery, BC 9,16,24,18 White fine sand, fine gravel, USCS SP
			blow counts 20,22,23,24 6" recovery
75	77		Yel/wh fine-med sand, fine gravel, USCS SP, BC 11,8,10,15 12" recovery
80	82		Yel/wh fine-med sand, fine gravel, USCS SP, BC 6,8,10,11 14" recovery
85	87		Yel/or fine sand, laminated, USCS SP, blow counts 6,7,10,12 13" recovery
90	92		Yel/wh med sand, damp, fine gravel, USCS SP, BC 6,7,9,11 10" recovery
95	97		Yel/wh fine-med sand, fine-med gravel, USCS SP, BC 4,5,5,8 12" recovery
100	102		upper 4" a br med sand, fine-med gravel, wet, USCS SP
			lower 10"a br coarse-v coarse sand, med-coarse gravel, wet, USCS SP, blow counts
			7,11,5,7

DRILLING / WELL LOG

Boring/Well #3 CLIENT LILCO Page 3 of 3

Location Port Jefferson Power Station Date 1/11/90

Total Depth Drilled 110' Type of Sample S.S. Interval 5'

Casing Installed 90' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv. Driller Don Klaus

Prepared By George Vassilev - Hydrogeologist

Sample Depth		OVA Reading	
From	To	(ppm)	Sample Description
			Well Consrtuction
			DTW approx. 100' TD of well 110'
			20' of 0.020" slotted screen 90' of PVC SCH 40 pipe
			110' to 87' #2 morie sand pack
			87' to 85' bentonite pellets 85' to 4' clean backfill
			4' to 3' bentonite pellets Finished to grade with manhole

CME 75

SAMPLE/CORE LOG

Boring/Well 1 Project/No. NORTHVILLE Page 1 of 4

Site Location SETAUKET Drilling Started 0800 Drilling Completed ______

Total Depth Drilled 110 feet Hole Diameter ______ inches Type of Sample/ Coring Device ______

Length and Diameter of Coring Device ______ Sampling Interval ______ feet

Land-Surface Elev. ______ feet ☐ Surveyed ☐ Estimated Datum ______

Drilling Fluid Used ______ Drilling Method ______

Drilling Contractor ______ Driller Jack K Helper TED / JIMM

Prepared By Jack K Hammer Weight ______ Hammer Drop ______ inches

Sample/Core Depth (feet below land surface) From	To	Core Recovery (feet)	Time/Hydraulic Pressure or Blows per 6 inches	Sample/Core Description
0		5		SAND
5		10		CLAY SAND
10		12		GRAVEL
12		15		SAND GRAVEL
15		35		SAND
35		37		GRAVEL
37		100'		SAND CLEAN
101		110'		SAND GAS ODOR

CT 350

DRILLING / WELL LOG

Boring/Well #1 CLIENT G.I. Page 1 of 2

Location Texaco 160 Manetto Hill Rd. Plainview Date 11/29/89

Total Depth Drilled 107' Type of Sample Inferred from auger cuttings Interval

Casing Installed PVC SCH 40 87' Solid Screen Installed 20' 0.020 slot (Monofle.

Drilling Method H.S.A. Drilling Fluid Used —

Drilling Contractor MPC Driller Don Klaus

Prepared By George Vassilev

Sample From	Depth To	OVA Reading (ppm)	Sample Description
0'	5'	—	Fine-Med, brown silty sand, 15-20% coarse gravel, some small cobbles, Poorly sorted, possible fill material.
5'	10'	—	Med.-Coarse, yellow/orange sand, 20-25% coarse gravel, Poorly sorted, possible fill material
10'	15'	—	V. Fine-Fine, buff/tan to Pinkish sand, <5% fine gravel, Moderately sorted.
15'	20'	—	Mostly Fine-Med., lt. brown to yellow/orange sand, <5% fine gravel, Moderately sorted.
20'	25'	—	Same, with hydrocarbon odors.
25'	30'	—	V. Fine-Fine, yellow/orange sand, trace fine gravel, Moderate to Well sorted, odors.
30'	35'	—	Same
35'	40'	—	Fine, yellow/orange sand, trace fine gravel, well sorted, odors.
40'	45'	—	Fine, silty, light brown sand, moderately sorted, odors
45'	50'	—	Same

DRILLING / WELL LOG

Boring/Well #1 CLIENT G.I. Page 2 of 2

Location Texaco, 160 Manetto Hill Rd., Plainview Date 11/29/89

Total Depth Drilled 107' Type of Sample ______ Interval ____

Casing Installed PVC, SCH 40 87' Solid Screen Installed 20' 0.020 slot

Drilling Method H.S.A Drilling Fluid Used —

Drilling Contractor MPC Driller D. Klaus

Prepared By George Vassilev

Sample Depth From	Sample Depth To	OVA Reading (ppm)	Sample Description
50'	55'	—	Fine, lt. brown sand, some silt,
			Moderately sorted, strong odors.
55'	60'	—	Mostly Fine to Med, reddish to light
			brown sand, trace of small gray clumps
			of silt, Moderately sorted, strong odors.
60'	65'	—	Same, light Brown sand.
65'	70'	—	Same
70'	75'	—	Same, tan to light Brown sand.
75'	80'	—	Same
80'	85'	—	Same
85'	90'	—	Same
90'	95'	—	Silty, Fine, tan/lt. brown sand, trace
			of small gray clumps of silt, Moderately
			sorted, damp, odors
95'	100'	—	Same
100'	105'	—	Silty brown sand wet, light odors.
105'	110'	—	Same

[illegible]

DRILLING / WELL LOG

Boring/Well #1 CLIENT LILCO Page 1 of 2

Location Port Jefferson Power Station Date 1/8/90

Total Depth Drilled 103' Type of Sample S.S. Interval 5'

Casing Installed 83' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv. Driller Don Klaus

Prepared By George Vassilev - Hydrogeologist

Sample Depth From	Sample Depth To	OVA Reading (ppm)	Sample Description
0	5		Brown sandy loam, med. gravel, hand dug
5	7		Yel./orange, fine-med sand, med gravel, USCS SP blow counts 37 20"recovery
10	12		upper 7" a white, fine-med sand, USCS SP mid 2" a brown silt, USCS OL lower 3" a yel/or, med-coarse sand, med gravel USCS SW, blow counts 6,9,10,25
15	17		upper 2" a yel/or, fine-med sand, med gravel, USCS SP mid 3" a white, fine-med sand, USCS SP lower 6" a yel/wh, med-coarse sand, USCS SP, blow counts 12,9,4,8
20	22		upper 7" a lt. br., fine sand ,USCS SM lower 13" a yel/red, fine-med sand, med gravel, USCS SP, blow counts 10,11,12,11
25	27		Brown sand, fine-med gravel, USCS SP blow counts 11,51,17,20 4" recovery
30	32		Lt. br., fine-med sand, med gravel, USCS SP, blow counts 14,13,22,21, 14" recov.
35	37		up 4" a br. fine-med sand, fine gravel, USCS SP lower 12" a white fine-med sand, fine gravel, USCS SP BC 12,19,26,27
40	42		Tan/wh med sand, fine-med gravel, USCS SP blow counts 5,15,30,28 12" recovery
45	47		White fine-med sand, fine-med gravel, USCS SP, BC 10,14,23,32, 12" recovery
50	52		White fine sand, USCS SM, blow counts 37,24,31,39 12" recovery
55	57		Yel/wh coarse-v coarse sand, fine-med gravel, BC 12,13,20,30 15" recovery

DRILLING / WELL LOG

Boring/Well #1 Client LILCO Page 2 of 2

Location Port Jefferson Power Station Date 1/8/90

Total Depth Drilled 103' Type of Sample S.S. Interval 5'

Casing Installed 83' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv. Driller Don Klaus

Prepared By George Vassilev - Hydrogeologist

Sample Depth From	To	OVA Reading (ppm)	Sample Description
60	62		White fine-med sand, fine gravel, USCS SP blow counts 10,25,26,26 15" recovery
65	67		upper 6" a white coarse-v coarse sand, med-coarse gravel, USCS SP
			lower 7" a white med-coarse sand, med gravel, USCS SP, BC 9,24,25,24
70	72		White fine-med sand, fine-med gravel, USCS SP, BC 14,17,28,17 17" recovery
75	77		upper 8" a yel/or fine-med sand, fine gravel, USCS SP
			lower 6" a yel/or med- coarse sand, med gravel, USCS SP, BC 10,10,11,25
80	82		upper 7" a yel/or med sand, med gravel, USCS SP
			lower 6" a white fine-med sand, fine gravel, USCS SP, BC 14,12,15,24
85	87		Buff/white fine sand, USCS SM, blow counts 7,14,16,26 12" recovery
90	92		Brown med-coarse sand, fine gravel, wet USCS SP, BC 13,13,13,23 12" recovery
100	105		Gray clay on last 5' of augers, USCS CH
			Well Construction
			DTW approx. 90' TD of well 103'
			20' of 0.020" slotted screen 83' of solid PVC SCH 40 pipe
			103' - 80' #2 morie sand pack
			80' - 78' bentonite pellets 78' - 4' clean backfill
			4' - 3' bentonite pellets Finished with stand-up casing

CT 356

DRILLING / WELL LOG

Boring/Well 1 CLIENT LILCO Page 1 of 2

Location Brookhaven Comb. Turb. Fac. Shoreman Date 11/30/89

Total Depth Drilled 100' Type of Sample S.S Interval 2'

Casing Installed 80' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv Driller D.Klaus

Prepared By Charles Sosik - Geologist

Sample Depth From	To	OVA Reading (ppm)	Sample Description
0	1/2'		bluestone base
1/2	2		topsoil, med gravel
2	5		tan med sand, fine gravel USCS sp
5	7		dk tan fine-coarse sand, fine gravel USCS sp 30HB
10	12		tan fine-coarse sand, fine gravel USCS sp 38HB
15	17		tan med-coarse sand, med gravel USCS sp 50HB
20	22		tan med sand, fine gravel, USCS sp 65HB
25	27		tan med sand, fine gravel, USCS sp 55HB
30	32		lt tan med-coarse sand, med gravel USCS sp 47HB
35	37		lt tan med-coarse sand, med gravel USCS sp 42HB
40	42		tan med sand, fine gravel USCS sp 32HB
45	47		lt tan med-coarse sand, fine gravel USCS sp 37HB
50	52		lt tan med-coarse sand, fine gravel USCS sp 39HB
55	57		lt tan fine sand USCS sm 39HB
60	62		lt tan fine-med sand USCS sp-sm 34HB
65	67		lt tan fine sand USCS sm 32HB

DRILLING / WELL LOG

Boring/Well 1 CLIENT LILCO Page 2 of 2

Location Brookhaven Comb. Turb. Fac. Shoreman Date 11/30/89

Total Depth Drilled 100' Type of Sample S.S Interval 2'

Casing Installed 80' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv Driller D.Klaus

Prepared By Charles Sosik - Geologist

Sample Depth From	To	OVA Reading (ppm)	Sample Description
70	72		lt tan fine sand USCS sm 30HB
75	77		lt tan med-fine sand USCS sp-sm 29HB
80	82		lt tan med-coarse sand,fine gravel USCS sp 27HB
85-	87		tan med-coarse sand, fine-med gravel USCS sp 26HB
90	92		tan fine sand, wet, USCS sm 18HB
92	100		Tan fine sand, wet, USCS sm

DRILLING / WELL LOG

Boring/Well 2 CLIENT LILCO Page 1 of 1

Location Brookhaven Comb. Turb. Fac. Shoreman Date 11/30/89

Total Depth Drilled 100' Type of Sample Auger Ret. Interval cont

Casing Installed 80' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv Driller D.Klaus

Prepared By Charles Sosik - Geologist

Sample Depth From	To	OVA Reading (ppm)	Sample Description
0	1/2'		Bluestone base
1/2	2		topsoil, med gravel
2	13		tan med sand, USCS sp-sm
13	25		tan med sand,coarse gravel USCS gm
25	55		tan med sand,very coarse gravel USCSgm
55	75		lt tan fine-med sand, fine gravel USCS sp
75	100		lt tan-white fine sand USCS sm

DRILLING / WELL LOG

Boring/Well 3 CLIENT LILCO Page 2 of 2

Location Brookhaven Comb. Turb. Fac. Shoreman Date 11/30/89

Total Depth Drilled 100' Type of Sample S.S Interval 2'

Casing Installed 80' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv Driller D.Klaus

Prepared By Charles Sosik - Geologist

Sample Depth From	To	OVA Reading (ppm)	Sample Description
70	72		lt tan fine sand USCS sm 29HB
75	77		lt tan fine sand USCS sm 25HB
80	82		lt tan med-fine sand USCS sp-sm 23HB
85	87		lt tan med-fine sand USCS sp-sm 20HB
90	92		lt tan med-fine sand USCS sp-sm 18HB
92	100		tan med-coarse sand, med gravel lt tan med-fine sand USCS sp-sm

DRILLING / WELL LOG

Boring/Well 4 CLIENT LILCO Page 1 of 1

Location Brookhaven Comb. Turb. Fac. Shoreman Date 11/30/89

Total Depth Drilled 100' Type of Sample Auger Ret. Interval cont

Casing Installed 80' Screen Installed 20'

Drilling Method H.S.Auger Drilling Fluid Used None

Drilling Contractor MPC Environmental Serv Driller D.Klaus

Prepared By Charles Sosik - Geologist

Sample Depth From	To	OVA Reading (ppm)	Sample Description
0	1/2'		Bluestone base
1/2	2		topsoil, med gravel
2	13		tan med sand, USCS sp-sm
13	25		tan med sand,coarse gravel USCS gm
25	55		tan med sand,very coarse gravel USCSgm
55	75		lt tan fine-med sand, fine gravel USCS sp
75	100		lt tan-white fine sand USCS sm

Blasland Bouck & Lee

Monitoring Well #1D

Boring Logs

SUBSURFACE LOG

PROJECT: Anchor Chemical

PROJECT NO: 269.01

DATE: 11/19/91

BORING NO: B-1D

RECORDED BY: J.A. Schaefer

DRILLER: Don Klaus (MPC)

DRILL TYPE: Hollow Stem Auger

BOREHOLE DIAMETER: 10"

BOTTOM OF BORING (BOB): 122'

SAMPLER TYPE: Split Spoon

WEATHER: Clear skies and sunny

SAMPLE NO.	DEPTH FROM-TO FEET	RECOV. (ft)	NO. OF BLOWS Per 6"	HNU (ppm)	SAMPLE DESCRIPTION
	0-.15	-	-		Asphalt
	.15-1	-	-		Poorly sorted sand and gravel.
1	1.5-3.5	1	5,10,20,28	1	Poorly sorted fine to coarse sand with some gravel; moist; no odor.
2	5-7	.65	7,17,19,36	1	Light brown fine to medium sand; dry.
3	10-12	.9	7,17,27,28	11	Light brown fine sand with some medium to coarse sand and gravel. Cobble chips throughout.
4	15-17	1.6	5,11,19,26	2.5	15-16' Same as above. 16-16.6' Fe stained poorly sorted sand and gravel; moist; no odor.

Remarks: HNU background levels ranged from 4 to 6 ppm throughout the day.

SUBSURFACE LOG

PROJECT: Anchor Chemical

PROJECT NO: 269.01

DATE: 11/19/91

BORING NO: B-1D

RECORDED BY: J.A. Schaefer

DRILLER: Don Klaus (MPC)

DRILL TYPE: Hollow Stem Auger

BOREHOLE DIAMETER: 10"

BOTTOM OF BORING (BOB): 122'

SAMPLER TYPE: Split Spoon

WEATHER: Clear skies and sunny

SAMPLE NO.	DEPTH FROM-TO FEET	RECOV. (ft)	NO. OF BLOWS Per 6"	HNU (ppm)	SAMPLE DESCRIPTION
5	20-22	1.6	7,12,17,23	1.5	20-20.66' Poorly sorted, light brown sand and gravel. 20.66-21.83' Fe stained coarse sand and gravel; trace cobbles.
6	25-27	0.16	5,7,11,22 7,11,14,0	28	Rock caught in spoon tip. Fine to medium sand; rock caught in tip of spoon.
7	30-32	1.25	5,11,15,21	1	Light brown fine sand and medium coarse sand.
8	35-37	-			No recovery.
9	36-38	1.25	5,7,11,15	1	Loose, poorly sorted medium to coarse sand with some gravel; moist; Fe stained.

Remarks:

SUBSURFACE LOG

PROJECT: Anchor Chemical

PROJECT NO: 269.01

DATE: 11/191

BORING NO: B-1D

RECORDED BY: J.A. Schaefer

DRILLER: Don Klaus (MPC)

DRILL TYPE: Hollow Stem Auger

BOREHOLE DIAMETER: 10"

BOTTOM OF BORING (BOB): 122'

SAMPLER TYPE: Split Spoon

WEATHER: Clear skies and sunny

SAMPLE NO.	DEPTH FROM-TO FEET	RECOV. (ft)	NO. OF BLOWS Per 6"	OVM (ppm)	SAMPLE DESCRIPTION
10	40-42	.16	5,11,13,17	3.7	Same as above.
11	45-47	0.25	5,11,17,26	1.8	Brown well sorted medium sand; moist, no odor.
12	50-52	1.5	5,9,13,20	1.4	Light brown medium to coarse sand with some gravel; moist; no odor.
13	57-59	0.75	Not recorded	1.1	Light brown medium sand with some coarse sand; trace gravel; moist; no odor.
14	59-61	0.25	4,7,7,10		Light brown medium sand with some gravel; no odor.

Remarks. The water table was encountered approximately 59.75' below land surface. Sample number 14 and 15 composited for laboratory BNA, PEST/PCB, Metals, PCBs and phenols analyses (sample time 1525).

SUBSURFACE LOG

PROJECT: Anchor Chemical

PROJECT NO: 269.01

DATE: 11/20/91

BORING NO: B-1D

RECORDED BY: J.A. Schaefer

DRILLER: Don Klaus (MPC)

DRILL TYPE: Hollow Stem Auger

BOREHOLE DIAMETER: 10"

BOTTOM OF BORING (BOB): 122'

SAMPLER TYPE: Split Spoon

WEATHER: Cloudy and cool

SAMPLE NO.	DEPTH FROM-TO FEET	RECOV. (ft)	NO. OF BLOWS Per 6"	OVM (ppm)	SAMPLE DESCRIPTION
15	61-63	0.75	4,6,10,13		Same as above.
16	65-67	1	6,8,13,24	0	Light brown fine sand with some medium sand, trace gravel.
17	70-72	1.1	4,6,6,9	0	Same as above with some silt.
18	75-77	-	5,6,7,2	0	Heaved sand in augers (2') going back down with spoon to clean out heaved sand
19	75-77	0.6	6,8,9,17	0	Light brown fine sand with some medium sand and silt, trace coarse sand.

Remarks:

SUBSURFACE LOG

PROJECT: Anchor Chemical

PROJECT NO: 269.01

DATE: 11/20/91

BORING NO: B-1D

RECORDED BY: J.A. Schaefer

DRILLER: Don Klaus (MPC)

DRILL TYPE: Hollow Stem Auger

BOREHOLE DIAMETER: 10"

BOTTOM OF BORING (BOB): 122'

SAMPLER TYPE: Split Spoon

WEATHER: Cloudy and cool; occasional showers.

SAMPLE NO.	DEPTH FROM-TO FEET	RECOV. (ft)	NO. OF BLOWS Per 6"	OVM (ppm)	SAMPLE DESCRIPTION
20	80-82	1.5	9,14,14,23	0	Light brown well sorted fine to medium sand, wet.
21	85-87	1.5	7,9,13,18	0	85-86.25 same as above. 86.25-86.6 Orange silte with a trace of fine sand. Silt 86.66-86.75 fine sand.
22	90-92	0.3	7,19,23,29	0	Light brown fine sand with some medium sand, trace gravel.
23	95-97	1.5	5,7,19,27	0	Light brown fine sand with some medium to coarse sand.

Remarks: Sand bailed heaved sand prior to sampling from 75' through 120' below land surface.

SUBSURFACE LOG

PROJECT: Anchor Chemical

PROJECT NO: 269.01

DATE 11/20/91

BORING NO: B-1D

RECORDED BY: J.A. Schaefer

DRILLER: Don Klaus (MPC)

DRILL TYPE Hollow Stem Auger

BOREHOLE DIAMETER: 10'

BOTTOM OF BORING (BOB): 122'

SAMPLER TYPE: Split Spoon

WEATHER: Cloudy and cool; occasional showers

SAMPLE NO.	DEPTH FROM-TO	RECOV. (ft)	NO. OF BLOWS Per 6"	OVM (ppm)	SAMPLE DESCRIPTION
24	100-102	1.6	8,11,12,19	0	Light brown poorly sorted fine to medium sand with some coarse sand.
25	105-107	.25	4,8,10,15	0	Same as above.
26	110-112	83	5,11,17,24	0	Poorly sorted fine to coarse sand, trace gravel
27	115-117	1.6	4,11,14,22	0	Same as above with a trace of coarse gravel
28	120-122	1.6	1,9,11,14	0	Tan fine sand with some medium sand

<u>Remarks:</u> Sample #28 retained for VOA, BNA, Pest, PCBs and Metals laboratory analysis (sample [illegible]).
Boring grouted up to land surface (1500 lbs of portland cement used).

Blasland, Bouck & Lee

Description of Soil Sample MW-1D (122')

D. Anson 1/28/92

J. DeMartinis 269.01

Soil Samples from the Anchor Chemical Site S.J. Sucharski

Based on our experience; discussions with the USGS, Nassau County DPW, and local well drillers; and review of reference materials (USGS Water Supply Papers 1613-A and 1825, for example), the following sedimentological and mineralogical characteristics apply to the Magothy Formation:

(1) Most of the sand layers/lenses in the Magothy are gray or tan in color (due to intergranular fillings of white clay);

(2) The sand in the upper part of the Magothy is generally fine to medium-grained.

(3) the sand consists mostly of quartz, some lignite, muscovite, and approximately 2-3% heavy minerals. Pyrite and iron oxide nodules are common; and

(4) The upper portion of the Magothy is somewhat clayey and generally contains layers of clay and silt.

The split-spoon core barrel samples that were collected and logged at the 10 to 120 - foot below grade levels were all consistent and indicated:

(1) A reddish-brown color (typical of upper glacial deposits).

(2) They were predominantly coarse-grained and gravelly (in places) as opposed to fine to medium-grained. No layers of clay and silt were encountered.

(3) No lignite, pyrite and/or iron oxide nodules was observed in any soil samples and in the drill cuttings.

(4) Quartz, though dominant, comprised only 70 - 80% of the mineral grains.

(5) The remainder of the sand grains consisted of feldspar, igneous and metamorphic rock fragments, chlorite, biotite and heavy minerals such as hornblende, pyroxene and garnet. Feldspar, rock fragments, biotite and chlorite are not characteristic of the Magothy Formation.

(6) The high percentage of heavy minerals, particularly garnet, is uncharacteristic of the Magothy.

(7) Very little muscovite was noted in the samples.

Blasland, Bouck & Lee

Resumes of Individuals

JAMES M. DEMARTINIS
VICE PRESIDENT

PROFESSIONAL HISTORY

Mr. DeMartinis has over 12 years of experience as a geologist and hydrogeologist with the United States Geological Survey, Maine Geological Survey, Geraghty & Miller, Inc., and Roux Associates. Mr. DeMartinis has managed numerous soil and ground-water investigations for industrial clients involving Superfund sites, RCRA facilities, hydrocarbon spills, and property transfers.

EDUCATION

MA/Geology, 1986, Queens College, City University of New York
BS/Geology, 1976, Bates College

PROFESSIONAL AFFILIATIONS

National Water Well Association
Geological Society of America
American Association for the Advancement of Science
American Society of Agronomy
Weed Science Society of America

EXPERIENCE

Agricultural Chemical Monitoring: Carried out an investigation of certain herbicides to determine potential impacts on ground-water quality. Selected 11 sensitive areas from coast to coast, visited individual farm/grove sites in these areas, and conducted a soil boring and monitoring well installation program. Project was designed to determine the probable impact of the selected herbicides on ground water in areas underlain by permeable soils.

Evaluated the soil and hydrogeologic characteristics of approximately 500 counties and 34 states to identify areas vulnerable to ground-water contamination. Surveyed state agencies in agricultural states to obtain information on pesticide monitoring programs and data. Performed detailed analyses of the soil and geology in counties where pesticides have been detected in ground water to assess the reasonableness of the findings. Participated in client's presentations to the USEPA and the USEPA Science Advisory Panel. The results of these vulnerability analyses were the basis for subsequent targeted ground-water monitoring programs.

Served as Study Director for two small-scale retrospective ground-water monitoring studies. Four sites were located, characterized, and are currently being monitored in the Atlantic Coastal Plain, Gulf Coastal Plain, and Southwestern United States.

Designed and implemented a nationwide ground-water monitoring program for three agricultural chemicals. Starting with a list of counties evaluated as vulnerable to pesticide leaching, a statistically similar subgroup was selected for monitoring of existing domestic supply wells. Individual wells were selected, inspected for proper siting and construction, and sampled according to a detailed protocol on a quarterly schedule. Samples were shipped to a contract laboratory, and analytical results were evaluated.

Conducted a domestic well sampling program in the Central Sand Plains region of Wisconsin to determine if agricultural chemicals present in ground water are the result of leaching from pesticide treated soils.

Conducted a small-scale prospective ground-water monitoring study for an agricultural chemical in the North Carolina Coastal Plain.

Investigated pesticide occurrences in soil and ground water in three California counties and reported findings to state regulatory agencies on behalf of the client. Testified at two public hearings pursuant to the California Food and Agriculture Code.

Investigated the extent of chlordane in soil and ground water at a former storage facility, leading to development of remedial alternatives.

Conducted a large-scale retrospective monitoring program for a herbicide in the Dougherty Plains, Georgia; the Central Sands Region, Wisconsin; and the midwest corn belt in Iowa and Illinois.

Conducted a detailed exploratory program involving test pit excavation and soil borings to determine the extent of fuel oil contamination in soil and ground water at a storage facility. Developed and implemented a remedial plan involving the removal of free product from the water table.

Evaluated the feasibility of in-situ bioreclamation for a pesticide formulating plant in Massachusetts. Successfully mapped complex ground-water flow patterns. Conducted detailed aquifer tests to determine the suitability of the aquifer for the withdrawal, treatment, and reinjection of water.

RI/FS, Hazardous Waste Site Investigations: As Project Manager, carried out the Remedial Investigation Feasibility Study (RI/FS) for a Superfund site in Woburn, Massachusetts. This site was originally ranked number four on the National Priorities List. Negotiated, planned and supervised subsurface investigations at this 300-acre site, including an extensive electromagnetic survey/collection of over 2,000 soil samples from test pits and borings, and the installation of 24 monitoring wells. Additional work, including a wetlands/floodplain assessment and subsurface evaluation has been completed, and the RI/FS accepted. A Record-of-Decision has been issued by the United States Environmental Protection Agency (USEPA) Region I. The conceptual design for a ground-water intercept system was prepared on the basis of this work.

Investigated Superfund site in Acton, MA. The purpose of the investigation was to determine the sources of 1,1,1- trichloroethane and other organic chemicals effecting a municipal supply well The investigation was designed to pinpoint the locations of contaminant sources through a detailed examination of ground-water flow and solute transport in the area of the contaminated well and by tracking the plume(s) of contaminants back to their sources

Investigated a New York State-listed Superfund site in Modena, NY Coordinated and carried out a soil and ground-water sampling program, analyzed the data generated, and prepared a hydrogeologic assessment report.

Managed the permit application for a non-hazardous industrial waste landfill in Upstate New York by conducting a detailed soils and hydrogeologic investigation.

Blasland, Bouck & Lee

Resumes of Individuals

BACHAR EL SABEH
SENIOR PROJECT ENGINEER

PROFESSIONAL HISTORY

Mr. El Sabeh has more than eight years of experience in civil and mechanical engineering, with emphasis on ground-water investigation activities. His project experience includes both field and administrative work, as well as performance of computer-aided design and analysis.

EDUCATION

BE/Mechanical Engineering, 1982, State University of New York at Stony Brook
MS program/Civil Engineering (Structural), Polytechnic Institute of New York

PROFESSIONAL AFFILIATIONS

Association of Ground Water Scientists and Engineers
American Society of Mechanical Engineers
American Society of Civil Engineers

EXPERIENCE

Municipal and Industrial Water Supply: Formerly responsible for the management of a large Long Island-based water well contracting firm, serving as Vice President of Engineering and Manager of Field Operations. Instrumental in building the business base and in setting up employee training programs in health and safety and hazardous waste management.

Widely experienced in the preparation and review of specifications for water wells and in the review of bidding documents:

- Directed the design and construction of municipal water well supply systems for numerous Long Island water companies.
- Directed design and construction for rehabilitation of low yield municipal water wells and complete evaluated of existing pumping systems.
- Directed and evaluated geologic studies to determine effectiveness of various drilling methods including reverse and direct rotary, hollow-stem auger, and cable tool.
- Participated in investigations to determine hydrogeologic characteristics of unconsolidated aquifers in Nassau County, Long Island. Performed drawdown/recovery and slug tests and collected water level data.
- Project Manager and lead Project Engineer for the design and construction of supply and diffusion water wells for heat pump systems for major Long Island office buildings and industries.
- Assisted in negotiation, compliance, and permitting for New York State Department of Environmental Conservation (NYSDEC) water supply drilling and discharge program.
- Assisted Nassau County Department of Public Works (NCDPW) in conducting drilling programs and gamma and electric resistivity surveys to evaluate water well sites for potential water supply development.
- Provided consulting service to local health department agencies in development and implementation of regulations governing the installation of test borings and water sampling for property subdivision.

Hazardous Waste Site Investigations: Involved in the design and installation of observation wells and test borings to investigate the presence and extent of contaminated soils and ground water. This work has been carried out in connection with environmental site assessments and in response to past soil and ground-water remediation.

Under previous employment, participated in an extensive environmental assessment and remediation program at the Huntington, Long Island landfill and a resource recovery center in East Northport.

Currently involved in the management of a $1 million environmental contracting project which includes on-the-job environmental monitoring services during construction of a major sludge dewatering plant for the city of New York on Wards Island. Responsible for all aspects of site remediation including drum removal and remediation of contaminated ground water.

Managed field investigations involving soil sampling and monitoring well installation at numerous waste disposal sites.

Supervised monitoring well installations in soils and refuse. Directed studies to evaluate effectiveness of various ground-water drilling techniques while complying with United States Environmental Protection Agency (USEPA) and NYSDEC regulations.

Supervised the installation and sampling of monitoring and recovery wells for the removal of gasoline-contaminated ground water at various Long Island sites.

Project Manager for study to determine saltwater intrusion potential; responsible for the design of monitoring programs to warn against intrusion.

Safety officer responsible for ensuring compliance of workers with corporate and OSHA standards at hazardous waste sites.

Designed and conducted a soil vapor installation used to extract vapors through vacuum pumps.

Performed Phase II investigation for the NYSDEC at several landfill sites on Long Island, New York.

Designed ground-water recovery and treatment systems for unconsolidated aquifers for multiple sites in New York. Recovery designs included single and multiple recovery well systems and reinjection wells.

Conducted ground-water evaluations on well design and maintenance, and completed rehabilitation of low yield wells. Rehabilitation included the use of advanced development methods and special chemicals.

JAMES A. SCHAEFER, JR.
PROJECT GEOLOGIST

PROFESSIONAL HISTORY

Mr. Schaefer has more than two years experience in geologic/hydrogeologic investigations and hazardous waste site remediation. Areas of specialty include ground-water monitoring and subsurface remediation.

EDUCATION

BS/Geology, 1988, State University College of New York at Cortland

PROFESSIONAL CERTIFICATION

OSHA-Approved 40-Hour Hazardous Waste Site Health and Safety Training (1989)

PROFESSIONAL AFFILIATIONS

National Water Well Association
Association of Ground Water Scientists
Engineering American Water Resources Association

EXPERIENCE

Hazardous Waste Investigation: Assisted in a landfill investigation on Long Island that involved the geologic logging of 50 test pits and 20 soil borings, and the installation of monitoring wells. Responsibilities included preparing all equipment and supplies needed for field operations; conducting continuous air monitoring during all test pit and drilling operations following health and safety guidelines; the daily calibration of instruments; photoionization screening of soil; supervising the installation and development of four-inch diameter monitoring wells to state specifications; sampling surface water and subsurface water; and organizing and incorporating geologic data into final report. Also responsible for overseeing entire remediation project which involved the excavation of all contaminated material on site; determining when virgin ground was exposed during all phases of excavation; and observing all hazardous situations.

Assisted in an exploratory soil boring program at a Superfund site Massachusetts undertaken to determine the extent of subsurface B soil contamination; collected samples used to evaluate edial alternative analysis.

Assisted in a Phase II subsurface investigation at a Superfund site in New Jersey. Duties involved geologic logging of soil borings; field screening of soil samples via photoionization; collection of soil samples for laboratory analyses following New Jersey Department of Environmental Protection (NJDEP) guidelines; and maintenance of a safe working environment.

Supervised a Phase I/Phase II river bank investigation in Massachusetts. The investigation involved ascertaining the areal extent of subsurface oil (PCB transformer oil), characterizing subsurface stratigraphy, determining the underlying aquifer's hydraulic coefficients, and designing and implementing a remedial plan to recover the subsurface oil before it entered the river. Responsibilities included supervising the drilling of 27 soil borings, the installation of 7 piezometers, and the installation of 15 well points.

Assisted in a six month statistical PCB sampling program in West Virginia. Responsibilities included calculating total surface areas in PCB contaminated rooms; determining PCB wipe sampling points by formulating surface area grids; sampling over 500 PCB wipe samples; and reviewing all generated calculations, grids, laboratory results, and other pertinent notes and data as part of Quality Assurance and Quality Control program.

Implemented a two phase PCB risk assessment in Evendale, Ohio. Determined the extent of PCB contamination in an industrial drainage ditch to evaluate potential risks to ground's crew workers. At designated locations in the ditch, 2-foot core sediment samples were taken at the center and corners of a hexagonal grid.

Supervised subsurface investigations at four Long Island, New York gasoline service stations. The investigations included the drilling of soil borings and installation of observation wells. The horizontal and vertical extent of the site's subsurface soil contamination was defined.

Soil Conservation: Served as Assistant to the District Conservationist (Cortland, New York) for the United States Department of Agriculture (USDA) Soil Conservation Service. Duties involved determining highly erodible lands and wetlands from interpretation of soil surveys, topographical maps, field investigations, and other means available; and contacting county land owners to gather physical data such as crop field history, field arrangements, land use, and other pertinent information. Recorded highly erodible land areas on aerial photographs, and assisted field office staff with engineering surveys.

Environmental Testing: As geologist for an environmental testing lab, acquired optical mineralogy skills. Able to identify air and bulk asbestos samples using phase contrast and polarized light microscopes, differentiating between amounts of asbestos, other silicates, carbonates, and other minerals. Experienced in operating a transmission electron microscope.

STANLEY J. SUCHARSKI
MANAGER, GEOLOGY

PROFESSIONAL HISTORY

Mr. Sucharski has over seven years of experience in the management of field investigations involving a wide variety of soil and ground-water sampling techniques for industrial clients involving Superfund sites, RCRA facilities, property transfer investigations, hydrocarbon spills and monitoring for agricultural chemicals (both small and large scale retrospective and prospective studies).

EDUCATION

BS/Geology, 1981, State University of New York at Brockport

CERTIFICATIONS

EPA AHERA Asbestos Inspector

PROFESSIONAL AFFILIATIONS

National Water Well Association
Association of Ground-Water Scientists and Engineers
Geologic Society of America'

EXPERIENCE

RI/FS Hazardous Waste Site Invesigations: Field Manager of Remedial Investigation phase at major Superfund site in Nassau County, N.Y. Responsible for design, coordination and implementation of all aspects of the investigation from start to finish including; geophysical surveys, soil vapor screening in the unsaturated zone, collection of soil samples from test borings for field screening and laboratory analysis, monitoring well installation, sampling of adjacent harbor sediments, monitoring well sampling, aquifer testing, and sample shipment to laboratory. All work was done under close scrutiny of NYSDEC and completed ahead of schedule and under budget. Tasks also included report writing and data analyses and interpretation.

Designed sampling and analysis protocol for stockpiled soils at a plant site to determine the ultimate disposition of such soils. Responsible for the design of a grid system for each pile so that representative samples could be collected and analyzed for PCBs, EP Toxicity and volatile organic compounds.

Prepared contractors bid package for the excavation, transportation, disposal and backfilling of soil contaminated with volatile organics at an electrical components facility in Arkansas. Responsibilities included the evaluation of contractor's bids, selection of contractors, and supervision of all remedial work.

Managed and coordinated five long duration pumping tests to aid in designing an intercept system for plumes of organic chemicals and chlorides.

Supervised the collection of over one thousand soil and ground-water samples for priority pollutant analyses as part of major Superfund investigations.

Supervised installation, development and sampling of monitoring wells at numerous waste disposal sites for compliance monitoring.

Supervised soil sampling and monitoring well installation programs to assess the feasibility of in situ bioreclamation as an alternative for remediation of organic and metals contamination at plant sites in New Jersey and New York.

Prepared and implemented ground-water monitoring programs for industrial plant compliance in Connecticut, New Jersey and New York.

Inspected, observed and critiqued EPA consultant install and sample monitoring wells at the Number 2 listed Superfund site.

Managed field investigations involving soil sampling, monitoring well installation and water quality sampling at a Superfund-listed site located in the recharge area of a sole source aquifer.

Investigated the extent of volatile organic chemicals in soil and ground water at a large manufacturing facility in Connecticut.

Investigated the possible source(s) of ground-water contamination that closed a municipal well field by installing monitoring wells and conducting detailed site evaluations.

Conducted volatile organic screening investigations of the unsaturated zone to determine optimum monitoring well locations at plant sites in New Jersey, Connecticut and New York.

Prepared, implemented and enforced Health and Safety Plan for field investigations at various Superfund-listed sites.

Part of field party in comprehensive, three to four month electrical earth resistivity surveys at a Superfund-listed site in Delaware and an industrial site in Kansas.

Agricultural Chemical Monitoring: Co-managed and implemented large-scale retrospective ground-water monitoring program for a herbicide. Over 240 wells in four counties throughout the United States were sampled. Responsibilities included well selection, design of sampling procedures and coordination of sampling activities.

Supervised the installation of monitoring wells at agricultural sites in Florida and New Jersey.

Managed detailed site explorations and conducted percolation tests to locate potential recharge/reinjection basins and trenches.

Participated in small-scale prospective ground-water monitoring study for agricultural chemical in North Carolina.

Hydrocarbon Investigations: Responsible for design and implementation of a ground-water exploration and monitoring program for major gasoline spill in Poughkeepsie, New York.

Conducted earth conductivity survey and test boring program to determine the source(s) and extent of an oil spill in a karst terrain.

Field managed a hydrogeologic investigation at a former manufacturing facility in Pennsylvania to delineate the source(s) and extent of subsurface contamination.

Conducted test boring and well drilling at former coal gasification facility to determine the extent of free product floating on the water table.

Blasland, Bouck & Lee

Drilling Contractor Contacts

Telephone Conversation Logs

MEMORANDUM

To:
S. Sucharski
From:
B. El Sabeh
Re:
Anchor Chemical

Date:
1/28/92
File No.:
269.01.01
cc:
J. DeMartinis

With regards to the above project, I contacted six (6) well drilling companies to solicit proposals for the drilling and installation of four (4) monitoring wells to a depth of 140 feet, each, by utilizing the Hollow-Stem Auger Method using 6.25" Internal Diameter (I.D.) or larger augers.

The specifications given to these companies were:
- Location of the site is West John Street, Hicksville, New York
- Drill and sample to 140 feet and possibly deeper depending where the Magothy Strata is encountered.
- Install 4-inch casing and screen.
- Install gravel pack, bentonite pellets and cement grout by the tremie method.

After speaking with each company, the following responses were documented:
- Hydro Group Inc. of Hauppauge, New York and Environmental Drilling Inc. of West Creek, New Jersey stated that their hollow-stem auger machines Gus Peck and Mobile will be able to drill to 100 feet, however they will not guarantee drilling any deeper. Cement grout can be placed using the tremie method but no guarantees can be made with regards to the gravel pack and bentonite pellets. The I.D. of the augers will be 6.25".
- Empire Soil Investigations of Highland Park, New Jersey are willing to guarantee the installation of the wells with 8-inch I.D augers and using the tremie method for gravel, bentonite pellets and cement grout to a maximum depth of 80 feet. The machine that will be used is the Failing F-10WT with a torque converter and it is one of the most powerful machines in the industry. Using 6.25" I.D. augers they will drill to 120 feet but they will not be able to use the tremie method for gravel pack and bentomite pellets because the annulus between 6.25" I.D. augers and 4-inch casing (4.50" Outside Diameter - O.D.) is:

  $$\frac{6.25-4.50}{2} = 0.875\text{" or 7/8 of an inch}$$

  and this is too small for any tremie pipe to be used on anything deeper than 80 feet.
- Parratt-Wolf, Inc. of East Syracuse, New York and M&R Soil Investigations, Inc. of Hammonton, New Jersey stated that their Mobile drilling equipment will not be able to drill deeper than 100 feet and they will not guarantee the tremie method for the gravel pack and bentomite pellets. The I.D. of the augers will be 6.25".

- D.L. Maher of North Reading, Massachusetts will be able to drill to 140 feet and possible to 200 feet and use the tremie method for the backfill material by using the BARBAR drilling equipment which is an Air-Rotary equipment and a borehole diameter of 10.00 inches. By using the Hollow-Stem Auger Method, they will guarantee drilling to 100 feet with a 6.25" I.D. augers and they cannot guarantee the tremie method for the gravel and bentonite pellets..

Most of these companies had expressed that they will attempt to drill deeper than the guaranteed depths that they committed to but that can only be accomplished after field activities start and no guarantees can be made on the final depths or the tremie method for the gravel and bentonite pellets. The use of bentonite slurry instead of bentonite pellets will make it much easier during the tremie operation.

Very Truly Yours,

Bachar Elsabeh

Bachar El Sabeh

List of companies and phone numbers:

-Hydro Group, Inc.
Hoffman Lane
Hauppauge, NY
(516) 234-1000
Mr. Bob Creci

-Parratt-Wolff, Inc.
East Syracuse, NY
(315) 437-1429
Mr. Mike Ellingworth

-Environmental Drilling,Inc.
Stafford Forge Road
West Creek, NJ
(908) 286-3232
Mr. Art Beck

-M&R Soil Investigations, Inc.
Hammonton, NJ
(609) 567-8960
Mr. Doug Walker

-Empire Soil Investigations
Highland Park, NJ
(908) 287-2224
Mr. Roger Logel

-D.L. Maher Company
North Reading, Massachusetts
(617) 933-3210
Mr. Kevin DeBasitis

Validated Laboratory Data

VOLATILE ORGANIC ANALYSIS - ANALYTICAL RESULTS - ALL SOLIDS REPORTED ON A DRY WEIGHT BASIS -page 1

Anson Environmental Sample Number Laboratory Sample Number			DW2 0095-01	DW3 0095-02	DW4 0095-03	Trip Blank 010 0095-04	DW01 0103-01	DW05 0103-02	DW06 0103-03	DW07 0103-04	DW08 0103-05	DW09 0103-06	DRAIN 0103-07	RB-1 0103-10	Trip Blank 011 0103-11
Remarks														Rinse Blank	
Units			ug/Kg	ug/Kg	ug/Kg	ug/L	ug/Kg	ug/Kg	ug/Kg	ug/Kg	ug/Kg	ug/Kg	ug/Kg	ug/L	ug/L
VOLATILE COMPOUNDS	Quant. Limit (Aq)	Quant. Limit (Sol)													
Chloromethane	10	10	UJ	UJ	UJ										
Bromomethane	10	10	UJ	UJ	UJ										
Vinyl Chloride	10	10	UJ	UJ	UJ										
Chloroethane	10	10	UJ	UJ	UJ								8		
Methylene Chloride	5	5	2100 B	1100 B	7 B	3 J	7 B	5 U	5 U	6 B	9 B	7 B	6 B	3 J	5 J
Acetone	10	10	UJ	UJ	UJ		10 R	5 B	27 R	16 R	15 B				
Carbon Disulfide	5	5	UJ	UJ	UJ					5 J		21			
1,1-Dichloroethene	5	5	UJ	UJ	UJ										
1,1-Dichloroethane	5	5	1600 J	UJ	UJ										
Total 1,2-Dichloroethene	5	5	UJ	UJ	UJ										
Chloroform	5	5	UJ	UJ	UJ										
1,2-Dichloroethane	5	5	UJ	UJ	UJ										
2-Butanone	10	10	UJ	UJ	UJ										
1,1,1-Trichloroethane	5	5	3300 J	UJ	UJ										
Carbon Tetrachloride	5	5	UJ	UJ	UJ										
Vinyl Acetate	10	10	UJ	UJ	UJ										
Bromodichloromethane	5	5	UJ	UJ	UJ										
1,1,2,2-Tetrachloroethane	5	5	UJ	UJ	UJ										
1,2-Dichloropropane	5	5	UJ	UJ	UJ										
trans-1,3-Dichloropropene	5	5	UJ	UJ	UJ										
Trichloroethene	5	5	UJ	UJ	UJ										
Dibromochloromethane	5	5	UJ	UJ	UJ										
1,1,2-Trichloroethane	5	5	UJ	UJ	UJ										
Benzene	5	5	UJ	UJ	UJ	UJ								UJ	UJ

VOLATILE ORGANIC ANALYSIS - ANALYTICAL RESULTS - ALL SOLIDS REPORTED ON A DRY WEIGHT BASIS -page 2

Ansan Environmental Sample Number			DW02	DW03	DW04	Trip Blank 010	DW01	DW05	DW06	DW07	DW08	DW09	DRAIN	RB-1	Trip Blank 011
Laboratory Sample Number			0095-01	0095-02	0095-03	0095-04	0103-01	0103-02	0103-03	0103-04	0103-05	0103-06	0103-07	0103-10	0103-11
Remarks														Rinse Blank	
Units			ug/Kg	ug/Kg	ug/Kg	ug/L	ug/Kg	ug/Kg	ug/Kg	ug/Kg	ug/Kg	ug/Kg	ug/Kg	ug/L	ug/L
VOLATILE COMPOUNDS	Quant. Limit (Aq)	Quant. Limit (Sol)													
cis-1,3-Dichloropropene	5	5	UJ	UJ	UJ										
Bromoform	5	5	UJ	UJ	UJ										
2-Hexanone	10	10	UJ	UJ	UJ										
4-Methyl-2-Pentanone	10	10	UJ	UJ	UJ										
Tetrachloroethene	5	5	UJ	UJ	UJ										
Toluene	5	5	4000 J	1100 D	64 J	1 J			5 J		200			UJ	UJ
Chlorobenzene	5	5	UJ	UJ	UJ	UJ								UJ	UJ
Ethylbenzene	5	5	4000 J	UJ	UJ	UJ								UJ	UJ
Styrene	5	5	UJ	UJ	UJ	UJ								UJ	UJ
Total Xylenes	5	5	67,000 J	UJ	UJ	UJ								UJ	UJ
Quantitation Limit Multiplier			420	230	1.4	1.0	1.3	1.1	1.0	1.1	1.9	1.4	1.2	1.0	1.0
Date of Sample Collection			8/10/91	8/10/91	8/10/91	8/10/91	8/11/91	8/11/91	8/11/91	8/11/91	8/11/91	8/11/91	8/11/91	8/11/91	8/11/91
Date Sample Received by Laboratory			8/11/91	8/11/91	8/11/91	8/11/91	8/12/91	8/12/91	8/12/91	8/12/91	8/12/91	8/12/91	8/12/91	8/12/91	8/12/91
Date of Sample Analysis			8/21/91	8/21/91	8/21/91	8/21/91	8/21/91	8/21/91	8/21/91	8/21/91	8/21/91	8/21/91	8/21/91	8/21/91	8/21/91
Instrument Used for Analysis			MS-5995A	MS-5995A	MS-5995A	MS-5995D	MS-5995A	MS-5995A	MS-5995A	MS-5995A	MS-5995A	MS-5995A	MS-5995A	MS-5995D	MS-5995D

NOTES:

- \- Compound was not detected.
- U This compound should be considered "not-detected" since it was detected in a blank at a similar level.
- R Unreliable result - Compound may or may not be present in this sample.
- J Quantitation is approximate due to limitations identified during the quality assurance review (data validation).
- UJ This compound was not detected, but the quantitation limit is probably higher due to a low bias identified during the quality assurance review.

Unvalidated Laboratory Data

		IB#3	IB#3	IB#3	IB#4	IB#4	IB#5	IB#5	IB#6	IB#6	MW-1D	MW-1D	MW-4	MW-4	MW-6	MW-6
		25-27'	30-32'	35-37	10-12'	15-17'	15-17'	35-37'	30-32'	40-42'	59-61'	120-122'	60-62'	75-77'	60-62'	74-76'
Compound																
Chloromethane	UG/KG															
Bromoethane	UG/KG															
Vinyl Chloride	UG/KG															
Chloroethane	UG/KG															
Methylene Chloride	UG/KG	5J	5B	6B	6B	6B	3BJ	2BJ	3BJ	2BJ	6B	7B	14B	7B	7B	6B
Acetone	UG/KG	26B	15B	15B	25D	19D	110B	110B	19B	12B	13B	110B	54B	39B	26B	11BJ
Carbon Disulfide	UG/KG															
1,1-Dichloroethene	UG/KG															
1,1-Dichloroethane	UG/KG															
Total 1,2-Dichloroethene	UG/KG															
Chloroform	UG/KG															
1,2-Dichloroethane	UG/KG															
2-Butanone	UG/KG															
1,1,1-Trichloroethane	UG/KG															
Carbon Tetrachloride	UG/KG															
Vinyl Acetate	UG/KG															
Bromodichloromethane	UG/KG															
1,1,2,2-Tetrachloroethane	UG/KG															
1,2-Dichloropropanone	UG/KG															
trans-1-,3-Dichloropropene	UG/KG															
Trichloroethene	UG/KG															
Dibromochloroethane	UG/KG															
1,1,2-Trichloroethane	UG/KG															
Benzene	UG/KG															
cis-1,3-Dichloropropene	UG/KG															
Bromoform	UG/KG															
2-Hexanone	UG/KG															
4-Methyl-2-Pentanone	UG/KG															
Tetrachloroethene	UG/KG															
Toluene	UG/KG															
Chlorobenzene	UG/KG															

		IB#3	IB#3	IB#3	IB#4	IB#4	IB#5	IB#5	IB#6	IB#6	MW-1D	MW-1D	MW-4	MW-4	MW-6	MW-6
		25-27'	30-32'	35-37	10-12'	15-17'	15-17'	35-37'	30-32'	40-42'	59-61'	120-122'	60-62'	75-77'	60-62'	74-76'
Compound																
Ethylbenzene																
Styrene																
Total Xylenes																
Notes																
B-Found in the blanks																
D-Used in decon procedure																
J-Estimated																

Anson Environmental

Environmental Audits
Hazardous Waste
Asbestos Management
Groundwater Remediation
Storage Tank Management
Impact Statements
Wetland Investigations

256 Main Street
Northport, NY 11768
516-757-7090
(fax) 516-757-1229

March 12, 1992

Bernard J. Bottomley, Director
Engineering and Administrative Services
Newsday
Melville, NY 11747

Re: 500 West John Street, Hicksville, New York (the "SITE")

Dear Mr. Bottomley,

We have been asked to explain in greater detail the information available about drywell #2 in the rear of the building at the above referenced site.

Drywell # 2 is a new drywell, installed after the tenancy of Anchor Chemical. The sediment sampled in drywell #2 did show the following compounds above the laboratory detection limits:

pesticide	-alpha BHC	183 µg/kg
	-endrin	36 µg/kg
metals	-lead	1210 mg/kg
	-barium	90.2 mg/kg
semivolatile	-napthalene	9800 µg/kg
	-2-methylnapthalene	4100 µg/kg
	-butylbenzylphtalate	5100 µg/kg
	-bis(2-ethylhexyl) phtalate	25,000 µg/kg

Environmental Standards Inc., the data validator, urged that the recorded amounts for alpha BHC, the napthalenes and the phtalates be viewed with caution as they may have been introduced in laboratory as part of the analytical procedures. This means that these chemical compounds were most likely introduced during laboratory analysis of the sediment samples.

2yy

DEFT'S EXH. ZZ
IDENT. 3.9.95

Printed on Recycled Paper

The alpha-BHC compound is commonly used as an insecticide. Given the proximity of the site to Cantiague Park, these compounds probably originated in the Park.

The chemical compounds found in the sediment layer of the drywell (approximately 19 feet below the surface of the driveway) are relatively common for drywells located in active parking lots and driveways.

The current laboratory analyses indicate the total amount of the compound in the sample. Before the drywell is cleaned out, the soil beneath it will be tested again to determine the appropriate disposal method. The analytical technique used will only identify the quantity of the compounds which could leach into the soil. This is the hazardous component of the chemical compound. In almost every instance, the concentrations of the chemical compound will be substantially less than the total amount identified above.

Removal of the soil in the drywell can be accomplished through excavation and disposal of the soil and power washing the sides of the drywell. The cleaning of the drywell is accomplished by removing the slotted cover and vacuuming the drywell contents. This pumpout commonly takes less than 3 hours. The pumpout is accomplished by a licensed hauler and disposed of in accordance with all Federal, state and local regulations.

In summary, it is important to remember that this material is located nineteen feet below the surface of the driveway and is not accessible to building occupants in the course of routine daily activities at this location.

The contents of the drywell do not pose a health risk or hazard to building occupants.

We trust that this information is satisfactory for your purposes.

Very truly yours,

Dean Anson II

Melville, NY 11747-4250

Bernard J. Bottomley
Director
Engineering and Administrative Services
516 454-2315

7

February 14, 1992

Mr. Art Sanders
Spiegel Associates
375 North Broadway
Jericho, NY 11753

FW:
FOR YOUR
INFO.:
NEWSDAY

Re: 500 W. John Street, Hicksville, New York

Dear Art:

This will confirm our meeting to review the environmental issues and proposed lease conditions pertaining to the referenced premises.

DATE: Friday, February 21, 1992, 9:30 AM

PLACE: Spiegel Associates, 375 North Broadway, Jericho.

ATTENDEES: Philip Kucera, Times Mirror, Associate General Counsel
Janette Payne, Times Mirror, Staff Counsel
Michael Youchah, Times Mirror, Director, Real Estate, Engineering & Facilities
Jim Fitzgerald, Newsday, Sr. Vice President, Finance & Administration
Art Sanders, Spiegel, President
Spiegel Environmental Counsel
Spiegel Environmental Consulting Group

During my absence from the office during the week of 2/17/92, please contract Jim Fitzgerald (Telephone number: 454-2365) in the event that you need a contact prior to the meeting.

Very truly yours,

B. J. Bottomley

Bernard J. Bottomley

BJB:km

cc: P. Kucera
J. Payne
M. Youchah
J. Fitzgerald
(Directions to follow)

A Times Mirror
Newspaper

DEFT'S EXH. BBB
IDENT. 3·9·95

ROSENMAN & COLIN

575 MADISON AVENUE, NEW YORK, NY 10022-2585

TELEPHONE (212) 940-8800
CABLE HUCORAY NEWYORK
TELECOPIER (212) 940-8776
(212) 935-0679
TELEX 427571 ROSCOL (ITT)
971520 RCFLC NYK (W U)

SAMUEL I. ROSENMAN (1896-1973)
RALPH F. COLIN (1900-1985)

WASHINGTON OFFICE
1300 19TH STREET, N. W.
WASHINGTON, D. C. 20036
TELEPHONE (202) 463-7177

RICHARD G. LELAND
(212) 940-8700

February 5, 1992

VIA TELECOPIER AND REGULAR MAIL

Janette Payne, Esq.
Times-Mirror
780 3rd Avenue
40th Floor
New York, New York 10017

Re: 500 West John Street
Hicksville, New York (the "Premises")

Dear Ms. Payne:

As per our conversations of Monday and this morning, I am enclosing herewith a photocopy of the two most recent monthly status report given by our client's consultant, Anson Environmental, to Dorothy Allen of Region II of the United States Environmental Protection Agency ("EPA"). As we agreed, my client will be sending you copies of the approved Work Plan and Project Operations Plan ("POP") for the remedial investigation under separate cover.

As we discussed Monday, our client's consultants have completed virtually all of the tasks set forth in the POP. All interior work has been completed. The remaining task is the installation of deep monitoring wells at the site. Completion of the installation of the deep wells is pending approval by the EPA of a written request, made January 29, 1992, for a modification of the POP to allow for shallower wells than originally called for.

This will also confirm the information which I have received from my consultant indicating that no hazardous substances were found in any of the interior samples. Rather, the only place were any hazardous substances have been found in sampling conducted to date is in dry well number 2, located north of the building in the parking lot. Please note that dry well number 2 is upgradiant of the former manufacturing areas at the Premises.

DEFT'S EXH. CCC
IDENT. 3-9-95

Janette Payne, Esq.
February 5, 1992
Page 2

Please let me know if you are in need of any further information regarding these Premises and the remedial investigation.

Very truly yours,

Richard G. Leland

RGL/ams-r
cc: Mr. Arthur D. Sanders (w/out enc.)
Mr. Fred Worfel (w/out enc.) ✓
Alan D. Eidler, Esq. (w/out enc.)

Anson Environmental Ltd.

Environmental Audits
Hazardous Waste
Asbestos Management
Groundwater Remediation
Storage Tank Management
Impact Statements
Wetland Investigations

256 Main Street
Northport, NY 11768
516-757-7090
(fax) 516-757-1229

September 14, 1992

Mr. Tom Taccone, Project Manager
United States Environmental Protection Agency
Region II
26 Federal Plaza
New York, NY 10278

Re: Anchor Chemical Superfund Site
Administrative Order No. II CERCLA-90208

Dear Mr. Taccone,

We have enclosed a copy of the validated data for the soil cuttings drummed during the installation of monitoring wells and the indoor borings. This package was addressed to Mr. Jon Greco of the New York State Department of Environmental Conservation for his concurrence on our proposal for disposal of this material.

We trust this information is satisfactory for your purposes.

Very truly yours,

Dean Anson

Dean Anson II
Co-Facility Coordinator

Enclosure

Mr. Dean Anson
Anson Environmental Ltd.
256 Main Street
Northport, New York 11768

Re: Anchor Chemical Superfund Site; Second Round of RI Sampling; Format for Submission of RI Data

The purpose of this letter is to provide you with the U.S. Environmental Protection Agency's (EPA) determination on the specific analytes which should be tested for in the second round of sampling. As you know, the Anchor Chemical Superfund Site remedial investigation/feasibility (RI/FS) workplan requires that in order for an entire category of analytes to be excluded from the second round of sampling, no analyte from that category should have been detected in the first round of groundwater or soil samples.

Based on a review of your February 4, 1992, July 24, 1992 and August 20, 1992, submissions of groundwater and soil sample data, only polychlorinated biphenyls can be excluded from the second round of groundwater sampling. All other target compound list (TCL) compounds must be analyzed. EPA's determination is also based on the September 29, 1992, telephone conversation between Tom Taccone and Fritzi Mazzola, of your staff. Ms. Mazzola confirmed during the conversation that a non-detect was reported in the above-referenced data submissions as a blank entry. When submitting the final set of RI data for the Site, please use the appropriate data qualifier for a non-detect, rather than a blank.

As required by Paragraph 43 of the March 31, 1992, Administrative Order (No.II CERCLA-20205), issued to Anchor/Lith-Kem Ko, Inc., please give EPA seven business days notice before starting any field work for the second sampling round.

In addition, attached you will find format specifications for the RI sample data. Please use these specifications when compiling the data for the first and second rounds of data. The database software specified is required in order to run the risk assessment calculations for the Site. Also, please submit a computer disk of the sample data along with the paper copy.

SYMBOL --->	WNYCSII	ENYCSII	NYCSBII					
SURNAME -->	TACCONE	HAUPTMAN	PETERSEN					
DATE ----->	Taccone 9/30/92	MH 9/30/92	9/30/92					

If there are any questions on this matter, please contact Mr. Taccone at (212) 264-9128.

Sincerely yours,

Carole Petersen, Chief
NY/Caribbean Superfund Branch II

Attachment

cc: J. Doyle, ORC-NYCSUP
    J. Greco, NYSDEC
    D. Sullivan, TRC

RISK ASSESSMENT DATA FORMAT REQUIREMENTS

In general, TRC requires validated data in either DBASE III or ASCII format. LOTUS or other file formats are more difficult and less cost effective for us. We would prefer to avoid handling LOTUS files if at all possible.

The following are general requirements that TRC has for the receipt of data.

1. There should be one DBASE (DBF) file or ASCII file for each chemical class:
    - volatiles
    - semi-volatiles
    - pesticides/PCBs
    - inorganics

2. Each record in the DBF or ASCII file should contain (at a minimum) the following information:
    - sample number
    - sample date (optional if <u>every</u> sample number is unique)
    - result (detected concentration or detection limit [e.g., CRQL] for non-detects)
    - qualifier (incorporates the results of validation)
    - chemical name
    - units

3. All computerized data must be validated such that rejected ("R") and estimated ("J") values are indicated in addition to non-detects ("U").

4. All results should be entered, i.e., all "hits" as well as chemical- and sample-specific detection limits for non-detects.

5. All ASCII files should be in fixed-column format (spaces preserved for missing data). A key to field width, start/stop columns, and content must be provided.

There should only be one result and qualifier field per record. In other words, the "result" field should contain either the detected concentration or the detection limit for non-detects. The qualifier field should incorporate the laboratory qualifier field plus any changes/additions from data validation.

In general, duplicates, re-extracts, and dilutions should be included in the data base. A list should accompany the data base to clearly identify these samples and provide guidance on which sample (re-extract/dilution or initial analysis) should be used.

Please provide a written sample inventory to accompany the data base. This list should identify all samples in the data base, medium, depth/screened interval, location, and analyses conducted. Blanks and other QA/QC should also be clearly identified. Such a list is invaluable for ensuring the integrity of the data base and will save us all time in the long run.

Anson Environmental, Ltd.

33 Gerard Street
Suite 100
Huntington, NY 11743

Fax Transmittal

Date: 30 apr 93

Number of pages (including cover page): 2

To: Dorothy Allen, USEPA

From: Dean Anson, Anson Environmental

Regarding: Anchor Chemical

Please contact us immediately if transmission is incomplete.
516-351-3555

Comments:

PLEASE NOTE OUR NEW
FAX #
(516) 351-3615

ANS N
ENVIRONMENTAL LTD.

33 Gerard Street • Suite 100
Huntington, New York 11743
516•351•3555
Fax 516•351•3615

April 28, 1993

Dorothy Allen, Project Manager
United States Environmental Protection Agency
26 Federal Plaza
New York, NY 10278

Re: Anchor Chemical Superfund Site
Administrative Order No. II CERCLA-90208

Dear Mrs. Allen,

The 120 drums of soil cuttings at the above referenced site have been scheduled for removal on May 10th and 11th. We trust this notification will arrive in time for scheduling a member of the Alliance Technologies staff. The appropriate New York State agencies have been notified. Appropriate waste manifests will be provided for the documentation of the disposal of the drums and soil.

In addition, Jon Greco, of the NYSDEC, suggested that we obtain your recommendation on the disposal of the drummed personal protective equipment.

We trust this information is satisfactory for your purposes.

Very truly yours,

Dean Anson

Dean Anson II
Co-Facility Coordinator

cc: Fred Werfel, Spiegel Associates

• Groundwater Remediation • Hazardous Waste Investigation • Site Investigation and Remediation •
• Asbestos Management • Wetland Investigation •

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ENVIRONMENTAL LTD.

33 Gerard Street • Suite 100
Huntington, New York 11743
516•351•3555
Fax 516•351•3615

June 21, 1993

Thomas Taccone
United States Environmental Protection Agency
26 Federal Plaza
New York, NY 10278

Re: Direction of Groundwater Flow
Anchor Chemical Superfund Site
Administrative Order No. II CERCLA-90208

Dear Mr. Taccone:

In response to your recent inquiry, enclosed are the depth to water (DTW) measurements in all wells taken on November 9, 1992 at the above referenced site. The measurements are listed below along with the surface elevations of the wells.

Well #	**DTW**	**Surface Elevation of Well**
MW1S	not used in calculation	137.93 feet
MW2	59.05 feet	136.89 feet
MW3	59.55 feet	137.29 feet
MW4	60.90 feet	138.58 feet
MW5S	57.41 feet	135.19 feet
MW6S	60.42 feet	138.88 feet
MW7S	56.75 feet	134.62 feet
MW1D	60.43 feet	138.34 feet
MW5D	57.66 feet	135.42 feet
MW6D	60.61 feet	138.61 feet
MW7D	56.75 feet	134.20 feet

The monitoring well location plan prepared by Albert W. Tay L.S. is included as Figure 3-2 in the remedial investigation report.

Figure 2-8 identifies the direction of groundwater flow in the vicinity of the Anchor Chemical Site to be south-southwest and was prepared by Dvirka and Bartilucci (Investigation of Contaminated Aquifer Segments Nassau County, NY, 1986).

If you have any questions, please call me.

Very truly yours,

Dean Anson

Dean Anson II
Co-Facility Coordinator

cc: F. Werfel, Spiegel Associates
A. Sanders, Spiegel Associates
R. Leland, Esq., Rosenman & Colin
D. Sullivan, Alliance Technologies Corp.
J. Doyle, Esq., USEPA, Region II
S. Sucharski, BB & L

ANSON
ENVIRONMENTAL LTD.

33 Gerard Street • Suite 100
Huntington, New York 11743
516•351•3555
Fax 516•351•3615

July 13, 1993

Thomas Taccone
United States Environmental Protection Agency
26 Federal Plaza
New York, NY 10278

Re: Direction of Groundwater Flow
Anchor Chemical Superfund Site
Administrative Order No. II CERCLA-90208

Dear Mr. Taccone:

As a result of our meeting at the above referenced site on July 7th, we have prepared the following information to respond to the issues raised by yourself and Mr. Hauptmann.

The groundwater measurements and surface elevations are as follows:

Well #	**Surface Elevation of Well**	**DTW (4/92)**	**(11/92)**
MW1S	137.93 feet	not used	61.19 feet
MW2	136.89 feet	59.05 feet	60.25 feet
MW3	137.29 feet	59.55 feet	60.73 feet
MW4	138.58 feet	60.90 feet	62.25 feet
MW5S	135.19 feet	57.41 feet	58.60 feet
MW6S	138.88 feet	60.42 feet	61.61 feet
MW7S	134.62 feet	56.75 feet	
MW1D	138.34 feet	60.43 feet	
MW5D	135.42 feet	57.66 feet	58.88 feet
MW6D	138.61 feet	60.61 feet	61.79 feet
MW7D	134.20 feet	56.75 feet	57.61 feet

• Groundwater Remediation • Hazardous Waste Investigation • Site Investigation and Remediation •
• Asbestos Management • Wetland Investigation •

In order to illustrate the implications of the depth to water measurements above, we have prepared a groundwater contour map for each measurement date-April and November, 1992. You will note from the maps that the groundwater flows generally southwest across the site.

This is confirmed by Figure 2-8 which identifies the direction of groundwater flow in the vicinity of the Anchor Chemical Site to be south -southwest and was prepared by Dvirka and Bartilucci (Investigation of Contaminated Aquifer Segments Nassau County, NY, 1986). The Phase II investigation of the MEK Spill at 530 West John Street which was conducted by Roy F. Weston Inc. for the USEPA indicates that the groundwater flows in a southerly direction (3.2 Site Geology section).

The depth to groundwater measurements were taken with an auditory water level indicator, Solinst Model No. 121. The measurements were taken to the rim of the protective manhole casing not the top of the well itself.

We have also enclosed, as you requested, the detail drawing of the drywell construction which was provided by Spiegel Associates, property manager for the site.

If you have any questions, please call me.

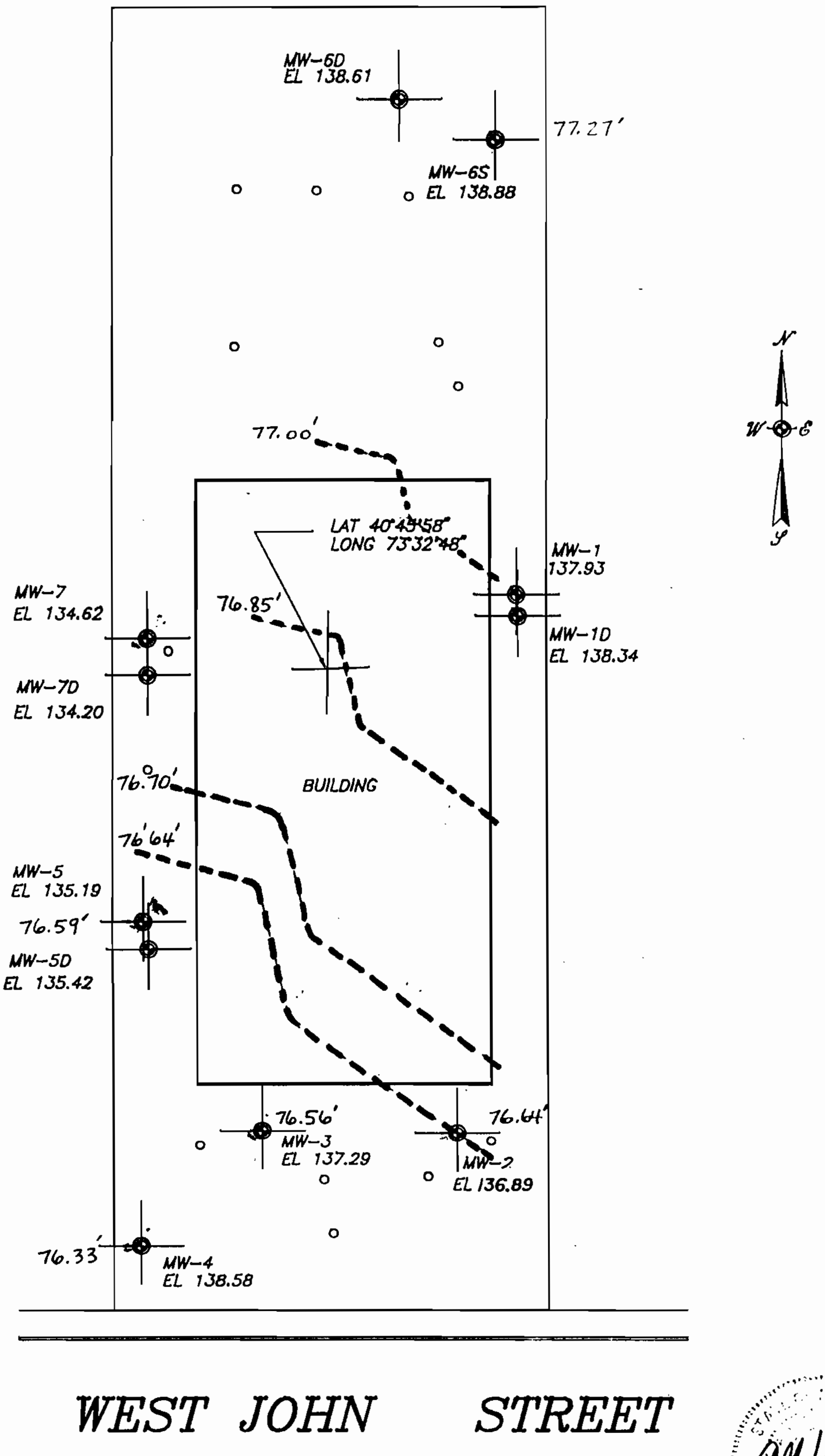
Very truly yours,

Dean Anson

Dean Anson II
Co-Facility Coordinator

cc: J. Greco, NYSDEC, Albany
F. Werfel, Spiegel Associates
A. Sanders, Spiegel Associates
R. Leland, Esq., Rosenman & Colin
D. Sullivan, Alliance Technologies Corp.
J. Doyle, Esq., USEPA, Region II
S. Sucharski, BB & L

11/92



MONITORING WELL LOCATION PLAN
HICKSVILLE, NEW YORK

N.C.T.M. SECT II BLK 499 LOT 87

SURVEYED APRIL 21, 1992

40 20 0 40
SCALE 1" = 40'

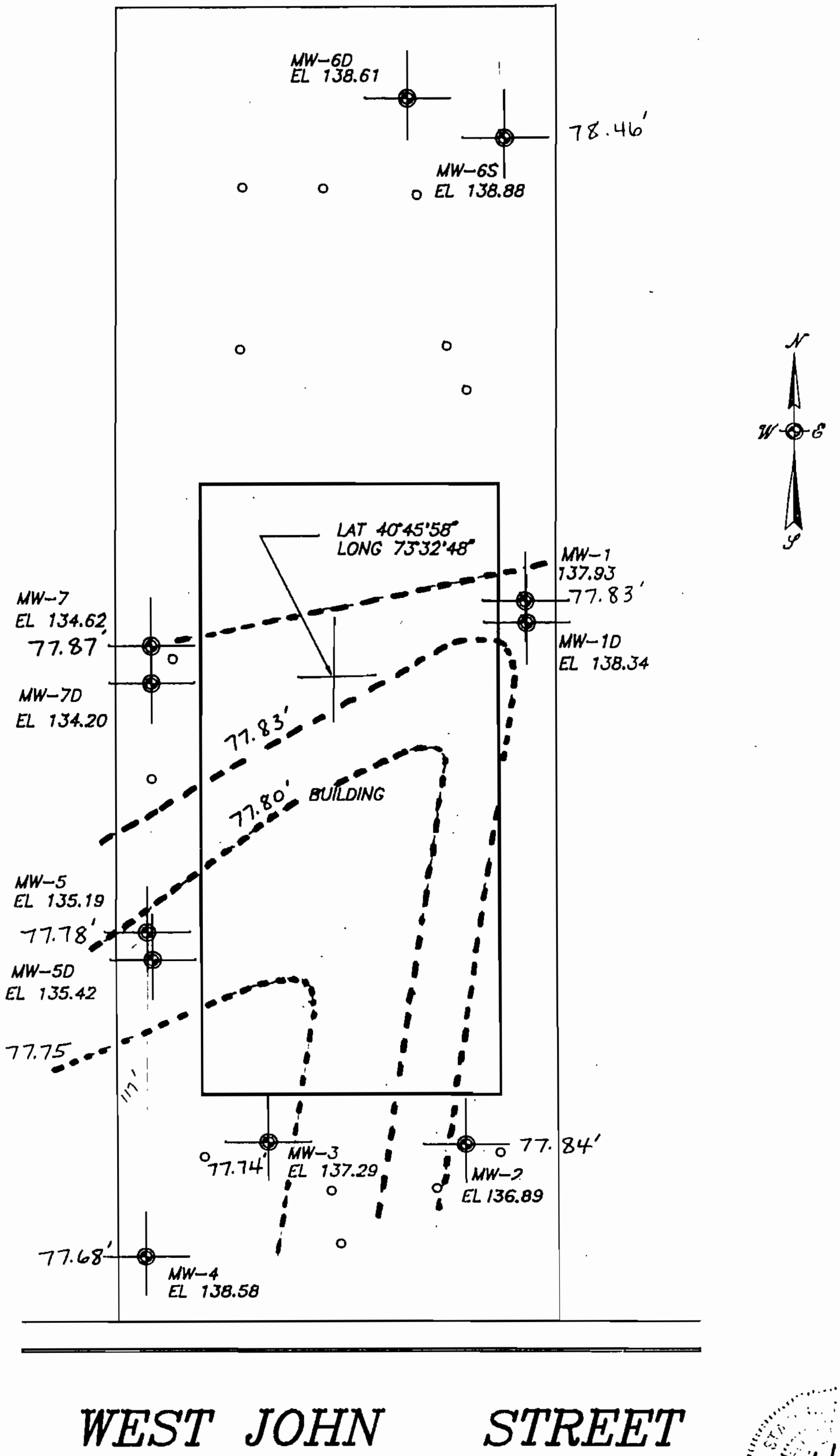
Albert W. Tay

ALBERT W. TAY
LICENSED LAND SURVEYOR
P.O. BOX 312, PLAINVIEW, NY 11803
TEL (516) 433-3725 FAX (516) 433-0409

302714

92233

4142



MONITORING WELL LOCATION PLAN
HICKSVILLE, NEW YORK

N.C.T.M. SECT II BLK 499 LOT 87

SURVEYED APRIL 21, 1992

40 20 0 40
SCALE 1" = 40'

ALBERT W. TAY
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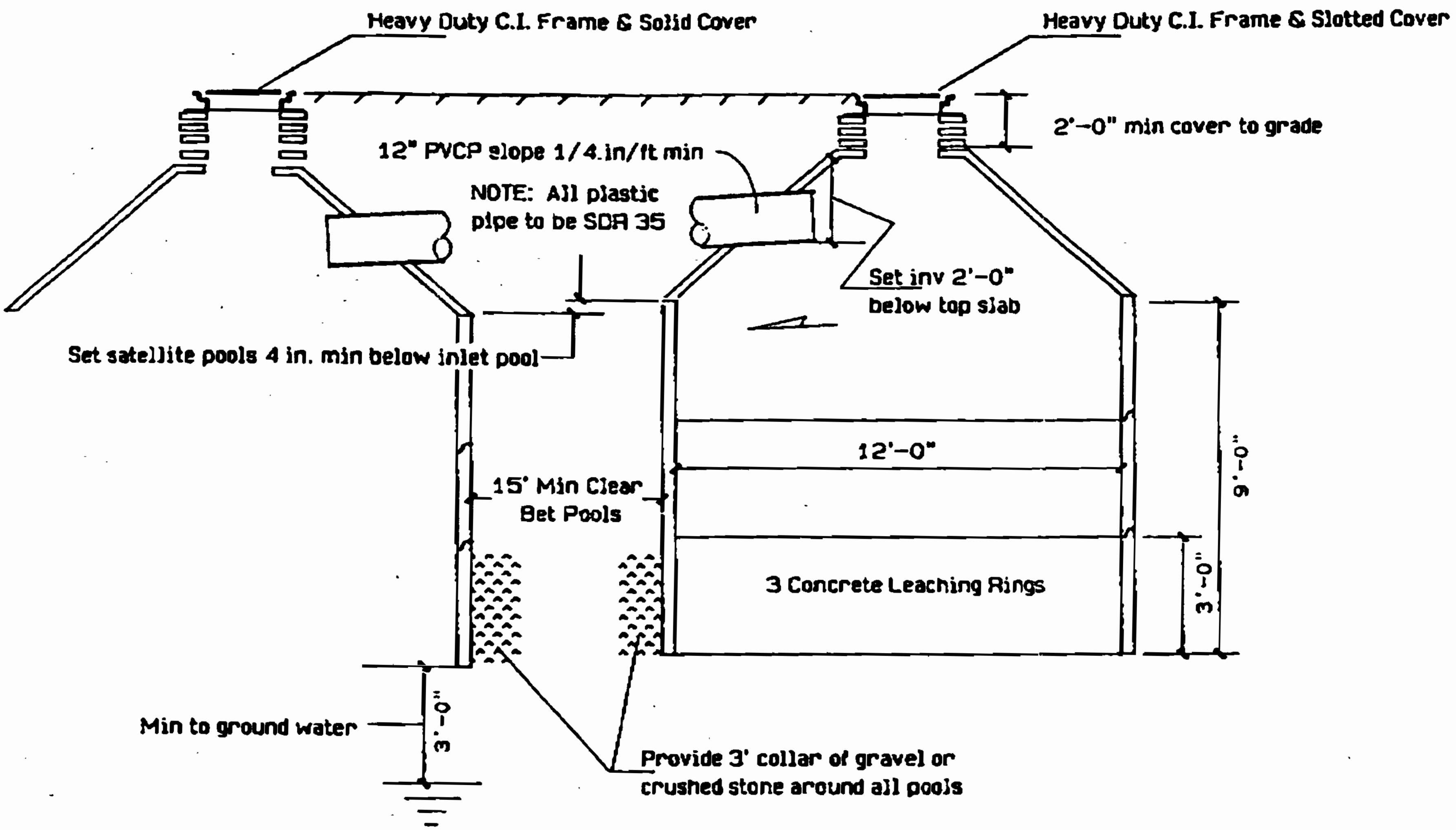
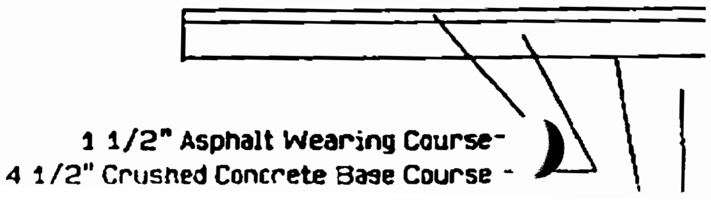
302715

92233



Typical Leaching Pool Detail

Scale 1/4'= 1'0"



AUG 5 1993

K.B. Company
c/o Jerry Speigel Associates
375 North Broadway
Gericho, New York 11753

Attention: Arthur D. Sanders, President
Jerry Speigel Associates

Richard G. Leland, Esq.
Rosenman and Colin
575 Madison Avenue
New York, New York 10022-2585

Dean Anson
Anson Environmental, Ltd.
33 Gerard Street, Suite 100
Huntington, New York 11743

Re: EPA Comments on the Draft Remedial Investigation Report for the Anchor Chemical Superfund Site

Dear [illegible] Sirs:

The U.S. Environmental Protection Agency (EPA) has completed its review of the draft Remedial Investigation (RI) report for the Anchor Chemical Superfund Site. EPA is concerned over the poor quality of the report, which needs major revision. Significant gaps in the report's content made a thorough review difficult. A much improved RI report needs to submitted in response to the comments below. If this does not happen, EPA will consider the imposition of penalties and may complete the report in-house.

<u>Overall Comments</u>

The draft report's Table of Contents should be revised to include page numbers for all sections, subsections, maps, figures, tables (please label all tables) and appendices. Volume 2 must be reprinted so that **all** sample results, compound names and sample locations are legible. Also, Volumes 2 and 3 should have tabs for each of the various sections.

SYMBOL --->	WNYSII	WNYSII	NY/CSBII					
SURNAME -->	Taccone	Lynch	Petersen					
DATE ----->	[illegible] 8/2/93	[illegible]	[illegible]					

Several tables and figures throughout the draft RI report lack pertinent information. Specifically, there are figures that need labels on particular items (i.e., cesspools, dry wells, boring logs), complete legends, and scales (fig. 1-3, 1-5, 2-2, 2-3, 2-6, 3-1, 3-2). A site map, that shows the location of the site within the Long Island region, is necessary. The site location and names of the geologic formations noted in the text should be included within the geologic cross-section (fig. 2-2) and the plan view (fig. 2-3) of Long Island.

A detailed map of the site (1"=10') is needed which shows the locations of the tanks and associated piping, sampling locations and levels of any detected compounds in the soils.

Potential chemical-specific ARARs need to be included in a table within the draft RI for comparison to site levels. These ARARs include such regulations as the Safe Drinking Water Act (SDWA) Maximum Contaminant Levels (MCLs) and New York State Department of Health MCLs.

Geological cross sections, which are based on the boring logs, should be included and discussed.

Specific Comments

Section 1.2.3 Previous Investigations

Page 1-11 - It is stated that ground water analyses show a decrease in the concentration of contaminants (Table 1-2), yet well No. 3 actually shows a slight rise after major decreases in (1,1,1-trichloroethane) over the past 3 sampling episodes. Possible explanations for this occurrence should be included in the text.

Page 1-11, first paragraph - Discussion indicates that a "liquid" sample from a drywell north of the building was taken in 1977. Revise this to be more specific. Was the liquid stormwater? What drywell was sampled?

Page 1-11 - Assuming that the above referenced drywell is drywell 2, previous investigations revealed elevated levels of 1,1,1-trichloroethane, trichloroethylene and tetrachloroethylene, yet the most recent sampling episode (1991) revealed only 1,1,1-trichloroethane in concentrations exceeding the 1977 sampling results. Besides biodegradation, explanations should be included as to why trichloroethylene and tetrachloroethylene were not found in the latest sampling events.

Page 1-12 - A brief description of the terms "textile, naphthol spirits and mineral spirits" should be included.

Page 1-12 - Footnote to table on page indicates that 1,1,1-trichloroethane was stored in tank 8 in 1965 and 1975. Was this solvent stored as a component of mineral spirits or was it the sole component stored? Table should be clarified.

Page 1-14 - First paragraph of page states that ground water measurements by LKB indicated a ground water flow of 0.45 feet per day. No direction of flow is given. This information should be included.

Section 2.4 Hydrogeology

Some discussion should be added to the text regarding the possible effects of the pumping of nearby supply wells on the site's hydrology as well as the effect of the nearby recharge basin.

Section 2.7 Drinking Water Supply Wells

Page 2-10 - Rather than referencing past conversations, the report should refer to documents which indicate that there are no private residential wells within two miles of the site. If possible, a map (with a scale) of residential wells should be included.

Section 2.7.2 Downgradient Water Supply Wells and Monitoring Wells

Page 2-17 - Monitoring wells WH-2 and WH-3 reveal increasing concentrations of total volatile organics from December 1984 until December 1985. Data for other downgradient wells, especially N8880 and N9341, also show significant contaminant levels, but the results for only one sampling event are referenced. If available, more recent data for these wells or their equivalents should be included in the report to demonstrate concentrations have increased, decreased or stabilized over time.

The dates of water level measurements shown on figure 2-4 and the names of tapped aquifers should be included in a table. The dates of the water level measurements should also be stated in the map's title block.

Section 3.2 Contaminant Source investigation

Discussion on pages 3-3 and 3-4 should be revised to reference Figure 3-3. Figure 3-3 should be revised to show the exact, not probable, locations of the six soil borings. Another figure is needed to show the exact locations of the cesspool and drywell samples. In addition, diagrams should be included which show the specific structures and dimensions of the cesspool and drywells sampled.

Section 3.3 Geological Investigation

Page 3-9 - Second paragraph appears to indicate that well redevelopment incorrectly occurred before each ground water sample was taken. If this did occur, the groundwater will have to be resampled. Please include a table, which provides for each round of samples and each well, the dates of well development and sampling. In addition, any necessary clarifications to the report must also be made.

Pages 3-10 and 3-11 - Report indicates that the water, which was left over from the groundwater sampling activity, was discharged into drywell 2. In actuality, the water was discharged to a publicly owned treatment works. Please correct.

Section 3.5 Underground Storage Tanks Investigation

Page 3-12 - Footnote to table on page indicates that 1,1,1-trichloroethane was stored in tank 8 in 1965 and 1975. Was this solvent stored as a component of mineral spirits or was it the sole component stored? Table should be clarified.

Section 4.0 General

Section 4 describes the analytical results for the sampling performed during the RI. However, except for the tabulated results at the end of the section, there is no mention of the high levels of lead and chromium found in the groundwater samples from monitoring wells 1D, 1S, 2, 3 and 5S. Please expand this section to discuss the elevated levels.

The report should state the rational and procedure for the selection of soil sampling depths for each boring. In Volume 2, Appendix A there are significant discrepancies in the boring logs between the organic vapor concentrations reported under the "OVM" column and the "sample description" columns. These discrepancies should be explained. Also, did the samples selected for laboratory analysis exhibit the highest field OVA concentration of volatile organics? If so, this should be stated.

Section 4.1 Drywell, Drain and Cesspool Analysis

Revise Tables 4-1 and 4-5 to specify the dates of sample analyses.

Pages 4-3 and 4-6 - Discussion on metals indicates that the metals, which were detected in the drywells and drain, also occur naturally. However, the source cited, EPA 1984 and Gallo 1990, cites metal concentrations for the eastern United States, which is too large of an area from which to derive meaningful background concentrations for the site. Discussion is needed on the metals which exceed the site background concentration levels found in the soil samples from the installation of upgradient monitoring wells 6D and 6S, and the areas on site where the elevated concentrations were found. For example, lead was detected at 1620 ppm in drywell 8, and chromium and cyanide were detected at 463 ppm and 430 ppm, respectively, in drywell 2. This is particularly significant since lead and chromium were also detected in the groundwater.

Pages 4-4 and 4-5 - As indicated in the report, both cesspools were sampled and the samples were lost during shipment to the laboratory. However, despite the fact that a split sample was taken, both cesspools should be resampled. The samples will confirm the results of EPA's split sample and answer any remaining questions on potential contaminant levels for the cesspool with no data. Completion of the RI report should not be held up by the collection and analysis of the samples. EPA believes that data for both cesspools will allow for a more defensible decision on site remediation.

Section 4.2 Tank investigation and Soil Borings Inside the Building

Pages 4-6 and 4-7 (also pages 4-10, 5-9, 6-3) - The rubber gloves which were worn by the sampling personnel were suggested as a probable source of bis (2-ethylhexyl) phthalate in the soil samples. This is not consistent with the RI/FS workplan and POP, which require that neoprene gloves be used. Neoprene does not contain phthalates. Discussion should address this discrepancy and the source of contamination.

The discussion of the inside boring program is incomplete. No mention is made of the extraordinarily high OVA readings nor of the levels of 2-butoxyethanol observed.

Section 4.3 Monitoring Well Analysis

Pages 4-7 and 4-9 - Revise the discussion to state that the underground storage tanks on-site also may be a source of methylene chloride and acetone contamination. Both compounds were stored in tanks, which according to Table 1-1 of the draft RI report, failed the tank tightness test. Moreover, the second round of groundwater sample results for MW 1S was reported to be 150 ppb of acetone, which is more than ten times the concentration detected in the field blank (10 ppb). EPA Region II's QA/QC guidance rules out the possibility of lab contamination if a compound's sample results are more than ten times the level detected in a blank.

Page 4-8 - Last paragraph states that no pesticides were detected in the groundwater. Heptachlor epoxide was detected in the sample for MW-1S at 0.076 ppb. Please eliminate this statement.

Page 4-8 - Please include some explanation on the possible origin of 1,4 dioxane (may be a breakdown product), which was detected in the first round of groundwater samples at 110 ppb, which is above the MCL of 50 ppb.

Section 4.4 In-Situ Specific Capacity Tests

Page 4-11 - Change "specific gravity" to "specific capacity."

Page 4-12, first full paragraph - Figure 3-2 is referred to as being a graphic depiction of the pumping tests. However, figure is a survey map. Please correct.

More discussion is needed on what the results of the pumping tests mean to the remedial investigation.

Section 4.5 Topographic Survey and Water Contours

Dean Anson's letter of June 21, 1993, which transmitted the groundwater elevation data and associated ground water flow maps, indicates a ground water flow to the south with flow components to the southeast and southwest. Given the relatively flat potentiometric surface across the site and the variability in flow direction at neighboring sites, a third round of ground water measurements is needed. Tom Taccone, of my staff, will be in touch with Mr. Anson to schedule a date for the measurements. A ground water flow map of the site using data from the third round should be submitted within one week of the measurements.

Page 4-13 - The report must include a table of the groundwater elevations for the two rounds of groundwater level measurements. A potentiometric map must be drawn for each round and included in the report.

Table 4-6 - Do the terms "decommissioned" and "filled with concrete" mean the same thing? If not, decommissioned should be explained.

Table 4-7 - Tank 16 Analysis - Barium has a reported concentration of 170 ppm. However, the analytical results reported in Appendix F show a concentration of 0.02 ppm. Please correct and update the text as appropriate.

Please include units of concentration for values reported in Tables 4-3, 4-8, 4-9 and 4-10.

Section 5.1 Potential Routes of Migration

Page 5-3, paragraph 2 - Site specific information is provided on permeability and porosity. Further explanation is needed on how the magnitude of these parameters affected the migration of site contaminants.

Page 5-4 - Delete the first full sentence. One cannot conclude that minimal levels of soil contamination at the time of sampling, meant that the groundwater was never affected.

Page 5-4, first paragraph - Change "As a result" to "At this time."

Page 5-4, last paragraph - Benzene was not detected in the sample for drywell 2. If a diesel spill did occur, one would expect some benzene to be present. Discussion should be revised.

Section 5.1.2 Drywells Cesspools and Drain Sediments

Page 5-4, last sentence - Report indicates that there is no continuity of contamination between the soil at 27 feet and the groundwater. What is this statement based on ? If the statement can not be supported by data, it should be deleted.

Page 5-5, last paragraph of section - The statement is made that the compounds detected in drywell 2 were not detected in any of the other drywells. This is not true. Levels of toluene were also detected in sediments from drywells 4, 6 and 8.

Section 5.2 Chemical Persistence

This section should be either eliminated or rewritten and made specific to the contaminants found on-site.

Section 5.2.1 Contaminant Persistence in Vadose Zone

As discussed above, the source cited, EPA 1984 and Gallo 1990, cites metal concentrations for the eastern United States, which is too large of an area from which to derive meaningful background data for the site. Discussion is needed on the metals which exceed the site background concentration levels found in the soil samples from the installation of upgradient monitoring wells 6D and 6S, and the areas on site where the elevated concentrations were found.

Section 5.3.1 Vadose Zone

Page 5-9, first paragraph - Revise to provide supporting information on why the pesticides identified are immobile and have a persistence which is greater than 1 year.

Drywells, Drain and Cesspool Sediments

This section contends that the site contaminants will be degraded to carbon dioxide and water. Eliminate this statement. This is an assumption which until supported by field data cannot be made.

Page 5-8, first paragraph - Please reference the data which supports the statement that "the cesspools are not a future source of chemical compounds."

Section 5.3.2 Saturated Zone

On page 5-11, the second paragraph states "Table 5-1 illustrates the levels of metals found in the groundwater in the vicinity of the site." Table 5-1, however, which is entitled "Common range of metals in natural soils," does not relate to the immediate vicinity of the site. After Table 5-1, is Table 5-2 which is a summary of ground water data from Westbury, Long Island. Again, sample results for upgradient wells MW 6S and MW 6D should be used for background concentrations. It is unclear why data from Westbury, L.I. has been included. Please remove all discussion relating to Table 5-2 and revise the report to compare on-site values with the values for the upgradient wells.

Section 6.0 Summary

Section 6.0 lacks overall consistency. Much of the text is devoted to stating that the site poses no threat to the ground water, however, remediation is proposed for several drywells. Please revise text.

Eliminate the last paragraph of Section 6.0, on page 6-2, which attempts to state the purpose of the RI. Replace it with the purpose as stated in the first sentence of Section 4 (page 21) of the approved RI/FS workplan.

Drywells, Cesspools and Drain

Page 6-3 - In addition to bis (2-ethylhexyl)phthalate, 2-butoxyethanol was also detected and was stored and used on-site. Please revise discussion to address both compounds.

Page 6-3 - Eliminate the sentence at the bottom of page 6-3, which states that the chemical compounds detected in the drywell, cesspools and drain do not pose a threat to the groundwater or public health. Any threats to public health will be evaluated in the risk assessment. This statement also seems to contradict the statement made on page 2-11 which indicates that the groundwater in Hicksville may be a threat to two water supply wells, downgradient from the site. It is also contradictory to the fact that several health based groundwater standards have been exceeded in the downgradient wells.

Section 6.1.1 Nature and Extent of Contamination

Drywells and Drain

Although found in small quantities, possible source areas for the pesticides (4,4'-DDE - 146 ppb, alpha BHC - 183 ppb) detected in drywell 2 should be given since a majority of the site is paved. If past agricultural uses are suspected, the approximate time frames of suspected use should be included in the operational history of the site.

Page 6-6, paragraph 2 - Eliminate the first sentence. This will be discussed in the risk assessment. The second statement, which indicates that the observed lead levels are typical of parking lots, needs to be substantiated with documentation and data.

Page 6-6, last paragraph - Benzene was not detected in the sample results. As indicated above, a diesel spill does not seem likely. Revise discussion.

Groundwater

Include in the discussion that lead and chromium were detected in the groundwater at elevated levels.

Section 6.1.2 Fate and Transport

Section 6.1.2 may discuss natural attenuation as a possible option for reduction of any remaining contaminants on site. Paragraph 2 on page 6-8, however, should be revised to eliminate the mention of any specific breakdown products of the contaminants. As mentioned above, the resulting effects of any degradation (biotic or abiotic) needs to be substantiated through an in depth evaluation.

Section 6.1.3 Recommended Remedial Action Objectives

Page 6-8, first paragraph - Eliminate the fact that sediments and soils will be excavated to a specific depth (18 feet) but add that excavation will be done to a depth which will be determined by an EPA approved soil clean up action level.

Page 6-9, top of the page - What is meant by "using bioremediation." Bioremediation as a remediation technology which involves some means of enhancing microbial degradation of contaminants by adding nutrients, oxygen or microorganisms. If natural attenuation is meant, it should be stated as such.

Page 6-9, second paragraph - Eliminate last sentence. The criterion to be used for determining the extent of removal will be determined by an EPA approved action level.

Page 6-9, last paragraph - Any determinations of whether soil/sediment contaminants are elevated will be based on EPA approved clean up levels, with input from the New York State Department of Environmental Conservation. Eliminate the last sentence of the paragraph.

The due date for a revised draft RI report will be dependent on the outcome of the third round of ground water measurements. If the third round confirms the previous two rounds, a revised draft should be submitted by September 10, 1993. If, however, inconsistencies are found, additional sampling may be needed and a schedule for developing a workplan for the sampling, the actual field work and a revised draft RI report, should be submitted by September 10, 1993. Finally, a date for when the two cesspool samples will be taken should be submitted by August 13, 1993.

Any questions on this matter, should be directed to Tom Taccone at (212) 264-9128.

Sincerely yours,

Carole Petersen, Chief
NY/Caribbean Superfund Branch II

cc: J. Doyle, ORC
J. Greco, NYSDEC

TRC *formerly Alliance Technologies Corporation*
TRC Environmental Corporation

291 Broadway, Suite 1206
New York, NY 10007
☎ (212) 349-4616
Fax (212) 349-4648

August 24, 1993

Thomas Taccone
Work Assignment Manager
U.S. Environmental Protection Agency
Emergency and Remedial Response Division
26 Federal Plaza
New York, NY 10278

Reference: Contract No. 68-W9-0003, TES6
Work Assignment No. C02026
Anchor Chemical Site
Compliance Oversight
(Ref. 1-635-108)

Subject: Ground Water Flow Direction and Sampling Needs

Dear Tom:

As requested, TRC has evaluated site-specific ground water flows regarding the Anchor Chemical site. TRC submitted a letter to EPA on July 14, 1993 that stated TRC was in agreement with a comment made by EPA. The comment eluded to the assumption that the ground water flow direction on site was southeasterly; however, after review of additional site-specific data TRC believes the flow is in a southwesterly direction. It is important to note that the ground water elevations across the site show little variance (approximately .1 feet). A contaminant plume emanating from the area of the building on site would have probably spread radially with a slight downgradient flow direction southwesterly. It is believed that given the monitoring well network location and sampling data, that any contaminants released from this site have migrated and dissipated into the ground water aquifer.

Due to the fact that monitoring wells are positioned in the southwesterly portion of the site, TRC no longer recommends the use of a hydropunch to obtain additional data. The above comments are a revision to statements made in TRC's letter to EPA dated July 14, 1993.

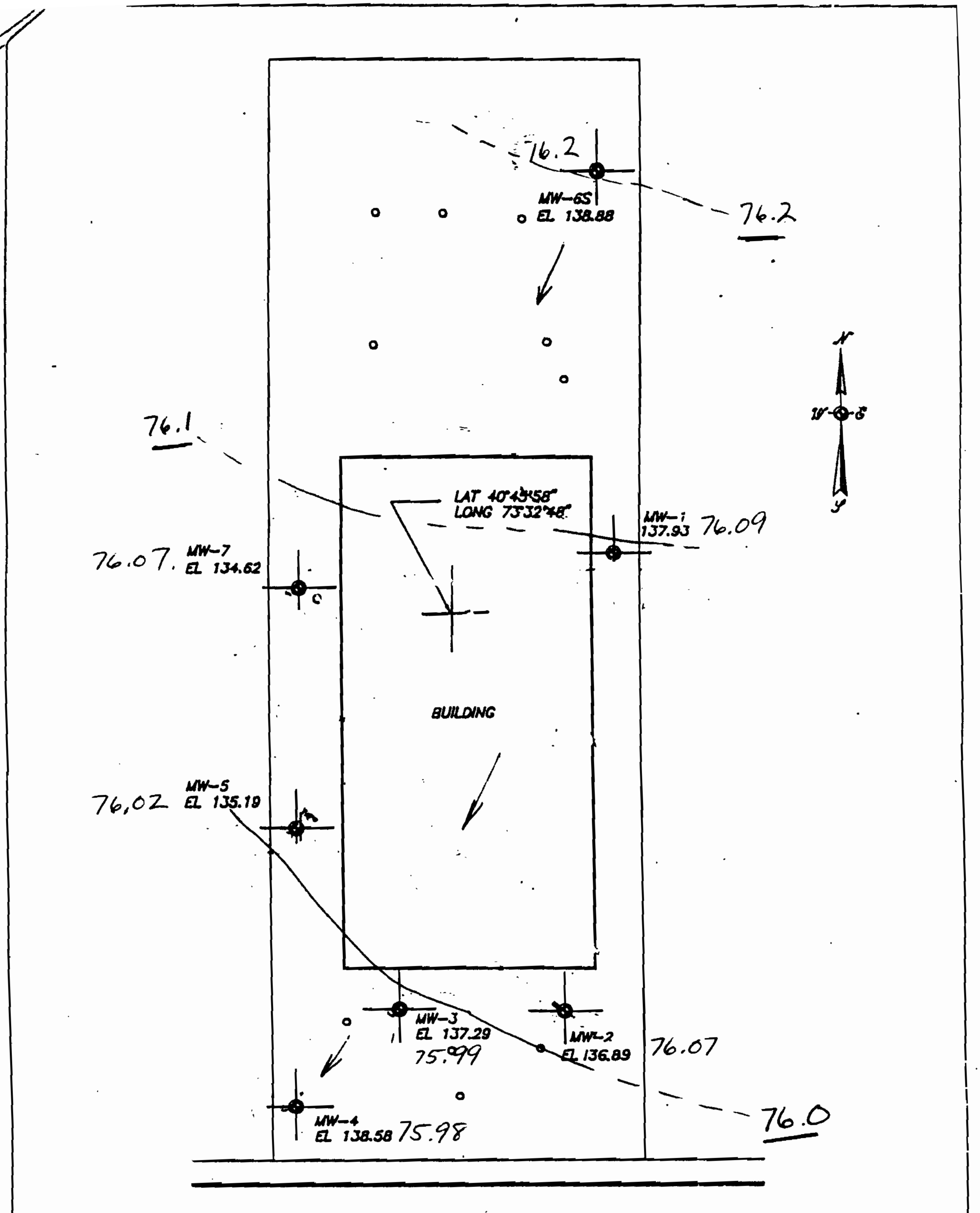
Attached is a figure showing ground water flow elevations for your use. The elevations shown were taken by TRC personnel on August 13, 1993.

If you have any questions or require additional information please call me at (212) 348-4616.

Sincerely,

for Andre Bridgett
Project Manager

cc: Erwin Smieszek/EPA Regional Project Officer
David Boyd/EPA TES-6 Contracting Officer (letter only)
Douglas Sullivan/TRC Regional Manager
TES ZPMO (letter only)



MONITORING WELL LOCATION PLAN
HICKSVILLE, NEW YORK
N.C.T.M. SECT 11 BLK 499 LOT 87

SURVEYED APRIL 21, 1992
40 20 0 40
SCALE 1" = 40'

ALBERT W. TAY
LICENSED LAND SURVEYOR
P.O. BOX 312, PLAINVIEW, NY 11803
TEL (516) 433-3725 FAX (516) 433-0409

Albert W. Tay

302729

92233

TRC
Environmental Solutions through Technology

TRC Environmental Corporation
Boott Mills South, Foot of John Street
Lowell, MA 01852
☎ (508) 970-5600

December 7, 1993

Mr. Thomas Taccone
Work Assignment Manager
U.S. Environmental Protection Agency
Emergency and Remedial Response Division
26 Federal Plaza
New York, NY 10278

Reference:	Contract Number 68-W9-0003, TES 6 Work Assignment Number C02026 Anchor Chemical, Hicksville, NY RI/FS Compliance Oversight Reference Number 1-635-108
Subject:	Clarification of Observed Field Sampling Procedures

Dear Mr. Taccone:

This letter addresses Anson Environmental's sampling procedures during the completion of six interior borings at the Anchor Chemical Superfund site in Hicksville, NY. The information contained herein is based upon my observations made more than two years ago, as gleaned from logbook entries covering three separate periods of on-site work. This work occurred on November 9 and 10, 1991 (borings IB-3 and IB-4); December 7 and 8, 1991 (borings IB-5 and IB-6); and January 4 and 5, 1992 (borings IB-1 and IB-2). It should be noted that, to the best of my knowledge, no approved sampling procedures for use at the Anchor site were presented by Anson in the Work Plan or other site-related documents.

Anson Environmental was expected to submit two soil samples from each of the six borings drilled with a skid rig inside the rear of the warehouse on the Anchor property; TRC collected split samples from three of these borings. Anson used the following procedures throughout the sampling of the interior borings. Soil volume was collected using a split spoon at five-foot intervals from the base of the abandoned underground storage tanks, approximately 10 feet below ground surface (bgs), to the water table, approximately 60 feet bgs. Adequate volume was collected for analysis for volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), pesticides/PCBs, and metals/cyanide, as well as a headspace sample.

Following the completion of each boring, it was my understanding that Anson sent for laboratory analysis the two samples from each boring measured to have the highest concentrations of organic vapors. These concentrations were based on two sets of photoionization detector (PID) readings: PID readings were obtained just below the surface of the soil in each split spoon at the time the spoon was opened, and readings were also taken on the headspace of dedicated jars filled at each five-foot depth at the time the volume was collected for potential analysis. PID readings were taken on the entire set of headspace jars from each boring at the completion of that boring. I believe the headspace jars were covered with aluminum foil and stored inside either a vehicle or building prior to taking the PID readings; however, these details could not be verified by logbook entries.

During my stint at the Anchor Chemical site in an oversight capacity, Anson placed soil samples collected and submitted for VOC analysis in VOA vials immediately upon opening each split spoon, and did not open the vials on site thereafter. In no case were these VOC samples used for headspace measurements, nor were they filled from sample material previously used in performing headspace measurements, with following exceptions.

At boring IB-2, only headspace volume was collected from 5-7 feet bgs, since the sampling plan did not specify sampling above the depth of the tank base. Following PID measurement of all headspace volumes collected from this hole, the sample collected from 5-7 feet was found to have the highest VOC concentrations. At this boring only, to submit sample volume from this depth at which elevated concentrations had been detected, VOA vials were filled from the headspace volume and submitted by Anson for analysis. Again, this is the only sample for which this alternate method was used.

In some cases, due to limited sample recovery, it was necessary to composite sample volume between two split spoons to collect volume sufficient for both the PRP and TRC samples. This was the case in boring IB-3. When this technique was used, sample volume for VOC analysis was collected first from the initial split spoon. Soil was then composited from the first split spoon as well as from a second split spoon and sampled for SVOC, pesticides/PCB, and metals/cyanide analysis. As a result, the total volume was collected over four feet of boring reach.

The only other sampling procedure deviation identified during my review of logbook entries is the collection of material for headspace measurements from composited soil; an Anson representative was observed using this method at IB-5. I instructed him to collect the headspace sample prior to compositing volume for extractable and metal/cyanide samples. In no way did the erroneous practice impact the integrity of samples collected and analyzed for VOCs. It may have resulted in somewhat lower headspace readings for IB-5; however, the field measurement and evaluation of PID readings at the split spoon as well as in headspace jars provided a means of confirming all sample depths selected. In most cases, these data were in agreement, indicating that headspace concentrations were not significantly altered based on the method used at IB-5.

Although there is no single logbook entry summarizing Anson's technique in its entirety, from opening split-spoons to shipping samples, the standard procedure outlined above is indirectly confirmed through several entries.

If any aspect of this letter is not clear, or if you have additional questions, please feel free to contact me at TRC's Lowell, MA office (508/970-5600 ext. 5267).

Sincerely,

Andrew G. Hargens

Andrew G. Hargens
TRC Geologist

cc: Douglas Sullivan/TRC Regional Manager
Andre Bridgett/TRC Project Manager
TES PMO

ANSON
ENVIRONMENTAL LTD.

33 Gerard Street • Suite 100
Huntington, New York 11743
516•351•3555
Fax 516•351•3615

February 15, 1994

Thomas Taccone, Project Manager
United States Environmental Protection Agency
26 Federal Plaza, Room 747
New York, NY 10278

Re: Anchor Chemical Superfund Site
Administrative Order No. II CERCLA-90208
Cesspool Sampling Locations

Dear Mr. Taccone:

As you requested, please find enclosed the locations of the cesspool samples which were taken in November, 1991 and September, 1993. We sampled in the same locations in 1993 but at adjacent depths to secure as similar samples as possible to the original samples taken in 1991.

We trust this information will be satisfactory for your purposes.

If you have any questions, please call me.

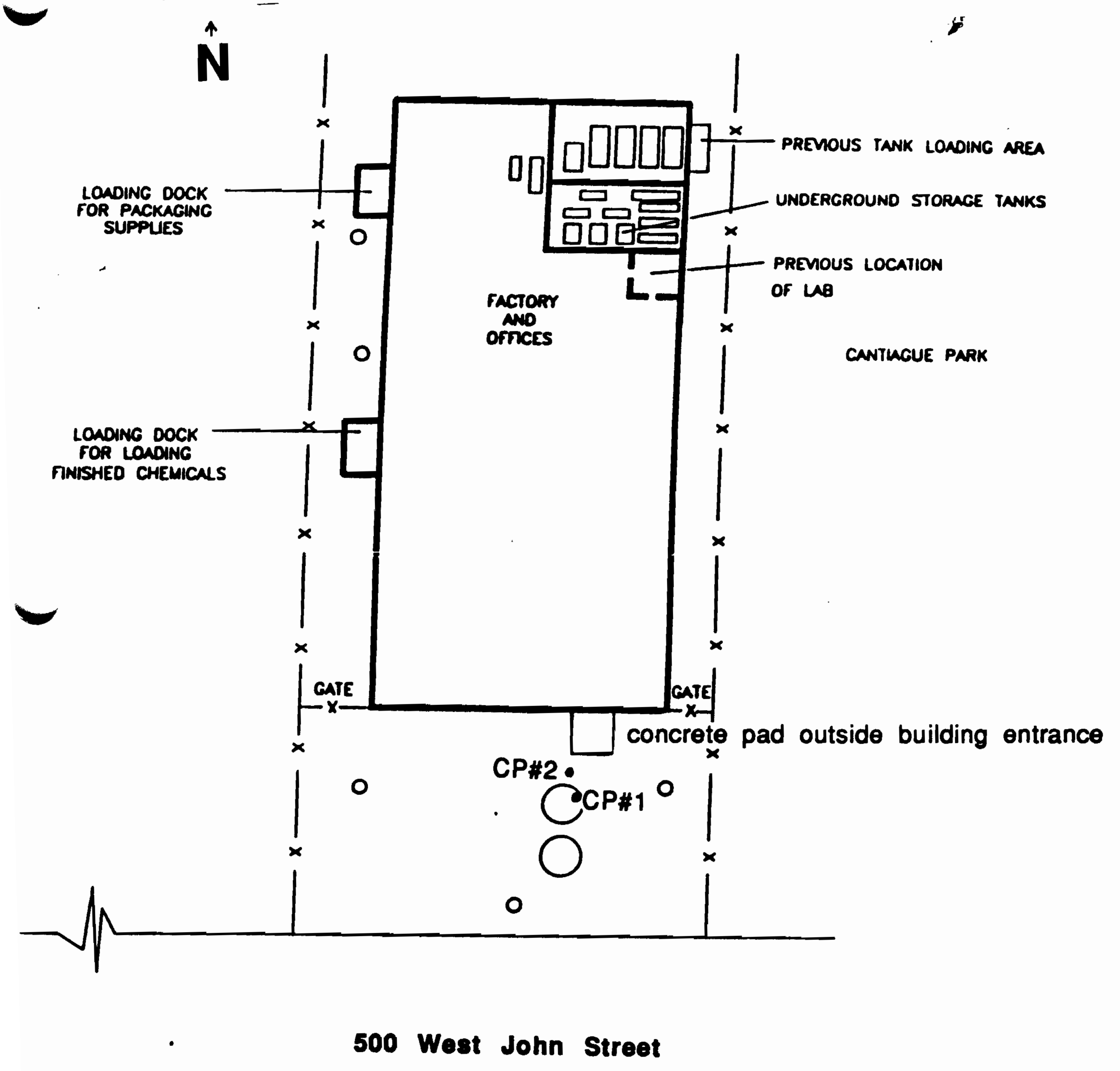
Very truly yours,

Dean Anson II

Dean Anson II
Co-Facility Coordinator

ANSON ENVIRONMENTAL LTD.

33 Gerard Street • Suite 100
Huntington, New York 11743
516•351•3555
Fax 516•351•3615



Cesspool Sampling Locations

CP#1 29' west of east side of bldg
22' south of southern side of bldg

CP#2 31' west of east side of bldg
19' south of southern side of bldg

TRC *formerly Alliance Technologies Corporation*
TRC Environmental Corporation

291 Broadway, Suite 1206
New York, NY 10007
☎ (212) 349-4616
Fax (212) 349-4648

April 5, 1994

Tom Taccone
EPA Work Assignment Manager
U.S. Environmental Protection Agency
Emergency and Remedial Response Division
26 Federal Plaza, Room 737
New York, NY 10278

Reference: Contract No. 68-W9-0003, TES 6
Work Assignment No. C02125
Anchor Chemical
Risk Assessment
(Ref. 1-635-354)

Subject: Tentatively Identified Compounds 1,4-Dioxane and 2-Butoxyethanol

Dear Tom,

As you requested on March 30, 1994, TRC evaluated the laboratory results for the tentatively identified compounds (TICs) 1,4-dioxane and 2-butoxyethanol reported in Volume 3, Validated Laboratory Data, of the Remedial Investigation Report for the Anchor Chemical site dated April 15, 1993 and prepared by Anson Environmental Ltd. This letter presents the results of that evaluation.

1,4-Dioxane was reported as a TIC at an estimated concentration of 110 ug/l in one sample collected from monitoring well #3 during the first ground water sampling round. It was not reported as detected in the second ground water sampling round. 2-Butoxyethanol was reported as a TIC in soil samples collected from boring locations #1 and #2. Estimated concentrations for 2-butoxyethanol as a volatile component were qualified as being based on "presumptive evidence" and ranged from 30 ug/kg to 100 ug/kg. It is important to note that the 2-butoxyethanol was also reported in the field blank at an estimated concentration of 200 ug/kg. Estimated concentrations for 2-butoxyethanol as a semivolatile component were also reported in one soil sample from boring location #2 at 2,100 ug/kg. Again, it is important to note that the field blank results show an estimated detected concentration of 6,400 ug/kg which exceeds all other reported concentrations. Estimated reported concentrations for 2-butoxyethanol as a semivolatile component were also qualified as being based on "presumptive evidence".

TICs were not included in the April 1, 1994 quantitative risk assessment for the Anchor Chemical site because their assigned

Offices in Alabama, California, Colorado, Connecticut, Illinois, Louisiana, Massachusetts, New Jersey, New York, North Carolina, Pennsylvania, Texas, Washington, Washington, D.C., and Puerto Rico
A TRC Company

identity is highly uncertain. In addition, the reported concentrations are only estimations, are highly questionable, and may be orders of magnitude higher or lower than actual concentrations. Due to the uncertainty associated with the reported concentrations for these compounds, risk estimates based on these results must be considered highly uncertain and significantly less meaningful than risk estimates based on positively identified compounds.

At your request, TRC performed an evaluation of 1,4-dioxane and 2-butoxyethanol using available EPA toxicity criteria listed in the National Library of Medicine's Integrated Risk Information System (IRIS) database. IRIS currently lists no toxicity information for 2-butoxyethanol except to state that an inhalation RfD for this compound is currently under review. As a result, TRC could not evaluate 2-butoxyethanol. IRIS lists 1,4-dioxane as a class B2 carcinogen and provides an oral slope factor of 1.1E-02 $(mg/kg/day)^{-1}$. Using this oral slope factor, the exposure factor for the carcinogenic ground water ingestion scenario used in the risk assessment (1.17E-02 l/kg/day), and a target risk of 1E-04, TRC calculated a health based preliminary remediation goal (PRG) of 0.769 mg/l for 1,4-dioxane as shown below:

$$\text{Preliminary Remediation Goal} = \frac{\text{Target Risk}}{\text{Exposure Factor} \times \text{Slope Factor}}$$

Detected concentrations of 1,4-dioxane below the calculated PRG 0.769 mg/l are not expected to pose an increased carcinogenic risk of greater than 1E-04 to potential human receptors. The reported concentration of 1,4-dioxane in site ground water, 0.110 mg/l, does not exceed this concentration; however, as stated previously, the identity and the reported concentrations of tentatively identified compounds are highly uncertain. 1,4-Dioxane may not be present, or it may be present at significantly higher or lower concentrations. The fact that 1,4-dioxane was reported in only one ground water sample and in only the first ground water sampling round indicates that it may not be present in site ground water at concentrations which could pose significant risk.

If you have any questions on the above, please do not hesitate to contact me at (508) 970-5600, extension 5271.

Sincerely,

for Susan W. Stoloff
Project Manager

cc: Erwin Smieszek/EPA Regional Project Manager
Doug Sullivan/TRC Regional Manager

MAY 06 1994

K.B. Company
c/o Jerry Speigel Associates
375 North Broadway
Gericho, New York 11753

Dean Anson
Anson Environmental
33 Gerard Street
Huntington, NY 11743

Attention: Arthur D. Sanders, President

Richard G. Leland, Esq.
Rosenman and Colin
575 Madison Avenue
New York, New York 10022-2585

S. Sucharski
Blasland, Bouck and Lee
1 Suffolk Square
Suite 210
Islandia, NY 11787

Re: EPA Comments on the Revised Draft Remedial Investigation Report for the Anchor Chemical Superfund Site

Dear Sirs:

The U.S. Environmental Protection Agency (EPA) has completed its review of the revised draft Remedial Investigation (RI) report, dated September 1993, for the Anchor Chemical Superfund Site. Several comments concerning the last draft of the RI report, which were provided to you in my letter of August 5, 1993, were not addressed in the resubmitted September draft. The revised report had no accompanying explanation of which comments were addressed, where modifications were made in response to those comments that you endeavored to address in the resubmitted report, nor any justifications for failing to address all the comments. If your response to this letter does not satisfactorily address the enumerated comments below, EPA may finalize the report, may also complete the remainder of the RI/FS, and may seek to recover those costs and any appropriate penalties, accordingly.

For ease of reference, enclosed is a copy of EPA's August 5, 1993 letter. You will note that each of the comments has been numbered. The comments below are also numbered to correspond to the comments as they are numbered in the enclosed August 5 comment letter.

Section 3.2 Contaminant Source investigation

Comment 11. Figure 3-3 was not revised to show the exact locations of the six soil borings. The figure must be revised to show actual, not probable locations. Also, another figure is needed to show the locations of the cesspool and drywell samples.

Finally, diagrams were requested to show the specific structures and dimensions of the cesspool and drywells sampled. On July 13, 1993, Dean Anson transmitted a diagram of a "Typical Leaching Pool." If, in fact, this diagram is a true representation (including dimensions) of both the cesspool and drywells on-site, it should be incorporated and referenced in the RI report.

Section 3.3 Geological Investigation

Comment 12. Pages 3-9 and 3-10 of the revised report discuss the methods used for well development. However, table 3-3 on page 3-22 does not make sense. On November 3, 1993, Anson Environmental transmitted a revised, corrected table. The revised table provides the needed information except for the development dates for wells 1S, 2 and 3. Instead of a providing a specific date, the term "prior to sampling" is used. Specific dates are needed for these wells also.

Section 4.0 General

1 New comment. The report does not adequately compare Site data with all applicable or relevant and appropriate requirements ("ARARs"). Groundwater data must be compared to both federal and New York state maximum contaminant levels (MCLs). Soil data cannot be compared to MCLs. MCLs are for contaminants in water only. Therefore, Table 4-7 must be revised or eliminated. In addition, another table is needed which compares the New York State Department of Environmental Conservation's (NYSDEC) soil clean up objectives (copy enclosed) with the Site soil and sediment data.

Comment 15. There is still no mention of the high levels of lead and chromium found in the groundwater samples from monitoring wells 1S, 1D, 2, 3, 4 and 5S. Please expand Section 4 to discuss the elevated levels.

Section 4.1 Drywell, Drain and Cesspool Analysis and

2 New comment. Page 4-2, top of page - The first full sentence states that "no volatile organic compounds were detected in drywells 1, 3, and 5..." However, toluene and methylene chloride were both detected at 1100 ppb in the sediment sample from drywell 3. Please revise the statement accordingly.

Comment 18. The report again, as in the last draft, compares the sediment sample results with data from soils in Hicksville, New York. As was stated in the previous comment letter, the background sample data from wells 6S and 6D should be used for this type of comparison. Please revise the report to address EPA's earlier comment.

Comment 19. Please discuss and incorporate into the RI report the results of the cesspool samples which were taken in September 1993.

Section 4.2 Tank investigation and Soil Borings Inside the Building

3. New comment. Page 4-8 - As a result of the detection of 2-butoxyethanol in the soil samples from indoor borings 1 and 2, additional soil samples are needed downgradient from tank 14, which stored the chemical. Two soil borings should be advanced 4 to 5 feet from the southeastern and southwestern corners of tank 14 and should be advanced and sampled using the same procedures which were used for indoor borings 1 through 6. Please submit a proposed date for the borings to Tom Taccone within 14 days of receipt of these comments.

4 New comment. Page 4-8 - The revised report incorrectly reports the concentrations of inorganics in parts per billion. The correct units are parts per million.

Comment 20. The revised report states that bis(2-ethylhexyl) phthalate is considered a laboratory contaminant. However, the data validation process did not reject or estimate all of the data. Therefore, the report should be revised to reflect that the compound is considered a Site contaminant. In addition, acetone and methylene chloride also should be recognized and indicated as soil contaminants.

Comment 21. The revised report still does not explain the significant discrepancy in the OVM headspace readings which were used for selecting soil samples for laboratory analysis and the OVM readings noted on the subsurface soil logs. This difference must be addressed. In addition, because neither the Site Remedial Investigation/Feasibility Study workplan nor the Project Operation Plan provided detailed procedures for selecting soil samples for laboratory analysis, procedures were developed in the field. Therefore, it is important that a more detailed description of the procedures used for selecting soil samples for laboratory analysis be provided in the report.

Section 4.3 Monitoring Well Installation and Sampling and Analysis - 2 Rounds

Comment 22. The report states that acetone, methylene chloride, and bis(2-ethylhexyl) phthalate are considered laboratory contaminants. However, the data validation process has not rejected or estimated all of the data. Therefore, the report should be revised to reflect that the compounds are considered Site contaminants.

5 New comment. Page 4-11, first full paragraph - The discussion on inorganics needs to be expanded. Please elaborate and explain which inorganics exceeded MCL limits. Also, identify the trends in the first and second rounds of samples.

6 New comment. As a result of the detection of 1,2 dioxane in monitoring well 3, a third round of groundwater samples should be collected from monitoring wells 2, 3 and 5S. The samples should be tested for the full target compound list (TCL) and be collected on the same date the soil samples at tank 14 are collected.

Section 4.4 In-Situ Specific Capacity Tests

Comment 26. Page 4-17 - On the top of the page there is a reference to figure 4-12 concerning a graphic presentation of groundwater draw down. However, the referenced figure should be figure 4-2, not figure 4-12. Please amend accordingly.

Section 5.1.2 Drywells, Cesspools and Drain Sediments

Comment 37. Page 5-7, second paragraph - The revised draft report maintains that there is no continuity of contamination between the soil at 27 feet and the groundwater at drywell 2. The draft report bases this conclusion on the OVA screening levels which were taken between 27 feet and the water table. EPA disagrees. There were still measurable OVA readings down to the water table, and no samples were taken to confirm that no VOCs are present. In addition, there may be semi-VOCs and inorganics present, which can not be detected by the OVA. Therefore, the report should not state that there is "no continuity of contamination between 27 feet and the water table."

Section 5.2.1 Contaminant Persistence in Vadose Zone

Comment 40. This section needs to be rewritten to reference the inorganics whicn were detected in the soils sampled from the installation of monitoring wells 6S and 6D. Data from soils in Hicksville, Long Island (Table 5-7) should not be used.

Drywells, Drain, and Cesspool Sediments

Comment 42. The revised report does not properly address EPA's comment. There are no field data which support the statement on page 5-11 that VOC and semi-VOC contaminants "will be broken down into less complex and nonhazardous organic compounds and ultimately into carbon dioxide and water." Unless or until supported by field data, this statement should be eliminated from the report.

Comment 43. Page 5-12, paragraph 2 - Revise this paragraph to reference the cesspool sample results and any data collected in accordance with requirements of the Nassau County Department of Health. In the statement, "the cesspools are not a future source of chemical compounds", replace "compounds" with "contamination", and the conclusion must be supported by Site data.

Section 5.3.2 Saturated Zone

Comment 44. As discussed above and in my letter of August 5, 1993, sample results from samples of upgradient wells MW 6S and MW 6D should be used for background concentrations. Data from Westbury, Long Island should not be referenced. Please remove any discussion relating to Table 5-8 and revise the report to compare on-site values with the values for the upgradient wells.

Section 6.1.1 Nature and Extent of Contamination
Drywells and Drain

Comment 50. Delete the first full paragraph on page 6-7 which states that lead is not a threat to human health.

Groundwater

Comment 52. The revised report does not include a discussion on lead and chromium, which were detected in the groundwater at elevated levels. Please revise the report to reference these results.

Also, delete the last sentence from paragraph 1 on page 6-8 of the revised report. Any discussion on a decision on proposed remedial measures is not appropriate for the RI report.

Section 6.1.2 Fate and Transport

Comment 53. Page 6-9, first paragraph - Delete "into nonhazardous organic compounds" from line 3.

Within 21 days of your receipt of this letter, please submit a description, for each comment above, of how the draft RI report will be revised. If the description adequately responds to EPA's comments, we will direct you to submit a revised draft report. If there are significant deficiencies, however, EPA will complete the report.

Any questions on this matter, should be directed to Tom Taccone at (212) 264-9128.

Sincerely yours,

Carole Petersen, Chief
NY/Caribbean Superfund Branch II

Enclosures

cc: J. Greco, NYSDEC
    C. Fines, CDM

bcc: J. Doyle, ORC

ROSENMAN & COLIN

575 MADISON AVENUE, NEW YORK, NY 10022-2585

TELEPHONE (212) 940-8800
CABLE ROCOKAY NEWYORK
TELECOPIER (212) 940-8776
(212) 935-0679
TELEX 427571 ROSCOL (ITT)
971520 RCFLC NYK (W. U.)

SAMUEL I. ROSENMAN (1896-1973)
RALPH F. COLIN (1900-1985)

WASHINGTON OFFICE
1300 19TH STREET, N. W
WASHINGTON, D. C. 20036
TELEPHONE (202) 463-7177

RICHARD G. LELAND
(212) 940-8700

May 12, 1994

Mr. Tom Taccone
Project Manager
United State Environmental Protection Agency
25 Federal Plaza, Room 747
New York, NY 10278

Re: EPA Comments on the Revised Draft Remedial Investigation Report for the Anchor Chemical Superfund Site

Dear Mr. Taccone:

As you know, we are counsel to K.B. Company. This letter will confirm our receipt on Tuesday, May 10, of your letter containing comments to the version of the RI study submitted in October 1993. We have spoken with Eric Schaaf to arrange a meeting to discuss these comments. Therefore, we assume that the fourteen day time limit for responses will be tolled until after that meeting.

Very truly yours,

Richard G. Leland/RLR

Richard G. Leland

RGL/rlr

cc: Mr. Dean Anson
James Doyle, Esq.
James O'Brien, Esq.
Mr. Arthur D. Sanders
Eric Schaaf, Esq.

ANSON
ENVIRONMENTAL LTD.

33 Gerard Street • Suite 100
Huntington, New York 11743
516•351•3555
Fax 516•351•3615

May 31, 1994

Thomas Taccone
US Environmental Protection Agency
Region II
Jacobs K. Javits Federal Building
New York, NY 10278-0012

Re: Responses to EPA May 6, 1994 Comments on the
Revised Draft Remedial Investigation Report
for the Anchor Chemical Superfund Site
Administrative Order No. II CERCLA-90208

Dear Sirs:

We have carefully reviewed the comments in your May 6, 1994, letter regarding the revised September 1993 draft Remedial Investigation (RI) report. We have catergorized the comments into three groups which are:

-Comments With Which We Agree
-Comments Dealing with the Usage of Data from Hickville
and Other Areas Offsite
-Comments With Which We Disagree

The May 6th comments are underlined and in one of the above categories along with our general response to each comment. When the RI is revised, each comment will be in additional detail with supporting documentation.

Comments With Which We Concur

Section 3.2 Contaminant Source Investigation - Comment 11 - Figure 3-3 will be modified and a new Figure added showing the location of the cesspools.

Typical leaching pool drawing will be added noting the depth of each drywell as measured in September 199.

Section 3.3 Geological Investigation - Comment 12 - Information will be added per the LKB report.

Section 4.0 General - New Comment - Applicable or relevant and appropriate requirements will be addressed including the information submitted by EPA.

Comment 15 - The lead and chromium levels found in the drywells and groundwater will be discussed.

Section 4.1 Drywell, Drain and Cesspool Analysis and - New Comment - "no volatile organic compounds ... statement will be revised by removing that portion of the statement and including information submitted by the data validator.

Comment 19 - Cesspool data will be incorporated into the RI report where applicable.

Section 4.2 Tank Investigation and Soil Borings Inside the Building - New Comment - The units will be changed to parts per million.

Comment 21 - More detailed descriptions of soil sample selection procedures will be made in the applicable sections with reference to the Anchor Chemcial Project Operations Plan and Work Plan.

Section 4.3 Monitoring Well Installation and Sampling and Analysis - 2 Rounds - New Comment - Page 4-11 - The discussion will be expanded and the trends discussed.

Section 4.4 In situ Specific Capacity Tests - Comment 26 - The requested amendment will be made.

Section 5.1.2 Drywells, Cesspools and Drain Sediments - Comment 37 - The requested changes will be made.

Drywells, Drain and Cesspool Sediments - Comment 42 - The statement will be eliminated as requested.

Drywells, Drain and Cesspool Sediments - Comment 43 - The requested changes will be made as requested.

Section 6.1.1 Nature and Extent of Contamination Drywells and Drain - Comment 50 - The comment regarding lead as a threat to human health will be deleted.

Groundwater - Comment 52 - A discussion of lead and chromium will be presented and the appropriate references made regarding the concentrations.

The comment in paragraph on page 6-8 will be deleted.

Section 6.1.2 Fate and Transport - Comment 53 - The statement "into nonhazardous organic compounds" will be deleted.

Comments Dealing with the Usage of Data from Hickville and Other Areas Offsite

Section 4.1 Drywell, Drain and Cesspool Analysis and - Comment 18 - The RI report will use data from 6S and 6D, as well as 1S and 1D as background data. Other data from areas offsite in the Hicksville area may also apply to the site and provide information pertinent to the site. Where these data are applicable they will be presented and their applicability discussed.

Section 5.2.1 Contaminant Persistence in the Vadose Zone - Comment 40 - The comment is the same as above.

Section 5.3.2 Saturated Zone - Comment 44 - The data from Westbury are relevant because the laboratory analyses were via target compound list via CLP package by Laboratory Resources, a EPA approved laboratory. Where these data are applicable they will be presented and their applicability discussed.

Comments With Which We Disagree

Section 4.2 Tank Investigation and Soil Borings Inside the Building - New Comment - The installation of additional soil borings to search for 2-butoxyethanol is not necessary for the following reasons:

chemical compound is not hazardous

compound was not detected in any groundwater samples
compound may be present beneath the building but will not be
"spread" as there are no liquids flowing through the
soils under the building

Section 4.2 Tank Investigation and Soil Borings Inside the Building - Comment 20 - The appropriate data validation comments will be included where appropriate as notes were made during the validation process indicating where bis(2-ethylhexyl)phthalate, acetone and methylene chloride are laboratory contaminants. Specific comments will be highlighted where appropriate.

Section 4.3 Monitoring Well Installation and Sampling and Analysis - 2 Rounds - Comment 22 - The appropriate data validation comments will be included where appropriate as notes were made during the validation process indicating where bis(2-ethylhexyl)phthalate, acetone and methylene chloride are laboratory contaminants. Specific comments will be highlighted where appropriate.

Section 4.3 Monitoring Well Installation and Sampling and Analysis - 2 Rounds - New Comment - The compound 1,4 dioxane is not hazardous. Additional sampling of groundwater is not required based on its detection.

If you have any questions, please do not hesitate to call me.

Very truly yours,

Dean Anson II

cc: Arthur Sanders, Jerry Spiegel Associates
Richard Leland, Esq., Rosenman & Colin