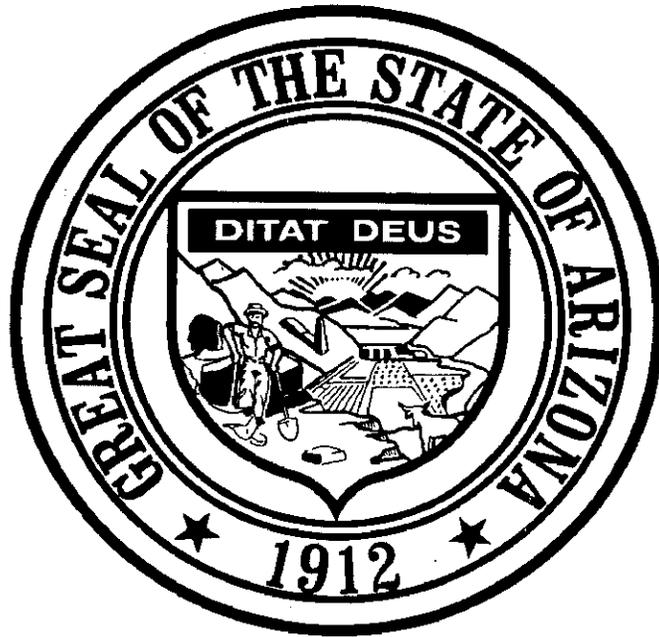


**Baseline Human Health Risk Assessment for
Sandy's Magic Touch Cleaners
Phoenix, Arizona**



Prepared By

*ARIZONA DEPARTMENT OF HEALTH SERVICES
Bureau of Epidemiology and Disease Control
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*Prepared For
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Executive Summary

The objective of this risk analysis is to provide an evaluation of human health risks that may result from exposure to solvents present in subsurface soils and groundwater near Sandy's Magic Touch Cleaners. The results suggest that, under current conditions, the subsurface soil contamination from the facility presents a negligible risk to shoppers and residents directly to the west of the facility. Insufficient data were available to estimate risks to employees from exposure to indoor vapors at Sandy's Cleaners and the adjoining businesses.

Data from a monitor well immediately downgradient of Sandy's Cleaners has been contaminated with tetrachloroethylene and its biodegradation products. Another nearby monitor well which is approximately 600 feet to the southwest of the facility has had one low level detection of tetrachloroethylene. A monitor well and a Salt River Project irrigation well approximately 800 feet to the east-northeast (upgradient) of the facility have not had any detections of tetrachloroethylene, suggesting that the tetrachloroethylene found in the groundwater in the monitor well behind Sandy's Cleaners may be attributable to releases from the facility.

While there are currently no complete exposure routes to water from wells in the impacted area, no institutional controls have been implemented to prevent the installation of domestic wells within the plume area. It is therefore possible that water may be drawn from contaminated portions of this aquifer in the future and used for domestic purposes. Potential future risks were evaluated using a conservative methodology that assumes steady-state conditions that do not consider attenuation and biodegradation. The site-related excess lifetime cancer risk in groundwater from the on-site monitor well (SMW-1) would range from 3E-04 (three-in-ten-thousand) for a central tendency residential exposure to 2E-03 (two-in-one-thousand) for reasonable maximum exposure. Both estimates are in excess of the acceptable range of risk established by the United States Environmental Protection Agency. The Hazard Index, which evaluates noncancer health effects, would be 5 under central tendency exposure conditions and 8 under a reasonable maximum exposure scenario, suggesting the potential for systemic health effects if water from this monitor well were used for domestic purposes.

1.0 INTRODUCTION

The purpose of this risk analysis is to evaluate the health risks that may be presented from exposure to volatile organic compounds (VOCs) in various media at Sandy's Magic Touch Cleaners.

1.1 Authority

Pursuant to Arizona Revised Statutes §§ 49-282, this risk analysis is written for the Arizona Department of Environmental Quality (ADEQ). This document was prepared using guidelines prescribed by the U.S. Environmental Protection Agency (USEPA) Risk Assessment Guidance for Superfund (RAGS), Volume I, Human Health Evaluation Manual: Part A¹ and RAGS Human Health Supplement.²

1.2 Overview

Sandy's Cleaners is located at 4730 East Indian School Road in Phoenix, Arizona. The facility is in a retail shopping center and is adjoined by other businesses. Asphalt parking areas are to the east and west of the facility. The retail center where Sandy's Cleaners is located was constructed in 1966 and the facility has contained a dry-cleaning business since then. Cleaners by George was at the site from 1966 until 1989, when Sandy's Cleaners took over the facility.

Subsurface soil and groundwater beneath Sandy's Cleaners have been contaminated with solvents. Earth Technology, Inc. has performed shallow soil, soil gas, and groundwater investigations at the site. The purpose of this risk analysis is to evaluate the health risks that may be presented by exposure to the contaminants in various media at the site.

1.3 Goals and Objectives

The goal of this risk analysis is to provide risk information necessary to assist decision-making within the risk management process. The objective of this risk analysis is to provide an evaluation of the current and potential future health risks that may result from exposure to solvents in subsurface soil and groundwater in the vicinity of the facility.

The analysis will be conducted using data collected from soils and wells in the vicinity of Sandy's Cleaners. Data used in the analysis consists of soil data collected and analyzed in 1996 and groundwater data collected and analyzed between 1984 and 1996, however, most of the data was collected between 1992 and 1996.

2.0 CHEMICALS OF CONCERN

Tetrachloroethylene (PCE), trichloroethylene (TCE), 1,1,1 trichloroethane (TCA), and 1,1 dichloroethylene are the chemicals of concern (COCs) in subsurface soils and soil gas. Seven compounds have been selected as COCs in groundwater including PCE and TCE. COCs selected for groundwater in a particular well may or may not be attributable to releases from the site.

2.1 Data Collection

Soil

This risk analysis uses soil data collected by the Earth Technology Corporation in May 1996. Thirty soil samples and one duplicate sample were collected from boreholes at depths of 7, 12, and 17 feet below ground surface. All of the boreholes were behind the building, with one borehole (DP-6) located directly next to the dry-cleaning equipment area. All samples were analyzed by EPA Method 8010 using an on-site mobile laboratory. The laboratory results were summarized in a June 1996 document published by Earth Technology, Inc.³ The document presents analytical results for detections of PCE, TCE, DCE, and TCA. Data in this document were converted into electronic format by ADHS staff for use in the risk assessment.

Soil Gas

This risk analysis uses soil gas data collected and analyzed by the Earth Technology Corporation in May 1996. Thirty soil vapor samples and 4 duplicate vapor samples were collected at 10 boreholes. Each borehole was sampled at 5, 10, and 15 feet below ground surface. All soil vapor samples were analyzed using EPA method 8010 using an on-site mobile laboratory. The laboratory results were summarized in a June 1996 document published by Earth Technology, Inc. The document presents analytical results for detections of PCE, TCE, DCE, and TCA. Data included in this document were converted into electronic format by ADHS staff for use in the risk assessment.

Groundwater

This risk assessment uses groundwater data from various sources collected and analyzed between 1992 and 1996. Included are data from three monitor wells (SMW-1, SMW-2, and SMW-3) drilled and sampled as part of an investigation of potential groundwater contamination from Sandy's Cleaners. Groundwater samples from these wells were collected in 1992, 1994 and 1996 by Earth Technology, Inc.,^{3 4,5,6} or Western

Technologies, Inc.⁷ Laboratory analyses were conducted by McKenzie Laboratories and Analytical Technologies, Inc. The laboratory results were summarized in documents published by Earth Technology, Inc. and Western Technologies, Inc. These data were converted into electronic format by ADHS staff for use in the risk assessment.

In addition, this risk assessment uses groundwater data from Salt River Project⁸ wells, piezometric wells operated by the Maricopa County Flood Control District, several monitor wells drilled to investigate contamination from a nearby underground fuel storage tank, and a private irrigation well identified as the "Peterson Well". Data for these wells were obtained on a disk prepared by Earth Technology, Inc. and from documents prepared by Earth Technology, Inc. and Western Technologies, Inc. These data have been compiled from a number of sources as part of the 1995 water quality investigation in East Central Phoenix. The water samples were collected by a number of organizations, and analyses were conducted by many different laboratories. The database includes analytical results from 1984 through 1996, however, most of the samples were collected between 1992 and 1996.

2.2 Data Evaluation

When more than one data point was available, the mean and the 95% upper confidence limit of the mean (UCL) concentrations of individual VOCs and inorganic chemicals were calculated for determination of COCs in shallow groundwater, surface soil, and soil vapors. The means and 95% UCLs were calculated using reported concentrations or one-half the Sample Quantification Limit (SQL) for each sample.

The screening levels used for soils include June 1995 Arizona non-residential Health-Based Guidance Levels (HBGLs)⁹ and proposed final Arizona Soil Remediation Levels (SRLs)¹⁰. Screening criteria for groundwater included USEPA Maximum Contaminant Levels (MCLs) and Arizona residential HBGLs for groundwater.

HBGLs and SRLs have been developed by the ADHS, and are levels of contaminants in soil considered to be safe. USEPA MCLs are concentrations of contaminants in water that may not be exceeded in public water systems and are promulgated by rule. ADHS water HBGLs are concentrations of contaminants in water considered to be safe.

2.3 Selection Methodology for Chemicals of Concern

This report assumes that all of the contamination at the site has been detected, and that chemicals of concern may be determined from investigation reports.

The selection methodologies described below use soil screening criteria discussed in Section 2.2 and current USEPA carcinogenicity Weight of Evidence (WoE) classifications.

WoE classifications have been assigned to many chemicals by the USEPA's Carcinogen Advisory Group. The WoE represents the carcinogenicity evidence from human and animal studies, and indicates the strength of the data. An A classification signifies that the chemical is a proven human carcinogen. Probable human carcinogens are designated either B1, showing that studies in humans are strongly suggestive but not conclusive, or B2 if the chemical has been conclusively carcinogenic in repeated animal studies but not conclusive in human studies. A chemical may be classified C, a possible human carcinogen, if a single high-quality animal study or several low-quality animal studies suggest carcinogenicity. If there is insufficient human and animal evidence to determine the carcinogenicity of the chemical, it is classified as D. A chemical conclusively shown to be non-carcinogenic to humans is in group E. This designation is rare due to the difficulty in producing the necessary negative data.

The following sections define the methodology used to select COCs in each media.

2.3.1 Soil

Chemicals were eliminated as COCs in soils if there were no positive detections in the data set; or the highest detected value was less than the June 1995 Arizona nonresidential HBGL and the proposed final SRL *and* the chemical is not recognized by the Integrated Risk Information System (IRIS)¹¹ as a possible (WoE=C), probable (WoE=B1,B2), or known human (WoE=A) carcinogen.

2.3.2 Soil Gas

Chemicals were eliminated as COCs in soil vapor only if there were no positive detections in the data set. The analysis uses all available soil gas data from all depths collected during the 1996 soil gas survey.

2.3.3 Groundwater

COCs in shallow groundwater were selected on a well by well basis. Chemicals were eliminated as COCs for a particular well if there were no positive detections in the data set for that well; or the highest detected value was less than the Maximum Contaminant Level (MCL) or current HBGL *and* the chemical is not recognized by IRIS as a possible (C), probable (B1, B2), or known human (A) carcinogen.

2.4 Identification of Chemicals of Concern

This section identifies the COCs in soils, soil vapor, and groundwater.

2.4.1 Soil

Table 2.4.1 summarizes the analytical results for soil samples collected during the 1996 soils investigation. Using the criteria outlined in Section 2.3.1, there are no COCs in the soil matrix.

**Table 2.4.1a Summary of 1996 Soil Sampling Results
7 Feet Below Ground Surface**

Chemical Name	Frequency of Detection Detect/Samples	Mean (mg/kg)	Standard Deviation (mg/kg)	Maximum (mg/kg)	95% Upper Confidence Limit (mg/kg)
Tetrachloroethylene (PCE)	7/7	0.014	0.017	0.050	0.030
Trichloroethylene (TCE)	0/7	N/A	N/A	N/A	N/A
1,1 Dichloroethylene (DCE)	1/7	0.600	1.587	4.200	2.266
1,1,1,Trichloroethane (TCA)	0/7	N/A	N/A	N/A	N/A

**Table 2.4.1b Summary of 1996 Soil Sampling Results
12 Feet Below Ground Surface**

Chemical Name	Frequency of Detection Detect/Samples	Mean (mg/kg)	Standard Deviation (mg/kg)	Maximum (mg/kg)	95% Upper Confidence Limit (mg/kg)
Tetrachloroethylene (PCE)	2/2	0.034	0.034	0.058	0.339
Trichloroethylene (TCE)	0/2	N/A	N/A	N/A	N/A
1,1 Dichloroethylene (DCE)	0/2	N/A	N/A	N/A	N/A
1,1,1,Trichloroethane (TCA)	0/2	N/A	N/A	N/A	N/A

**Table 2.4.1c Summary of 1996 Soil Sampling Results
17 Feet Below Ground Surface**

Chemical	Frequency of Detection Detect/Samples	Mean (mg/kg)	Standard Deviation (mg/kg)	Maximum (mg/kg)	95% Upper Confidence Limit (mg/kg)
Tetrachloroethylene (PCE)	4/4	0.072	0.088	0.200	0.212
Trichloroethylene (TCE)	1/4	0.001	0.000	0.001	0.001
1,1 Dichloroethylene (DCE)	1/4	0.002	0.003	0.006	0.006
1,1,1,Trichloroethane (TCA)	0/4	N/A	N/A	N/A	N/A

2.4.2 Soil Vapor

Table 2.4.2 summarizes the analytical results for soil vapor samples collected during the 1996 investigation. PCE, TCE, 1,1,1- TCA, 1,1 DCE have been selected as COCs in soil vapor. For off-site residential exposures, concentrations of solvents in groundwater were converted to soil gas concentrations in soil gas at the groundwater/alluvium interface.

**Table 2.4.2 Summary of 1996 Soil Vapor Sampling Results
Sandy's Cleaners Risk Assessment**

Chemical	Frequency of Detection Detect/Samples	Mean (µg/l)	Standard Deviation (µg/l)	Maximum (µg/l)	95% Upper Confidence Limit (µg/l)
Tetrachloroethylene (PCE)	31/34	1620	1509	5600	2148
Trichloroethylene (TCE)	4/34	7	5	13	9
1,1 Dichloroethylene (DCE)	11/34	17	22	86	25
1,1,1,Trichloroethane (TCA)	3/34	8	7	39	10

2.4.3 Groundwater

Table 2.4.3.1 displays the list of organic chemicals detected in groundwater in at least one well. Table 2.4.3.2 summarizes the concentrations of contaminants found in the entire data set. Table 2.4.3.3 displays the list of organics that did not meet the criteria for selection of COCs. Table 2.4.3.4 displays the list of COCs identified in at least one well.

Table 2.4.3.1 - Organic chemicals detected in groundwater,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Detects	Det %
O R G A N I C			
1. Benzene (BNZ)	71-43-2	1/ 13	7.7%
2. Bromodichloromethane (THM) (BDCM)	75-27-4	1/ 13	7.7%
3. Chloroform (THM) (CLFM)	67-66-3	2/ 13	15.4%
4. Dibromochloromethane (THM) (DBCM)	124-48-1	1/ 13	7.7%
5. Dichlorodifluoromethane (DCDFM)	75-71-8	1/ 8	12.5%
6. 1,1-Dichloroethylene (DCE)	75-35-4	1/ 13	7.7%
7. trans-1,2-Dichloroethylene	156-60-5	1/ 13	7.7%
8. 1,2-Dichloropropane (DCP2)	78-87-5	1/ 13	7.7%
9. Tetrachloroethylene (PCE)	127-18-4	8/ 15	53.3%
10. Toluene (TOL)	108-88-3	1/ 13	7.7%
11. Trichloroethylene (TCE)	79-01-6	1/ 15	6.7%

Table 2.4.3.2 - Organic chemical summary for all groundwater samples, Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	WOB HBGL	MCL
1. Benzene (BNZ)	71-43-2	µg/L	3.9	8.1	7	0.6	1/ 13	7.7% ✓	A	1E+00 5E+00
2. Bromobenzene	108-86-1	µg/L					0/ 5	0.0% --	ND	
3. Bromodichloromethane (THM) (BDCM)	75-27-4	µg/L	3.4	7.6	7	1	1/ 13	7.7% ✓	B2	6E-01 1E+02
4. Bromoform (THM) (BRFM)	75-25-2	µg/L					0/ 13	0.0% --	B2	4E+00 1E+02
5. Bromomethane (BM)	74-83-9	µg/L					0/ 13	0.0% --	D	1E+01
6. Carbon tetrachloride (CCL4)	56-23-5	µg/L					0/ 13	0.0% --	B2	3E-01 5E+00
7. Chlorobenzene (monochlorobenzene) (MCB)	108-90-7	µg/L					0/ 13	0.0% --	D	1E+02 1E+02
8. Chloroethane (CE)	75-00-3	µg/L					0/ 13	0.0% --	ND	
9. 2-Chloroethylvinyl ether (CEVE)	110-75-8	µg/L					0/ 8	0.0% --	ND	
10. Chloroform (THM) (CLFM)	67-66-3	µg/L	3.8	8	6.9	3.2	2/ 13	15.4% ✓	B2	6E+00 1E+02
11. Chloromethane (CM)	74-87-3	µg/L					0/ 13	0.0% --	C	3E+00
12. o-Chlorotoluene	95-49-8	µg/L					0/ 5	0.0% --	D	1E+02
13. para-Chlorotoluene	106-43-4	µg/L					0/ 5	0.0% --	ND	
14. Dibromochloromethane (THM) (DBCM)	124-48-1	µg/L	3.4	7.6	7	0.7	1/ 13	7.7% --	C	4E-01 1E+02
15. 1,2-Dichlorobenzene (DCB2)	95-50-1	µg/L					0/ 13	0.0% --	D	6E+02 6E+02
16. 1,3-Dichlorobenzene (DCB3)	541-73-1	µg/L					0/ 13	0.0% --	D	6E+02
17. 1,4-Dichlorobenzene (DCB4)	106-46-7	µg/L					0/ 13	0.0% --	C	2E+00 8E+01
18. Dichlorodifluoromethane (DCDFM)	75-71-8	µg/L	7.6	18	13	34	1/ 8	12.5% --	D	1E+03
19. 1,1-Dichloroethane (DCA)	75-34-3	µg/L					0/ 13	0.0% --	C	7E+01
20. 1,2-Dichloroethane (DCA2)	107-06-2	µg/L					0/ 13	0.0% --	B2	4E-01 5E+00
21. 1,1-Dichloroethylene (DCE)	75-35-4	µg/L	3.8	8.19999999	7.2	9	1/ 13	7.7% ✓	C	6E-02 7E+00
22. cis-1,2-Dichloroethylene	156-59-2	µg/L					0/ 13	0.0% --	D	7E+01 7E+01
23. trans-1,2-Dichloroethylene	156-60-5	µg/L	3.7	8	7.1	7	1/ 13	7.7% --	D	1E+02 1E+02
24. Dichloromethane (DCM)	75-09-2	µg/L					0/ 13	0.0% --	B2	5E+00 5E+00
25. 1,2-Dichloropropane (DCP2)	78-87-5	µg/L	4.6	10	9.6	29	1/ 13	7.7% ✓	B2	5E-01 5E+00
26. 1,3-Dichloropropane	142-28-9	µg/L					0/ 5	0.0% --	ND	
27. 2,2-Dichloropropane		µg/L					0/ 5	0.0% --	ND	
28. 1,1-Dichloropropene	563-58-6	µg/L					0/ 5	0.0% --	ND	
29. cis-1,3-Dichloropropene (cDCP3)	10061-01-5	µg/L					0/ 13	0.0% --	B2	
30. trans-1,3-Dichloropropene (tDCP3)	10061-02-6	µg/L					0/ 8	0.0% --	B2	
31. 1,3-Dimethylbenzene (Xylene-m)	108-38-3	µg/L					0/ 5	0.0% --	ND	1E+04
32. 1,4-Dimethylbenzene (Xylene-p)	106-42-3	µg/L					0/ 8	0.0% --	ND	1E+04
33. Ethylbenzene (EB)	100-41-4	µg/L					0/ 13	0.0% --	D	7E+02 7E+02
34. Ethylene dibromide (EDB)	106-93-4	µg/L					0/ 5	0.0% --	B2	4E-04 5E-02
35. Styrene	100-42-5	µg/L					0/ 5	0.0% --	C	1E+02 1E+02
36. 1,1,1,2-Tetrachloroethane	630-20-6	µg/L					0/ 5	0.0% --	C	1E+00
37. 1,1,1,2,2-Tetrachloroethane (TET)	79-34-5	µg/L					0/ 13	0.0% --	C	2E-01

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HRGL or was less than the MCL and the WOE is not "A" or "B2".

Table 2.4.3.2 - Organic chemical summary for all groundwater samples, Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	MoE	HBGL	MCL
38. Tetrachloroethylene (PCE)	127-18-4	µg/L	850	1600	1300	7.5	4000	8/15	53.3%	✓	B2 7E-01 5E+00
39. Toluene (TOL)	108-88-3	µg/L	3.8	8.1	7	0.7	0.7	1/13	7.7%	--	D 1E+03 1E+03
40. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L						0/15	0.0%	--	D 6E+02 2E+02
41. 1,1,2-Trichloroethane (TCRA2)	79-00-5	µg/L						0/13	0.0%	--	C 6E-01 5E+00
42. Trichloroethylene (TCE)	79-01-6	µg/L	8.4	18	18	35	35	1/15	6.7%	✓	B2 3E+00 5E+00
43. Trichlorofluoromethane (TCFM)	75-69-4	µg/L						0/8	0.0%	--	D 2E+03
44. 1,2,3-Trichloropropane	96-18-4	µg/L						0/5	0.0%	--	D 5E-03
45. Vinyl chloride (VC)	75-01-4	µg/L						0/13	0.0%	--	A 2E-02 2E+00

ORGANIC

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the MoE is not "A" or "B2".

Table 2.4.3.3 - Organic chemicals in groundwater eliminated from the risk as Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Detects	Det %
O R G A N I C			
1. Bromobenzene	108-86-1	0/ 5	0.0%
2. Bromoform (THM) (BRFM)	75-25-2	0/ 13	0.0%
3. Bromomethane (BMM)	74-83-9	0/ 13	0.0%
4. Carbon tetrachloride (CCL4)	56-23-5	0/ 13	0.0%
5. Chlorobenzene (monochlorobenzene) (MCB)	108-90-7	0/ 13	0.0%
6. Chloroethane (CE)	75-00-3	0/ 13	0.0%
7. 2-Chloroethylvinyl ether (CEVE)	110-75-8	0/ 8	0.0%
8. Chloromethane (CM)	74-87-3	0/ 13	0.0%
9. o-Chlorotoluene	95-49-8	0/ 5	0.0%
10. para-Chlorotoluene	106-43-4	0/ 5	0.0%
11. Dibromochloromethane (THM) (DBCM)	124-48-1	1/ 13	7.7%
12. 1,2-Dichlorobenzene (DCB2)	95-50-1	0/ 13	0.0%
13. 1,3-Dichlorobenzene (DCB3)	541-73-1	0/ 13	0.0%
14. 1,4-Dichlorobenzene (DCB4)	106-46-7	0/ 13	0.0%
15. Dichlorodifluoromethane (DCDFM)	75-71-8	1/ 8	12.5%
16. 1,1-Dichloroethane (DCA)	75-34-3	0/ 13	0.0%
17. 1,2-Dichloroethane (DCA2)	107-06-2	0/ 13	0.0%
18. cis-1,2-Dichloroethylene	156-59-2	0/ 13	0.0%
19. trans-1,2-Dichloroethylene	156-60-5	1/ 13	7.7%
20. Dichloromethane (DCM)	75-09-2	0/ 13	0.0%
21. 1,3-Dichloropropane	142-28-9	0/ 5	0.0%
22. 2,2-Dichloropropane		0/ 5	0.0%
23. 1,1-Dichloropropene	563-58-6	0/ 5	0.0%
24. cis-1,3-Dichloropropene (cDCP3)	10061-01-5	0/ 13	0.0%
25. trans-1,3-Dichloropropene (tDCP3)	10061-02-6	0/ 8	0.0%
26. 1,3-Dimethylbenzene (Xylene-m)	108-38-3	0/ 5	0.0%
27. 1,4-Dimethylbenzene (Xylene-p)	106-42-3	0/ 8	0.0%
28. Ethylbenzene (ETB)	100-41-4	0/ 13	0.0%
29. Ethylene dibromide (EDB)	106-93-4	0/ 5	0.0%
30. Styrene	100-42-5	0/ 5	0.0%
31. 1,1,1,2-Tetrachloroethane	630-20-6	0/ 5	0.0%
32. 1,1,2,2-Tetrachloroethane (TET)	79-34-5	0/ 13	0.0%
33. Toluene (TOL)	108-88-3	1/ 13	7.7%
34. 1,1,1-Trichloroethane (TCA)	71-55-6	0/ 15	0.0%
35. 1,1,2-Trichloroethane (TCA2)	79-00-5	0/ 13	0.0%
36. Trichlorofluoromethane (TCFM)	75-69-4	0/ 8	0.0%
37. 1,2,3-Trichloropropane	96-18-4	0/ 5	0.0%
38. Vinyl chloride (VC)	75-01-4	0/ 13	0.0%

Table 2.4.3.4 - Summary of organic chemicals of potential concern in groundwater
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	MoE HBSL	MCL
1. Benzene (BNZ)	71-43-2	µg/L	3.9	8.1	7	0.6	1/ 13	7.7% ✓	A	1E+00 5E+00
2. Bromodichloromethane (THM) (BDCM)	75-27-4	µg/L	3.4	7.6	7	1	1/ 13	7.7% ✓	B2 6E-01	1E+02
3. Chloroform (THM) (CLFM)	67-66-3	µg/L	3.8	8	6.9	3.2	2/ 13	15.4% ✓	B2 6E+00	1E+02
4. 1,1-Dichloroethylene (DCE)	75-35-4	µg/L	3.8	8.19999999	7.2	9	1/ 13	7.7% ✓	C 6E-02	7E+00
5. 1,2-Dichloropropane (DCPZ)	78-87-5	µg/L	4.6	10	9.6	29	1/ 13	7.7% ✓	B2 5E-01	5E+00
6. Tetrachloroethylene (PCE)	127-18-4	µg/L	850	1600	1300	7.5	4000	53.3% ✓	B2 7E-01	5E+00
7. Trichloroethylene (TCE)	79-01-6	µg/L	8.4	18	18	35	1/ 15	6.7% ✓	B2 3E+00	5E+00

O R G A N I C

2.5 Data Quality

Quality Assurance/Quality Control (QA/QC) for soil and soil vapor samples included the collection and analysis of duplicate samples. These results suggested that the soil vapor data is of adequate quality for use in this risk assessment.

QA/QC procedures for groundwater data included the collection of equipment and trip blanks, and laboratory spike analyses. The results suggest that the data are of adequate quality for use in this risk assessment.

3.0 EXPOSURE ASSESSMENT

This exposure assessment focuses on current and potential future human exposure to COCs in soil, soil vapor, and shallow groundwater near Sandy's Cleaners.

3.1 Exposure Setting

3.1.1 Physical Setting

Location

Sandy's Cleaners is located at 4730 East Indian School Road in Phoenix, Arizona. The facility is in a shopping center that is primarily surrounded by residential and commercial property.

Meteorology

Sandy's Cleaners is in the Salt River Valley within the Sonoran Desert Climate Region. The area is characterized by hot summers and mild winters. Average maximum daily temperatures range from a high of 105° F in July, to a low of 65° F in December. Precipitation averages approximately 7 inches annually; with most rainfall occurring during the summer (July through September), and the winter (December through March). Average annual pan evaporation is approximately 106 inches, allowing little rainfall infiltration below the root zone.

Wind velocities are recorded at Phoenix Sky Harbor Airport. Climatic data indicates wind velocities of 0 - 3 mph, 4 - 6 mph, 7 - 10 mph, and greater than 10 mph occur 13 %, 43 %, 31 %, and 8% of the time. Winds are calm an average of 5% of the time.

Geology

The geology of the site is a two-layer system of alluvium overlying bedrock. Features of particular interest include a well-defined bedrock high to the west (Papago Buttes) and south. Overall downward slope to the west-southwest.

The Salt River Valley is a series of coalesced alluvial basins. Sandy's Cleaners is built on an alluvial fan, situated near the eastern margin of the West Basin of the Salt River Valley. This basin is a structural depression bounded by the Papago Buttes.⁶

Soil

The Salt River Valley is commonly filled to depths of more than 1,000 feet by sand, gravel, silt and clay that have eroded from the uplifted bedrock uplands. Although fine-grained lacustrine and evaporite deposits have accumulated at the lower elevations during periods of wetter climates, deposits on the alluvial fans and aprons are predominantly coarse-grained sand and gravel.

The soils underlying the site and the immediate surrounding area are loams to gravelly loams, including soils classified in the Gavelt, Rillito, and Laveen series. Permeability is moderate and available water capacity is high, with an average vertical gradient of about 0.48 in/ft. In some places the soil is strongly cemented by carbonates to form caleche, but the cementation is variable and does not appear to form continuous layers.

Hydrology

Groundwater near Sandy's Cleaners occurs in two distinct water bearing hydrogeologic formations, alluvium and bedrock, as part of a regional flow system. The water table near the facility is in the alluvium, and occurs about 20 feet below ground surface. The alluvial aquifer underlying the study area consists of saturated unconsolidated sands and gravel with varying amounts of silt and clay. A lower unconsolidated unit is alluvial valley fill material most probably derived from erosion during local Tertiary Basin and Range development.

The bedrock underlying the study site is dominated by Tertiary sedimentary Camels Heads and Tempe formations. Groundwater flow in bedrock occurs primarily in open fractures, joints and bedding planes. Near the site, the predominant direction of regional groundwater flow is to the west/southwest.

The Salt River channel flows about 4 miles south of Sandy's Cleaners. Flow in the Salt River is regulated, and the channel is usually dry except during periods of heavy precipitation or releases from dams up river. The Arizona Canal flows from the southeast to the northwest just to the north of Sandy's Cleaners. It is normally used to supply irrigation water to the central and western portions of the Salt River Project Irrigation District.

3.1.2 Potentially Exposed Populations

The populations potentially exposed to contaminants in soil, soil vapor or groundwater in the study area include:

Current Exposure:

- Individuals working at Sandy's Cleaners
- Individuals working at other businesses at the shopping center
- Individuals shopping at the center
- Individuals living directly behind the facility

Potential Future Exposure:

- Individuals working at Sandy's Cleaners
- Individuals working at other businesses at the shopping center
- Individuals shopping at the center
- Individuals living directly behind the facility
- Individuals that may live over the contaminated soil if land uses change to residential in the future
- Individuals that may drill wells and drink water from the contaminated portion of the aquifer near the facility

3.2 Exposure Pathway Identification

A potentially complete human exposure pathway describes the route a chemical may take from the source to a receptor. A complete exposure pathway includes the following components:

- 1) A source and mechanism of release to the environment.
- 2) A medium for the transport of the released chemical to the environment.
- 3) A point of potential human contact with the contaminated medium (an exposure point).
- 4) An exposure route at the exposure point, (ingestion, inhalation, dermal contact).

3.2.1 Source and Receiving Media

The source of anthropogenic chemicals in the soil and groundwater may be attributed to releases from standard operating procedures. Soil likely served as the receiving media, releasing the chemicals to groundwater. In the saturated zone, VOCs may have existed as undissolved free product in the soil pore spaces. These VOCs likely slowly migrated into the groundwater.

Unlike most volatile organic compounds, inorganic constituents occur naturally in groundwater requiring the identification, evaluation, and estimation of inorganic chemicals in a relation to natural background concentrations. Although ambient water quality characteristics are influenced primarily by natural processes, they may also be affected by land use such as agricultural irrigation. This risk assessment does not address inorganic constituents in soil or groundwater.

3.2.2 Fate and Transport in Release Media

The role of each environmental medium in the accumulation, release, transport and transformation of COCs are discussed below.

Groundwater

Chemicals infiltrate and leach through the unsaturated and saturated zones of the soil to reach groundwater. VOC contamination has been detected in monitor wells placed both on and off-site.

The evaluation of the anthropogenic contribution of inorganics can be difficult to quantify due to their natural occurrence in groundwater. Factors affecting fate and transport of inorganics include:

- 1) Variable natural processes dependent on hydrogeologic environments.
- 2) Artificial and natural recharge to the aquifer may cause local dilution.
- 3) Changes in land use, irrigation patterns, and groundwater pumping rates.

Surface Water

Natural surface waters near the facility are intermittent and occur primarily as runoff from storms. The Arizona Canal is the nearest permanent surface water feature, but runoff near the facility would not be expected to discharge into the canal. Under current land use conditions for the site, artificial surface coverings and storm water runoff systems prevent most opportunities for contaminants in soil or groundwater to contact surface water.

Air

Some chemicals may be released into the outdoor air through volatilization from soil. In addition, the potential exists for release of volatile contaminants into buildings through cracks in floors and foundations.

Soil

VOCs present in soil can be adsorbed on the soil matrix; percolate through unsaturated soils to groundwater; or be released to the air through volatilization. Adsorption can lead to immobility and increased resistance to chemical or biological degradation. The soils at the site have air conductivities that are sufficient for rapid percolation and for underground volatilization and vapor movement to occur.

Degradation and Transformation

Most of the organic compounds detected in groundwater at the site are chlorinated solvents. Transformations in subsurface areas are believed to be responsible for the TCE and 1,1 DCE.

3.2.3 Exposure Points and Routes

The potentially complete exposure route to the contaminants in soil is inhalation of vapors that diffuse through soils. Exposure routes are discussed in more detail in this section.

Exposure to PCE may be possible from inhalation of vapors diffusing through soil and vapor barriers. Soil and soil vapor data will be used quantitatively to estimate exposure to vapors from these releases. Ingestion and dermal contact with contaminated soils are not quantitatively evaluated since no complete exposure route exists.

There is currently no exposure to COCs in groundwater from the facility since there are no domestic use private or public wells in the area influenced by the contamination. However, exposure to contaminated groundwater near the facility is considered possible in the future since there is no limitation of future installation of wells in the aquifer.

Currently Complete Exposure Routes:

- Individuals working at the Sandy's Cleaners may inhale solvent vapors diffusing through soil and vapor barriers.
- Individuals working at other businesses at the shopping center may inhale solvent vapors diffusing through soil and vapor barriers.
- Individuals shopping at the center may inhale solvent vapors diffusing through soil and vapor barriers.
- Individuals living directly behind the facility may inhale solvent vapors diffusing through soil and vapor barriers.

Potential Future Exposure Routes:

- Individuals working at Sandy's Cleaners that may inhale solvent vapors diffusing through soil and vapor barriers.
- Individuals working at other businesses at the shopping center that may inhale solvent vapors diffusing through soil and vapor barriers.
- Individuals shopping at the center that may inhale solvent vapors diffusing through soil and vapor barriers.
- Individuals living directly behind the facility that may be inhale solvent vapors diffusing through soil and vapor barriers.
- Individuals that may live over the contaminated soil if land uses change in the future may inhale vapors diffusing through soil and vapor barriers.
- Individuals may use water from the contaminated portion of the aquifer near the facility for domestic purposes (ingestion, inhalation, and dermal contact).

3.2.4 Summary of Complete Exposure Pathways

The following tables summarize current and potential future pathways at the site.

Table 3.2.4.1: Current Exposure Pathway Summary

Potential Exposed Population	Exposure Point	Exposure Route	Path Evaluated	Path Selected	Exposure Type	Rationale
CURRENT LAND USE						
Workers	Contaminated groundwater in on-site shallow groundwater.	Ingestion Inhalation Dermal	Yes Yes Yes	No No No	None	No current use of on site shallow groundwater
Off-site Residents	Contaminated groundwater in off-site shallow groundwater	Ingestion Inhalation Dermal	Yes Yes Yes	No No No	None	No current contamination of private wells in shallow aquifer
Soil/Soil Vapors						
Workers	Contaminated sub-surface soils	Ingestion Inhalation Dermal	Yes Yes Yes	No Yes No	None Potential None	Possible inhalation of solvent vapors diffusing through vapor barriers, no accessible surface soil
Off Site Residents	Contaminated sub-surface soils	Ingestion Inhalation Dermal	Yes Yes Yes	No Yes No	None Potential None	Possible inhalation of solvent vapors diffusing through vapor barriers, no accessible surface soil

Table 3.2.4.2: Potential Future Exposure Pathway Summary

Potential Exposed Population	Exposure Point	Exposure Route	Path Evaluated	Path Selected	Exposure Type	Rationale
POTENTIAL FUTURE LAND USE						
Groundwater						
Workers	Contaminated groundwater in on-site shallow groundwater.	Ingestion Inhalation Dermal	Yes Yes Yes	Yes Yes Yes	Potential	No restriction on installing new wells
Off-site Residents	Contaminated groundwater in off-site shallow groundwater	Ingestion Inhalation Dermal	Yes Yes Yes	Yes Yes Yes	Potential	No restriction on installing new wells
Soil/Soil Vapors						
Workers	Contaminated sub-surface soils	Ingestion Inhalation Dermal	Yes Yes Yes	No Yes No	None Potential None	Possible inhalation of solvent vapors diffusing through vapor barriers, no accessible surface soil
Off-site Residents	Contaminated sub-surface soils	Ingestion Inhalation Dermal	Yes Yes Yes	No Yes No	None Potential None	Possible inhalation of solvent vapors diffusing through vapor barriers, no accessible surface soil

3.3 Quantification of Exposures

Estimates of exposure concentrations and pathway specific intake doses must be made to quantify exposures. Repeated, prolonged (chronic) exposures are assumed, due to the low levels of exposure via environmental media. Exposures from inhalation of solvent vapors indoors and outdoors will be estimated for current conditions. Potential future exposures to groundwater will also be estimated.

The upper 95% confidence limits (UCL) of the concentrations were used to estimate exposure concentrations in air. The formula for calculating the UCL is as follows:

$$\text{UCL} = \text{mean} + t_{(n-1)} * (\sigma / \sqrt{n})$$

3.3.1 Soil Vapor Estimation Methods

The health risks presented from inhalation of vapors that may infiltrate into the facilities and outdoor air near the facility were modeled using a conservative methodology. Exposure estimates were made using soil gas concentrations for on site transient exposure. Exposure estimates for off site residential exposures were calculated using the concentration in water, and converting it to a soil gas concentration at the interface of the groundwater and dry alluvium. Concentrations of solvents in groundwater (in mg/L) were converted to soil gas concentrations in soil gas (in mg/L) at the groundwater/alluvium interface by multiplying the chemical concentration in groundwater by the chemical-specific dimensionless Henry's Law constant (H').

Flux Estimation Methods

Flux from the solvent in soil gas was calculated using the Karimi *et al.*¹² model as described in the EPA Superfund Exposure Assessment Manual (SEAM)¹³ and the Air/Superfund National Technical Guidance Study Series Document: Assessing Potential Indoor Air Impacts for Superfund Sites.¹⁴ The Karimi model assumes zero concentration of volatilizing material at the soil surface and a non-diminishing and continuous source of contaminants in a system in equilibrium.

Each of the measured soil gas concentrations of were used in the Karimi model to calculate flux as represented by the following equation:

$$J_i = (D_i)(C_g)(P_a^{3.33}/P_t^2)/L$$

where:

J_i	=	Flux Rate of Component i (mg/m ² •sec)
D_i	=	Diffusion Coefficient in Air of Component i (m ² /sec)
C_g	=	Concentration in Soil Gas of Component i (mg/m ³)
P_a	=	Air Filled Porosity of the Soil (0.25 dimensionless)
P_t	=	Total Soil Porosity (0.45 dimensionless)
L	=	Depth to Contamination (m)

The upper 95% confidence limit (UCL) of the flux values was used to estimate flux for the entire facility. The formula for calculating the UCL is as follows:

$$UCL = \text{mean} + t_{(n-1)} * (\sigma/\sqrt{n})$$

Indoor Air Estimation Methods

A conservative indoor air model was used to predict concentrations of contaminants in indoor air for receptors. The concentration in indoor air was calculated using default dimensions using the following model:

$$IAC = (J_i)(a)(F)/(ACH/3600)(v)$$

where:

IAC	=	Indoor Air Concentration (mg/m ³)
J_i	=	Flux Rate of Component i (mg/m ² •sec)
a	=	Area of Building Floor (m ²)
F	=	Fraction of Floor Through Which Soil Gas May Enter (unitless)
ACH	=	Building Air Changes Per Hour (air changes/hr)
v	=	Volume of Building (m ³)

The indoor air concentration (IAC) is dependent on the fraction of floor (F) through which soil gas may enter, the volume of the building (v) and the number of air changes per hour (ACH). The value for F was assumed to be 0.001.¹⁴ The value for ACH was conservatively assumed to be 0.8. The volumes of the off-site houses were assumed to be 400 m³, based upon observations during a site visit. The 95% UCL of flux was used as a measure of flux for the houses.

Outdoor Air Concentration Estimation Methods

A "box model" was used to predict conservative ambient concentrations of contaminants in outdoor air for receptors outside the building. The model predicts conservative ambient concentrations of contaminants for receptors at the downwind edge of the exposure area, and assumes that vapors within the box are well mixed. The concentration of the component in outdoor air was calculated using the following "box model"¹⁴:

$$OAC = (J_i)(a)(F)/(w)(h)(u)$$

where:

OAC	=	Outdoor Air Concentration (mg/m ³)
J _i	=	Flux Rate of Component i (mg/m ² •sec)
a	=	Area of Emission (m ²)
F	=	Fraction of Surface Available for Diffusion (dimensionless)
w	=	Square Root of Box Area (m)
h	=	Height of Box (m)
u	=	Wind Velocity (m/sec)

The model to estimate outdoor air concentrations above the parking lot assume vapors in soil gas diffuse into an imaginary "box", with dimensions of 10m x 10m x 3m. The average annual wind speed for Phoenix of 2.6 m/sec was used in the equation. The estimate assumes that the outdoor area is unpaved and assumes an F value of 0.001, which represents the fraction of cracks in pavement available for diffusion.

Predicted outdoor air concentrations were then used in the exposure scenario outlined in Table 3.2 to quantify potential health risks.

3.4 Exposure Estimates

Appendix Table A displays estimated CDIs for each of the complete exposure pathways under current and potential future conditions. Sections 3.4.1 and 3.4.2 discuss the formulas and assumptions for each receptor by pathway.

3.4.1 Inhalation of Vapors

Variable values used to estimate occupational and residential exposures incorporate standard assumptions adopted by the USEPA. Transient exposures were quantified using professional judgement and standard default inhalation rates. The following equations and assumptions were used to quantify exposure to solvent vapors:

Table 3.4.1 - Calculation of inhalation intakes from exposure to vapors

$$\text{CHRONIC DAILY INTAKE: } \text{CDI} = \frac{(\text{AC})(\text{IR})(\text{EF})(\text{ED})}{(\text{BW})(\text{AT})}$$

Where:

- AC = Chemical concentration in air (mg/m³)
- IR = Inhalation rate (m³/day or workday)
- EF = Exposure frequency (days/year)
- ED = Exposure duration (years)
- BW = Body weight (kilograms)
- AT = Averaging time (days)

Variable Values:

	<u>Occupational</u>	<u>Residential</u>	<u>Transient</u>
AC:	95% UCL (mg/m ³)	95% UCL (mg/m ³)	95% UCL (mg/m ³)
IR:	20 (m ³ /workday)	20 (m ³ /day)	2 (m ³ /day)
EF:	250 (days/year)	350 (days/year)	50 (days/year)
ED:	25 (years)	30 (years)	6 (years)
BW:	70 (kg)	70 (kg)	70 (kg)
AT: (carc.)	25,550 (days)	25,550 (days)	25,550 (days)
AT: (non-carc.)	9,125 (days)	10,950 (days)	2,190 (days)

3.4.2 Ingestion of Groundwater

Variable values used to estimate potential future residential exposure incorporate standard assumptions adopted by the USEPA.

The CDI values using the formula in Table 3.4.2 are calculated using the contaminant concentrations displayed in Appendix Table A. The CDIs are displayed in Appendix Table B.

Table 3.4.2 - Groundwater Ingestion Intake Formula.

$$\text{Chronic Daily Intake: } \text{CDI} = \frac{(\text{CW})(\text{IR})(\text{EF})(\text{ED})}{(\text{BW})(\text{AT})}$$

Where:

- CW = Chemical Concentration in Water (mg/L)
- IR = Water Ingestion Rate (L/day)
- EF = Exposure Frequency (days/year)
- ED = Exposure Duration (years)
- BW = Body Weight (kg)
- AT = Averaging Time (days)

Potential Future Residential Exposures-Monitor Well Data

Variable Values:		Central Tendency	RME
CW:	(mg/L)	Mean	95 % UCL*
IR:	(L/day)	2	2
EF:	(days/year)	350	350
ED:	(years)	9	30
BW:	(kg)	70	70
AT:	For carcinogenic effects = 70 years x 365 days/year For noncarcinogenic effects = ED x 365 days/year		

* Maximum detected concentration used when 95% UCL exceeded maximum detected concentration

Groundwater: Inhalation and Dermal

Dermal exposures assume that organic compounds in contact with any part of the body may be absorbed proportionally to the body surface area contacted. Human skin, however, acts as a relatively impermeable physical barrier, often preventing substantial absorption of contacted chemicals. The skin's protective effect is influenced by the properties of the organic compound, by the presence of soil particles on the skin or in the delivery media, by the amount of dilution and the diluent, and by any abrasions present.

In this risk assessment, USEPA Region IX guidance for calculation of risks from ingestion exposures to residential water supplies was followed. The sum of the risk or hazard due to inhalation and dermal exposures were assumed to be equal to that for ingestion for VOCs. The rationale for this approach is the wide range of estimated residential exposures by the inhalation and dermal routes. It is believed that this is a conservative assumption based on available data and does not impart a false sense of precision to the estimate. The use of this simplifying assumption requires that only the ingestion CDI be calculated, ingestion risk is then estimated and the result is multiplied by two to estimate total risk.

3.5 Uncertainties in the Exposure Assessment

All exposure parameters were chosen to produce conservative estimates of total risk from exposure to contaminants.

Exposures calculated from soil gas and soil concentrations of solvents were not measured, but were modeled using conservative methodologies. The major modeling efforts in this assessment are related to the releases of VOCs to the atmosphere from soil. It should be recognized that when a model is used the uncertainty of the estimated quantities is greater than if an accurate measurement were taken. Modeling creates uncertainties in the exposure analysis, however, due to the conservative models used, actual exposure is likely to be less than that estimated here.

All exposure parameters were chosen to produce conservative estimates of total risk from exposures to contaminants. Exposure concentrations used in the calculation of reasonable maximum intakes are 95% upper-bounds estimates.

Exposure doses (CDI) used in the calculation of carcinogenic risks and noncarcinogenic hazard quotients are also included in the risk calculation worksheets in the Appendix. These doses are based on the assumptions and calculations shown in previous sections. They may be considered upper-bound estimates. The estimated doses are used with slope factors (carcinogenic risk calculations) and reference doses (noncarcinogenic calculations) to produce probability estimates of carcinogenic risk, and hazard quotients for noncarcinogenic adverse health effects.

4.0 TOXICITY ASSESSMENT

The toxicological information on the chemicals of concern for this study is summarized in this chapter. Emphasis is placed upon the non-carcinogenic and carcinogenic effects with discussions on the dose-response variables (reference dose, slope factor) used in the statement of risk. Each chemical is summarized with regard to use, interactions with other chemicals, exposure routes, toxicokinetics, toxic (health) effects, and carcinogenicity. Detailed toxicological profiles for each COC are included in the Appendix.

4.1 Dose-Response Variable for Non-Carcinogenic Effects

The reference dose (RfD) is used as a dose-response variable for assessing the non-carcinogenic effects of exposure to chemicals. The chronic RfD is used in calculating the risk of long-term exposure to specific chemicals. USEPA defines the chronic reference dose as "an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chronic RfDs are specifically developed to be protective for long-term exposure to a compound".¹ The USEPA derives the RfDs from animal and, when available, human studies by taking the highest dose at which no adverse effect is seen (NOAEL or no-observed-adverse-effect level) and dividing it by the product of the uncertainty factor (UF) and modifying factor (MF) as shown in the formula below (1). The UF is usually 10 or factors of 10 and estimates the uncertainty in the data from which the NOAEL is derived, especially if it is obtained from animal studies. The MF usually ranges from 0 to 10 and suggests further uncertainty as judged by the professional.

$$\text{RfD} = \text{NOAEL}/\text{UF} \times \text{MF}$$

The RfD is measured in mg/kg-day and assumes a threshold or level of exposure at which no adverse health effect will be seen. Although the subchronic RfD is available for short-term exposures, the chronic RfD is used in this study to measure the long-term, non-carcinogenic effect from exposure to the chemicals of concern. The noncarcinogenic hazard quotient (HQ) is computed by dividing the exposure level for the chemical of concern by the specific RfD for that chemical. The noncarcinogenic hazard index (HI) is computed by summing the HQ for individual chemicals for an exposure pathway and represents an estimate of the total hazard for that pathway. Adverse health effects may occur when the HQ or HI exceeds one. RfDs for non-carcinogenic toxicity were obtained from the USEPA on-line Integrated Risk Information System (IRIS)¹¹ database, and the USEPA Health Effects Assessment Summary Tables (HEAST)¹⁵, FY-1995. Table 4-1 displays RfDs for the COCs.

Table 4.1: Reference Dose (RfD) for Ingestion and Inhalation

Chemical	Inhalation RfD (mg/kg-d)	Ingestion RfD (mg/kg-d)	Confidence in Data (Oral)	Sensitive Organs and Systems Affected	RfC/RfD Source	UF/NF
Benzene (BNZ)	---	---	---	Neurological / Blood, Immune, Neurological, Respiratory, Skin	---	---
Bromodichloromethane (THM) (BDCM)	2E-2	2E-2	Medium / ---	Kidney/Liver Lesions	IRIS / IRIS	1,000/1 / ---
Chloroform (THM) (CLFM)	1E-2	1E-2	Medium / ---	Blood, Developmental, Gastrointestinal, Kidney, Liver, Neurological, Respiratory / Cardiovascular, Gastrointestinal, Kidney, Liver, Neurological, Respiratory	IRIS / IRIS	1,000/1 / ---
1,1 Dichloroethylene (DCE)	9E-3	9E-3	Medium / ---	Hepatic Lesions	IRIS / IRIS	1,000/1 / ---
1,2-Dichloropropane (DCP2)	1.1E-3	1.1E-3 (R3) (4E-3)	--- / Medium	Blood, Gastrointestinal, Heart, Kidney, Liver, Neurological / Blood, Gastrointestinal, Kidney, Liver, Neurological, Respiratory, Skin	IRIS/ IRIS	--- / 300/1
Tetrachloroethylene (PCE)	1E-2	1E-2	Medium / ---	Liver, Gastrointestinal, Neurological / Cardiovascular, Eye, Kidney, Liver, Neurological, Reproductive, Respiratory	IRIS / IRIS	1,000/1 / ---
1,1,1 Trichloroethane (TCA)	N/A	N/A			IRIS / IRIS	
Trichloroethylene (TCE)	N/A	N/A			IRIS / IRIS	

4.2 Dose-Response Variable for Carcinogenic Effects

The slope factor (SF) is utilized as the dose-response variable for assessing the carcinogenic effects of exposure to chemicals. USEPA defines the slope factor as "a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime. The slope factor is used to estimate an upper-bound probability of an individual developing cancer as a result of a lifetime of exposure to a particular level of a potential carcinogen".¹ The SF is an estimate of the quantitative relationship between dose and carcinogenic response.

The SF is measured in units of $(\text{mg}/\text{kg}\text{-day})^{-1}$ and is usually determined using the upper 95 percent confidence limit of the slope of the linearized multi-stage model. The model assumes that there is no threshold for the initiation of cancer (i.e. any exposure poses a risk of cancer). Since data on carcinogenicity is often derived from high-dose experiments on animals, extrapolations are made from these high doses to lower doses. When available, human data are used to determine the slope factor. Excess cancer risk is expressed as a function of exposure and is calculated by multiplying an estimated dose of a chemical by the slope factor (SF). The application of the nonthreshold assumption and the use of the upper 95 percent confidence limit for estimating the slope factor provides a conservative estimate of potential carcinogenic risk.

From human and animal experimental data, the USEPA's Carcinogen Advisory Group has grouped chemicals by weight-of-evidence (WoE) into classes from A to E which designate their potential as a cancer-causing agent. The WoE represents the carcinogenicity evidence from human and animal studies and indicates the strength of the data. An A classification signifies that the chemical is a proven human carcinogen. Probable human carcinogens are designated either B1, showing that studies in humans are strongly suggestive but not conclusive, or B2 if the chemical has been conclusively carcinogenic in repeated animal studies but not conclusive in human studies. A chemical may be classified C, a possible human carcinogen, if a single high-quality animal study or several low-quality animal studies indicate carcinogenicity. If there is insufficient human and animal evidence to determine the carcinogenicity of the chemical, it is classified as D. A chemical conclusively demonstrated to be non-carcinogenic to humans is in group E. This designation is rare due to the difficulty in producing the necessary negative data.

RfDs for non-carcinogenic toxicity and slope factors for carcinogenic toxicity were obtained from the USEPA on-line IRIS database, and the USEPA Health Effects Assessment Summary Tables HEAST, FY-1995.

Table 4.2: Slope Factor (SF) for Carcinogenic Chemicals of Concern

Chemical	W01E	Slope Factor/Unit Risk ¹		Type of Cancer	Study Source of SF	Reference for SF	
		Inhalation (mg/kg-day) ⁻¹	Ingestion (ug/L) ⁻¹ (mg/kg-day) ⁻¹				Inhalation / Ingestion
Benzene (BNZ)	A	2.9E-2 [8.3E-7]	2.9E-2 [8.3E-6]	Lymphomas / Leukemia	Human / Human	IRIS / HEAST	
Bromodichloromethane (THM) (BDCM)	B2	—	6.2E-2 [1.8E-6]	Kidney Tumor / Liver Carcinoma	Rat / Mouse	IRIS / IRIS	
Chloroform (THM) (CLFM)	B2	6.1E-3 [1.7E-7]	8.1E-2 [2.3E-5]	Kidney and Liver Carcinoma / —	Rat / Mouse	IRIS / HEAST	
1,1-Dichloroethylene (DCE)	C	6.0E-1 [1.7E-5]	1.8E-1	Liver Tumor / Kidney and Mammary Cancers, Leukemia, Lung Tumor	Rat / Mouse	IRIS / HEAST	
1,2-Dichloropropane (DCP2)	B2	6.8E-2 [1.9E-6]	—	Mammary and Liver Carcinoma / —	Mouse / —	HEAST / —	
Tetrachloroethylene (PCE)	B2	5.1E-2 [1.5E-6]	1.8E-3 [5.2E-7]	Liver Cancer / Liver Tumor	Mouse / Rat, Mouse	HEAST / HEAST	
Trichloroethylene (TCE)	B2	1.1E-2 [3.2E-7]	1.7E-2 [1.7E-6]	Liver Carcinoma / Lung Adenocarcinoma	Mouse / Mouse	HEAST / HEAST	

5.0 RISK CHARACTERIZATION

Inhalation risks from subsurface soil contamination are evaluated in this chapter using exposure and toxicology information previously discussed. The risk characterization is presented in a quantitative and qualitative format.

5.1 Risk Estimation Methods

Risk estimation methods used in this report were based on USEPA guidelines.

5.1.1 Calculation of Carcinogenic Risk

Carcinogenic risk is calculated as the incremental probability of an individual developing cancer over a lifetime (70 years), due to exposure to a carcinogenic compound. This is also referred to as incremental or excess lifetime cancer risk (ELCR) and represents the increased risk of developing cancer above the background rate, estimated to be about 3E-1 (30%).

Estimates of ELCR were based on calculations developed in the following order. Information on exposure pathways, exposure concentrations, and toxicology was assembled or calculated. Chronic daily intakes (CDI) were then calculated using assumptions from the exposure and toxicity reviews presented in Chapters 3 and 4. Chemical specific carcinogenic slope factors (SF), were used to convert estimated CDI, averaged over a lifetime, to ELCR.

The dose-response relationship is considered to be linear under the low dose conditions usually encountered in environmental exposures. Under this assumption, the SF is a constant, and risk is directly related to intake. Therefore, the linear low-dose cancer risk equation is:

$$\text{Risk} = \text{CDI} \times \text{SF}$$

where:

Risk = a unitless probability of an individual developing cancer;

CDI = Chronic daily intake (dose) averaged over 70 years (mg/kg-day);

SF = Slope Factor, expressed in (mg/kg-day)⁻¹.

The SF usually represents an upper 95th percentile confidence limit of the probability of response, based on experimental animal data. Therefore, the risk estimate will also be an upper-bound estimate and *true risk* is likely to be less than predicted by this model.

For known or suspected carcinogens, the USEPA considers exposure levels that present an excess lifetime cancer risk to an individual of between 1E-4 to 1E-6 to be within the acceptable range of risk. Risk estimates less than 1E-6 (one-in-one-million) are considered negligible.

5.1.2 Noncarcinogenic Effects

Noncarcinogenic effects include neurotoxic, hepatotoxic, nephrotoxic, teratogenic, reproductive reactions, and any other noncancer related systemic toxic responses. The potential for an individual suffering a noncarcinogenic effect is not expressed as a probability, but as a ratio or quotient. The hazard quotient (HQ) is the ratio of an exposure level over a specified period (CDI) to the chemical specific reference dose (RfD) which is not expected to produce toxic effects over the period of concern. The HQ is calculated as follows:

$$\text{Noncancer Hazard Quotient} = \text{CDI/RfD}$$

CDI = Daily intake (dose) in mg/kg-day;

RfD = reference dose in mg/kg-day.

The HQ is not expressed as a probability. If the HQ exceeds 1 the possibility that exposed individuals may experience adverse health effects cannot be ruled out. The higher the HQ, the greater the concern. Effects can be evaluated over three time periods; short term, usually less than 2 weeks (acute), 2 weeks to 7 years (subchronic), and more than 7 years (chronic). In this assessment only chronic exposures were evaluated.

5.2 Risk Analysis Under Current Conditions

ELCR and HQ estimates were made in order to evaluate the potential health risks that may be presented by inhalation of vapors diffusing through soil and vapor barriers. A model was used that estimates air concentrations of solvents as they diffuse through soil and mix with outdoor air. The model uses the 95% UCL of the flux estimates to calculate exposure concentrations.

5.2.1 Occupational Exposure at Sandy's Cleaners

All soil and soil vapor samples collected during the environmental investigations conducted thus far have been directly to the west of Sandy's Cleaners, at the rear of the facility. No soil or soil vapor samples have been collected beneath the foundation. Therefore, insufficient data exists to estimate indoor air concentrations under current conditions, and no risk or noncancer hazard estimates may be made for this exposure scenario. A risk analysis that relates various levels of vapor contamination to potential human risk is expected to be developed for this facility in the future.

5.2.2 Occupational Exposure at Other Shopping Center Businesses

All soil and soil vapor samples collected during the environmental investigations conducted thus far have been directly to the west of Sandy's Cleaners, at the rear of the facility. No soil or soil vapor samples have been collected beneath the foundation. Therefore, insufficient data exists to estimate indoor air concentrations, and no risk or noncancer hazard estimates may be made for this exposure scenario.

5.2.3 Transient Exposure to Shoppers

All soil and soil vapor samples collected during the environmental investigations conducted thus far have been directly to the west of Sandy's Cleaners, at the rear of the facility. No soil or soil vapor samples have been collected in front of the building where shoppers would be more likely to be exposed to solvent vapors. However, the nature of the release is such that solvent vapor concentrations in soils would be expected to be higher behind the building than in front of the building. Therefore, conservative risk estimates for the front of the building may be made using existing data.

Risks to shoppers were evaluated using the transient exposure scenario presented in Section 3.4.1. Shoppers were assumed to be exposed to solvent vapors diffusing through cracks in the pavement one hour per week for five years. The concentrations of the COCs in soil vapor were assumed to be equivalent to those behind the facility. The reasonable maximum ELCR estimate for this exposure scenario was $1E-12$ (one-in-one-trillion) which is considered negligible. The noncancer Hazard Quotient was much less than one, indicating that noncancer health effects would not be expected.

5.2.4 Residential Exposure West of Sandy's Cleaners

All soil and soil vapor samples collected during the environmental investigations conducted thus far have been directly to the west of Sandy's Cleaners, at the rear of the facility. No soil or soil vapor samples have been collected in the residential neighborhood to the west of the facility. However, data available from

SMW-1 (located directly behind the facility) indicate that shallow groundwater (20 feet bgs) is contaminated with PCE and other solvents. Data from this well may therefore be used to estimate flux and exposure in the residential neighborhood behind the facility.

Exposure estimates for off-site residential exposure assume steady-state conditions. Soil gas concentrations were calculated by converting the solvent concentration in water from SMW-1 to a concentration in soil gas at the interface of the groundwater and dry alluvium. The conversion was made by multiplying the 95% UCL of the chemical concentration in groundwater by the chemical-specific dimensionless Henry's Law constant (H'). Flux and exposure concentrations were estimated using the equations outlined in Section 3.3.1.

Residential risks were evaluated using the standard default exposures and diffusion assumptions presented in Section 3.4.1. Residents were assumed to be exposed to solvent vapors diffusing through vapor barriers and into indoor air 350 days per year for 30 years. Estimates were also made for risks from exposure to outside air. The reasonable maximum ELCR estimate from exposure indoors was $3E-7$ (three-in-ten-million) which is considered negligible. The noncancer Hazard Quotient was much less than one, indicating that noncancer health effects would not be expected. Outdoor air risks were substantially less than indoor estimates.

5.2.5 Groundwater

At present there are no population groups exposed to shallow groundwater contamination originating from Sandy's Cleaners. There are 2 private wells and 4 irrigation wells near the affected area within 1 mile downgradient of the contamination according to the most recent available data. There are no public drinking water supply wells operating in the affected shallow aquifer.

5.3 Risk Analysis Under Potential Future Conditions

5.3.1 Occupational Exposure at Sandy's Cleaners

All soil and soil vapor samples collected during the environmental investigations conducted thus far have been directly to the west of Sandy's Cleaners, at the rear of the facility. No soil or soil vapor samples have been collected beneath the foundation. Therefore, insufficient data exists to estimate indoor air concentrations, and no risk or noncancer hazard estimates may be made for this exposure scenario.

5.3.2 Occupational Exposure at Other Shopping Center Businesses

All soil and soil vapor samples collected during the environmental investigations conducted thus far have been directly to the west of Sandy's Cleaners, at the rear of the facility. No soil or soil vapor samples have been collected beneath the foundation. Therefore, insufficient data exists to estimate indoor air concentrations, and no risk or noncancer hazard estimates may be made for this exposure scenario.

5.3.3 Transient Exposure to Shoppers

All soil and soil vapor samples collected during the environmental investigations conducted thus far have been directly to the west of Sandy's Cleaners, at the rear of the facility. No soil or soil vapor samples have been collected in front of the building where shoppers would be more likely exposed to solvent vapors. However, the nature of the release is such that solvent vapor concentrations in soils would be expected to be higher behind the building than in front of the building. Therefore, conservative risk estimates for the front of the building may be made using existing data.

Risks to shoppers were evaluated using the transient exposure scenario presented in Section 3.4.1. Shoppers were assumed to be exposed to solvent vapors diffusing through cracks in the pavement one hour per week for five years. The concentrations of the COCs in soil vapor were assumed to be equivalent to those behind the facility. The reasonable maximum ELCR estimate for this exposure scenario was $1E-12$ (one-in-one-trillion) which is considered a negligible risk. The noncancer Hazard Quotient was much less than one, indicating that noncancer health effects would not be expected.

5.3.4 Residential Exposure West of Sandy's Cleaners

All soil and soil vapor samples collected during the environmental investigations conducted thus far have been directly to the west of Sandy's Cleaners, at the rear of the facility. No soil or soil vapor samples have been collected in the residential neighborhood to the west of the facility. However, data available from SMW-1 (located directly behind the facility) indicate that shallow groundwater (20 feet bgs) is contaminated with PCE and other solvents. Data from this well may therefore be used to estimate flux and exposure in the residential neighborhood behind the facility.

Future exposure estimates for off site residential exposure assume steady-state conditions, and were calculated by converting the concentration in water to a concentration in soil gas at the interface of the groundwater and dry alluvium. The concentration in groundwater was converted to a soil gas concentration by multiplying the 95% UCL of the chemical concentration in groundwater by the chemical-specific

dimensionless Henry's Law constant (H'). Flux and exposure concentrations were then estimated using the equations outlined in Section 3.3.1.

Residential risks were evaluated using the standard default exposures and diffusion assumptions presented in Section 3.4.1. Residents were assumed to be exposed to solvent vapors diffusing through vapor barriers and into indoor air 350 days per year for 30 years. Estimates were also made for risks from exposure to outside air. The reasonable maximum ELCR estimate from exposure indoors was 3E-7 (three-in-ten-million) which is considered negligible. The noncancer Hazard Quotient was much less than one, indicating that noncancer health effects would not be expected. Outdoor air risks were substantially less than indoor estimates.

5.3.5 Residential Exposure to Contaminated Groundwater

At present there are no population groups exposed to shallow groundwater contamination originating from Sandy's Cleaners. There are 2 private wells and 4 irrigation wells near the affected area that have not been influenced by the contamination according to the most recent available data. Three of these wells are downgradient from the contaminated portion of the aquifer. There are no public drinking water supply wells operating in the effected shallow aquifer.

Data from monitor well SMW-1 indicate that groundwater immediately downgradient of Sandy's Cleaners has been contaminated with PCE and its biodegradation products. SMW-3, which is located approximately 600 feet to the south-west (downgradient) of the facility has had one low level detection PCE. Other nearby monitor wells installed to investigate a former leaking underground fuel tank have also had samples that contain low levels of PCE. PCE in these wells may or may not be attributable to releases from Sandy's Cleaners. Monitor well SMW-2 and an SRP irrigation well located approximately 800 feet to the east-northeast (upgradient) of the facility has not had any detections of PCE.

While there are no currently complete exposure routes to water from wells in the impacted area, there are no institutional controls preventing the installation of wells within the plume area. It is therefore reasonable to assume that water may be drawn from contaminated portions of this aquifer in the future.

This risk assessment uses analytical data from monitor wells to estimate exposure concentrations at a potential future exposure point. Current concentrations are used to represent potential future concentrations assuming steady-state conditions.

Well SMW-1

The site-related ELCR in groundwater would range from 3E-04 (three-in-ten-thousand) for central tendency residential exposure to 2E-03 (two-in-one-thousand) for RME. Both of these risk estimates are in excess of the acceptable range of risk established by the USEPA. The Hazard Index (HI), which evaluates noncancer health effects, would be 5 under central tendency exposure conditions and 8 under an RME scenario, indicating the potential for systemic health effects if water from this monitor well were used for domestic purposes.

Well SMW-2

No COCs have been identified in SMW-2, and no site-related risk would exist in the future if it remains unaffected.

Well SMW-3

The site-related ELCR in groundwater would range from 5E-07 (five-in-ten-million) for central tendency residential exposure to 5E-06 (five-in-one-million) for RME. The central tendency risk estimate is considered negligible, and the RME estimate is within the acceptable range of risk established by the USEPA. The Hazard Index (HI), which evaluates noncancer health effects, would be less than one under both scenarios, indicating that systemic health effects would be unlikely if water from this well were used for domestic purposes.

Wells AE-1 through AE-8

Site-related risk estimates for these wells would range from 7E-07 (seven-in-ten-million) to 2E-06 (two-in-one-million) for central tendency residential exposure. RME risk estimates would range from 4E-06 (four-in-one-million) to 1E-05 (one-in-one-hundred-thousand). Both of these risk estimates are within the acceptable range of risk established by the USEPA. The Hazard Index (HI), which evaluates noncancer health effects, was less than 1 under both scenarios for each of the wells, indicating that noncancer health effects would be unlikely if the water were used for drinking.

SRP Irrigation Wells 18.5E-7.5N and 19E-7.6N

Site-related risk estimates for well 18.5E-7.5N would range from 1E-07 (one-in-ten-million) for central tendency exposure to 7E-07 (seven-in-ten-million) for reasonable maximum exposure. Site-related risk

estimates for well 19E-7.6N would range from 1E-08 (one-in-one-hundred-million) for central tendency exposure to 6E-08 (six-in-one-hundred-million) for reasonable maximum exposure.

5.4 Uncertainties in the Risk Characterization

All risk estimates are based on a number of assumptions regarding contaminant concentrations, exposures, and toxicity information. Uncertainty is present at all stages in this process. Care is taken at each step in the process to insure that the assumptions made are upper-bound estimates.

Risk and hazard estimates are based on dose-response relationships observed, primarily, in experimental animals. This introduces several sources of uncertainty into the final estimates that are used to characterize risk. There may be differences between animals and humans in metabolic response to a chemical. The test animals may have genetic predispositions that are not considered. High doses are administered to small populations and then low dose response is estimated by extrapolation. Experimental animals have naturally short life spans, whereas humans do not. The toxicity values used were developed singly and responses may differ when complex mixtures are present.

5.5 Summary

The objective of this risk analysis is to provide an evaluation of health risks that may result from exposure to solvents present in subsurface soils and groundwater in the vicinity of Sandy's Magic Touch Cleaners. The results indicate that, under current conditions, the subsurface soil contamination from the facility likely presents a negligible risk to shoppers and residents directly to the west of the facility. *Insufficient data were available to estimate risks to employees from exposure to indoor vapors at Sandy's Cleaners and the adjoining businesses.*

At present there are no known population groups exposed to shallow groundwater contamination originating from Sandy's Cleaners. There is one private well identified as the "Peterson" well, and 4 irrigation wells near the affected area that have not been influenced by the contamination according to data collected through 1996. Three of these wells are downgradient from the contaminated portion of the aquifer.

Data from a monitor well called SMW-1 indicates that groundwater immediately downgradient of Sandy's Cleaners has been contaminated with tetrachloroethylene and its biodegradation products. Another monitor well (SMW-3), which is located approximately 600 feet to the south-west of the facility has had a detection of tetrachloroethylene at a concentration of 7.5 ug/L. Other nearby monitor wells installed to investigate a former leaking underground fuel tank have also had samples that contain tetrachloroethylene at concentrations ranging from 7 to 21 ug/L. Tetrachloroethylene in these wells may be attributable to releases

from Sandy's Cleaners since they are screened in the shallow aquifer and only approximately 400 feet from the facility. Monitor well SMW-2 and a Salt River Project irrigation well located approximately 800 feet to the east-northeast (upgradient) of the facility has not had any detections of tetrachloroethylene, further suggesting that releases from the facility may be responsible for the tetrachloroethylene contamination in the shallow aquifer.

While there are no known currently complete exposure routes to water from wells in the impacted area, there are no institutional controls preventing the installation of wells within the plume area. It is therefore reasonable to assume that water may be drawn from contaminated portions of this aquifer in the future. Potential future risks were evaluated using a conservative methodology that assumes steady-state conditions that do not consider attenuation and biodegradation.

The site-related excess lifetime cancer risk from using groundwater from SMW-1 for domestic purposes would range from 3E-04 (three-in-ten-thousand) for central tendency residential exposure to 2E-03 (two-in-one-thousand) for reasonable maximum exposure. Both of these risk estimates are in excess of the acceptable range of risk of 1E-4 to 1E-6 (one-in-ten-thousand to one-in-one-million) established by the United States Environmental Protection Agency. The Hazard Index, which evaluates noncancer health effects, would be 5 under central tendency exposure conditions and 8 under a reasonable maximum exposure scenario, indicating the potential for systemic health effects if water from this monitor well were used for domestic purposes.

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Appendix

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	WoE HBGL	MCL
Water Sample										
Sample Site: 18.5E-7.5N Usage:										
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L	0.32	0.47	0.27	0.1	1.34	3/ 16	18.8% ✓	B2 7E-01 5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L	0.36	0.53	0.3	0.17	1.2	3/ 15	20.0% --	D 6E+02 2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L	2.1	3.3	2.2	0.7	9.35999999	11/ 15	68.8% ✓	B2 3E+00 5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the WoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	MOE HBGL	MCL
Water Sample										
Sample Site: 19E-7.6N Usage:										
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L					0/ 12	0.0%	-- B2 7E-01	5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L				0/ 11	0.0%	-- D	6E+02	2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L	0.33	0.5	0.26	1.2	1/ 12	8.3%	✓ B2 3E+00	5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the MOE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	WoE HBGL	MCL
Water Sample										
Sample Site: 19E-8.1N Usage:										
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L					0/ 8	0.0%	-- B2	7E-01 5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L	0.41	0.67	0.28	0.6	2/ 7	28.6%	-- D	6E+02 2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 8	0.0%	-- B2	3E+00 5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the WoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	MoE HBGL	MCL
Water Sample										
Sample Site: AB-1	Usage:									
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L	6.8	65	6.5	13.3	1/ 2	50.0% ✓	B2	7E-01 5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/ 2	0.0% --	D	6E+02 2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 2	0.0% --	B2	3E+00 5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the MoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	WoE HBGL	MCL
Water Sample										
Sample Site: AE-2 Usage:										
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L	5.6	53	5.3	10.9	1/ 2	50.0% ✓	B2 7E-01	5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/ 2	0.0% --	D 6E+02	2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 2	0.0% --	B2 3E+00	5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the WoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	WoE HBGL	MCL
Water Sample										
Sample Site: AE-3										
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L	6.4	61	6.1	12.5	1/ 2	50.0% ✓	B2 7E-01	5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/ 2	0.0% --	D 6E+02	2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 2	0.0% --	B2 3E+00	5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the WoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	MoE	HBGL	MCL
Water Sample											
Sample Site: AE-4	Usage:										
O R G A N I C											
1. Tetrachloroethylene (PCE)	127-18-4	µg/L	3.8	36	3.6	7.4	1/ 2	50.0%	✓	B2 7E-01	5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/ 2	0.0%	-- D	6E+02	2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 2	0.0%	-- B2	3E+00	5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the MoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	Woe HBGL	MCL
Water Sample										
Sample Site: AE-5										
Usage:										
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L	6.8	66	6.6	13.4	1/ 2	50.0% ✓	B2 7E-01	5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/ 2	0.0% --	D 6E+02	2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 2	0.0% --	B2 3E+00	5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the Woe is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	WoE HBGL	MCL
Water Sample										
Sample Site: AE-6										
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L	11	100	10	21.2	1/ 2	50.0% ✓	B2 7E-01	5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/ 2	0.0% --	D 6E+02	2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 2	0.0% --	B2 3E+00	5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the WoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	Woe HBGL	MCL
Water Sample										
Sample Site: AE-7 Usage:										
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L	10	100	10	20.2	1/ 2	50.0% ✓	B2 7E-01	5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/ 2	0.0% --	D 6E+02	2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 2	0.0% --	B2 3E+00	5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the Woe is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	WoE HBGL	MCL
Water Sample										
Sample Site: AE-8	Usage:									
O R G A N I C										
1. Benzene (BNZ)	71-43-2	µg/L					0/1	0.0%	-- A	1E+00 5E+00
2. Bromodichloromethane (THM) (BDCM)	75-27-4	µg/L					0/1	0.0%	-- B2	6E-01 1E+02
3. Bromoform (THM) (BRFM)	75-25-2	µg/L					0/1	0.0%	-- B2	4E+00 1E+02
4. Bromomethane (BM4)	74-83-9	µg/L					0/1	0.0%	-- D	1E+01
5. Carbon tetrachloride (CCl4)	56-23-5	µg/L					0/1	0.0%	-- B2	3E-01 5E+00
6. Chlorobenzene (monochlorobenzene) (MCB)	108-90-7	µg/L					0/1	0.0%	-- D	1E+02 1E+02
7. Chloroethane (CE)	75-00-3	µg/L					0/1	0.0%	-- ND	
8. 2-Chloroethylvinyl ether (CEVE)	110-75-8	µg/L					0/1	0.0%	-- ND	
9. Chloroform (THM) (CLFM)	67-66-3	µg/L	1.9	1.9	0.00000001	1.9	1/1	100.0%	✓ B2	6E+00 1E+02
10. Chloromethane (CM)	74-87-3	µg/L					0/1	0.0%	-- C	3E+00
11. Dibromochloromethane (THM) (DBCM)	124-48-1	µg/L					0/1	0.0%	-- C	4E-01 1E+02
12. 1,2-Dichlorobenzene (DCB2)	95-50-1	µg/L					0/1	0.0%	-- D	6E+02 6E+02
13. 1,3-Dichlorobenzene (DCB3)	541-73-1	µg/L					0/1	0.0%	-- D	6E+02
14. 1,4-Dichlorobenzene (DCB4)	106-46-7	µg/L					0/1	0.0%	-- C	2E+00 8E+01
15. Dichlorodifluoromethane (DCDFM)	75-71-8	µg/L					0/1	0.0%	-- D	1E+03
16. 1,1-Dichloroethane (DCA)	75-34-3	µg/L					0/1	0.0%	-- C	7E+01
17. 1,2-Dichloroethane (DCA2)	107-06-2	µg/L					0/1	0.0%	-- B2	4E-01 5E+00
18. 1,1-Dichloroethylene (DCE)	75-35-4	µg/L					0/1	0.0%	-- C	6E-02 7E+00
19. cis-1,2-Dichloroethylene	156-59-2	µg/L					0/1	0.0%	-- D	7E+01 7E+01
20. trans-1,2-Dichloroethylene	156-60-5	µg/L					0/1	0.0%	-- D	1E+02 1E+02
21. Dichloromethane (DCM)	75-09-2	µg/L					0/1	0.0%	-- B2	5E+00 5E+00
22. 1,2-Dichloropropane (DCP2)	78-87-5	µg/L					0/1	0.0%	-- B2	5E+00
23. cis-1,3-Dichloropropene (cDCP3)	10061-01-5	µg/L					0/1	0.0%	-- B2	7E+02 7E+02
24. trans-1,3-Dichloropropene (tDCP3)	10061-02-6	µg/L					0/1	0.0%	-- B2	7E+02 7E+02
25. Ethylbenzene (ETB)	100-41-4	µg/L					0/1	0.0%	-- B2	7E+02 7E+02
26. 1,1,2,2-Tetrachloroethane (TET)	79-34-5	µg/L	5.6	20	9.3	21.8	1/4	25.0%	✓ B2	7E-01 5E+00
27. Tetrachloroethylene (PCE)	127-18-4	µg/L					0/1	0.0%	-- D	1E+03 1E+03
28. Toluene (TOL)	108-88-3	µg/L					0/4	0.0%	-- D	6E+02 2E+02
29. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/1	0.0%	-- C	6E-01 5E+00
30. 1,1,2-Trichloroethane (TCA2)	79-00-5	µg/L					0/4	0.0%	-- B2	3E+00 5E+00
31. Trichloroethylene (TCE)	79-01-6	µg/L					0/1	0.0%	-- D	2E+03
32. Trichlorofluoromethane (TCFM)	75-69-4	µg/L					0/1	0.0%	-- A	2E-02 2E+00
33. Vinyl chloride (VC)	75-01-4	µg/L					0/1	0.0%	-- A	2E-02 2E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the WoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	WOE HBGL	MCL
Water Sample										
Sample Site: AE-8										
Usage:										
O R G A N I C										
34. Xylenes (total) (XYL)	1330-20-7	µg/L					0/ 1	0.0%	-- D	1E+04 1E+04

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the WOE is not "A", "B1" or "E2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	Woe HBGL	MCL
Water Sample										
Sample Site: DM-1										
Usage:										
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L					0/ 1	0.0%	-- E2 7E-01	5E+00
2. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 1	0.0%	-- E2 3E+00	5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the Woe is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	Woe HBGL	MCL
Water Sample										
Sample Site: MCFC#8										
Usage:										
O R G A N I C										
1. Tetrachloroethylene (PCE)	127-18-4	µg/L						0/ 1	0.0%	-- B2 7E-01 5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L						0/ 1	0.0%	-- D 6E+02 2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L						0/ 1	0.0%	-- B2 3E+00 5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the WOE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	WoE	HBGL	MCL
Water Sample											
Sample Site: Peterson Usage:											
O R G A N I C											
1. Tetrachloroethylene (PCE)	127-18-4	µg/L					0/ 2	0.0%	-- B2	7E-01	5E+00
2. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/ 2	0.0%	-- D	6E+02	2E+02
3. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 2	0.0%	-- B2	3E+00	5E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the WoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest	Detects	Det %	WoE	HBGL	MCL
Water Sample												
Sample Site: SMW1	Usage:											
O R G A N I C												
1. Benzene (BNZ)	71-43-2	µg/L						0/ 6	0.0%	-- A	1E+00	5E+00
2. Bromobenzene	108-86-1	µg/L						0/ 4	0.0%	-- ND		
3. Bromodichloromethane (THM) (BDCM)	75-27-4	µg/L						0/ 6	0.0%	-- B2	6E-01	1E+02
4. Bromoform (THM) (BRFM)	75-25-2	µg/L						0/ 6	0.0%	-- B2	4E+00	1E+02
5. Bromomethane (BM)	74-83-9	µg/L						0/ 6	0.0%	-- D	1E+01	
6. Carbon tetrachloride (CCl4)	56-23-5	µg/L						0/ 6	0.0%	-- B2	3E-01	5E+00
7. Chlorobenzene (monochlorobenzene) (MCB)	108-90-7	µg/L						0/ 6	0.0%	-- D	1E+02	1E+02
8. Chloroethane (CE)	75-00-3	µg/L						0/ 6	0.0%	-- ND		
9. 2-Chloroethylvinyl ether (CEVE)	110-75-8	µg/L						0/ 2	0.0%	-- ND		
10. Chloroform (THM) (CLFM)	67-66-3	µg/L						0/ 6	0.0%	-- B2	6E+00	1E+02
11. Chloromethane (CM)	74-87-3	µg/L						0/ 6	0.0%	-- C	3E+00	
12. o-Chlorotoluene	95-49-8	µg/L						0/ 4	0.0%	-- D	1E+02	
13. para-Chlorotoluene	106-43-4	µg/L						0/ 4	0.0%	-- ND		
14. Dibromochloromethane (THM) (BDCM)	124-48-1	µg/L						0/ 6	0.0%	-- C	4E-01	1E+02
15. 1,2-Dichlorobenzene (DCB2)	95-50-1	µg/L						0/ 6	0.0%	-- D	5E+02	6E+02
16. 1,3-Dichlorobenzene (DCB3)	541-73-1	µg/L						0/ 6	0.0%	-- D	6E+02	
17. 1,4-Dichlorobenzene (DCB4)	106-46-7	µg/L						0/ 6	0.0%	-- C	2E+00	8E+01
18. Dichlorodifluoromethane (DCDFM)	75-71-8	µg/L	30	70	4.5	34	34	1/ 2	50.0%	-- D	1E+03	
19. 1,1-Dichloroethane (DCA)	75-34-3	µg/L						0/ 6	0.0%	-- C	7E+01	
20. 1,2-Dichloroethane (DCA2)	107-06-2	µg/L						0/ 6	0.0%	-- B2	4E-01	5E+00
21. 1,1-Dichloroethylene (DCE)	75-35-4	µg/L	8	17	8.9	9	9	1/ 6	16.7%	✓ C	6E-02	7E+00
22. cis-1,2-Dichloroethylene	156-59-2	µg/L						0/ 6	0.0%	-- D	1E+02	1E+01
23. trans-1,2-Dichloroethylene	156-60-5	µg/L	7.7	17	8.9	7	7	1/ 6	16.7%	-- D	1E+02	1E+02
24. Dichloromethane (DCM)	75-09-2	µg/L						0/ 6	0.0%	-- B2	5E+00	5E+00
25. 1,2-Dichloropropane (DCP2)	78-87-5	µg/L	9.69999999	23	12	29	29	1/ 6	16.7%	✓ B2	5E-01	5E+00
26. 1,3-Dichloropropane	142-28-9	µg/L						0/ 4	0.0%	-- ND		
27. 2,2-Dichloropropane		µg/L						0/ 4	0.0%	-- ND		
28. 1,1-Dichloropropene	563-58-6	µg/L						0/ 4	0.0%	-- ND		
29. cis-1,3-Dichloropropene (cDCP3)	10061-01-5	µg/L						0/ 6	0.0%	-- B2		
30. trans-1,3-Dichloropropene (tDCP3)	10061-02-6	µg/L						0/ 2	0.0%	-- B2		
31. Ethylbenzene (EB)	100-41-4	µg/L						0/ 6	0.0%	-- D	7E+02	7E+02
32. Ethylene dibromide (EDB)	106-93-4	µg/L						0/ 4	0.0%	-- B2	4E-04	5E-02
33. Styrene	100-42-5	µg/L						0/ 4	0.0%	-- C	1E+02	1E+02

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the WoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	Woe HBGL	MCL
Water Sample										
Sample Site: SMW1	Usage:									
O R G A N I C										
34. 1,1,1,2-Tetrachloroethane	630-20-6	µg/L					0/ 4	0.0%	-- C	1E+00
35. 1,1,2,2-Tetrachloroethane (TET)	79-34-5	µg/L					0/ 6	0.0%	-- C	2E-01
36. Tetrachloroethylene (PCE)	127-18-4	µg/L	1800	3100	1400	220	7/ 7	100.0%	✓ B2	7E-01 5E+00
37. Toluene (TOL)	108-88-3	µg/L					0/ 6	0.0%	-- D	1E+03 1E+03
38. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/ 7	0.0%	-- D	6E+02 2E+02
39. 1,1,2-Trichloroethane (TCA2)	79-00-5	µg/L	18	39	23	35	1/ 7	14.3%	✓ B2	3E+00 5E+00
40. Trichloroethylene (TCE)	79-01-6	µg/L					0/ 2	0.0%	-- D	2E+03
41. Trichlorofluoromethane (TCFM)	75-69-4	µg/L					0/ 4	0.0%	-- D	5E-03
42. 1,2,3-Trichloropropane	96-18-4	µg/L					0/ 6	0.0%	-- A	2E-02 2E+00
43. Vinyl chloride (VC)	75-01-4	µg/L					0/ 2	0.0%	-- D	1E+04 1E+04
44. Xylenes (total) (XYL)	1330-20-7	µg/L								

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the Woe is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest	Detects	Det %	Woe	HBGL	MCL
Water Sample												
Sample Site: SMW2	Usage:											
O R G A N I C												
1. Benzene (BNZ)	71-43-2	µg/L						0/ 3	0.0%	-- A	1E+00	5E+00
2. Bromobenzene	108-86-1	µg/L						0/ 1	0.0%	-- ND		
3. Bromodichloromethane (THM) (BDCM)	75-27-4	µg/L						0/ 3	0.0%	-- B2	6E-01	1E+02
4. Bromoform (THM) (BRFM)	75-25-2	µg/L						0/ 3	0.0%	-- B2	4E+00	1E+02
5. Bromomethane (BM)	74-83-9	µg/L						0/ 3	0.0%	-- D	1E+01	
6. Carbon tetrachloride (CCL4)	56-23-5	µg/L						0/ 3	0.0%	-- B2	3E-01	5E+00
7. Chlorobenzene (monochlorobenzene) (MCB)	108-90-7	µg/L						0/ 3	0.0%	-- D	1E+02	1E+02
8. Chloroethane (CE)	75-00-3	µg/L						0/ 3	0.0%	-- ND		
9. 2-Chloroethylvinyl ether (CEVE)	110-75-8	µg/L						0/ 2	0.0%	-- ND		
10. Chloroform (THM) (CLFM)	67-66-3	µg/L						0/ 3	0.0%	-- B2	6E+00	1E+02
11. Chloromethane (CM)	74-87-3	µg/L						0/ 3	0.0%	-- C	3E+00	
12. o-Chlorotoluene	95-49-8	µg/L						0/ 1	0.0%	-- D	1E+02	
13. para-Chlorotoluene	106-43-4	µg/L						0/ 1	0.0%	-- ND		
14. Dibromochloromethane (THM) (DBCM)	124-48-1	µg/L						0/ 3	0.0%	-- C	4E-01	1E+02
15. 1,2-Dichlorobenzene (DCB2)	95-50-1	µg/L						0/ 3	0.0%	-- D	6E+02	6E+02
16. 1,3-Dichlorobenzene (DCB3)	541-73-1	µg/L						0/ 3	0.0%	-- D	6E+02	
17. 1,4-Dichlorobenzene (DCB4)	106-46-7	µg/L						0/ 3	0.0%	-- C	2E+00	8E+01
18. Dichlorodifluoromethane (DCDFM)	75-71-8	µg/L						0/ 2	0.0%	-- D	1E+03	
19. 1,1-Dichloroethane (DCA)	75-34-3	µg/L						0/ 3	0.0%	-- C	7E+01	
20. 1,2-Dichloroethane (DCA2)	107-06-2	µg/L						0/ 3	0.0%	-- B2	4E-01	5E+00
21. 1,1-Dichloroethylene (DCE)	75-35-4	µg/L						0/ 3	0.0%	-- C	6E-02	7E+00
22. cis-1,2-Dichloroethylene	156-59-2	µg/L						0/ 3	0.0%	-- D	1E+02	1E+02
23. trans-1,2-Dichloroethylene	156-60-5	µg/L						0/ 3	0.0%	-- D	7E+01	7E+01
24. Dichloromethane (DCM)	75-09-2	µg/L						0/ 3	0.0%	-- B2	5E+00	5E+00
25. 1,2-Dichloropropane (DCP2)	78-87-5	µg/L						0/ 3	0.0%	-- B2	5E-01	5E+00
26. 1,3-Dichloropropane	142-28-9	µg/L						0/ 1	0.0%	-- ND		
27. 2,2-Dichloropropane		µg/L						0/ 1	0.0%	-- ND		
28. 1,1-Dichloropropene	563-58-6	µg/L						0/ 1	0.0%	-- ND		
29. cis-1,3-Dichloropropene (cDCP3)	10061-01-5	µg/L						0/ 3	0.0%	-- B2		
30. trans-1,3-Dichloropropene (tDCP3)	10061-02-6	µg/L						0/ 2	0.0%	-- B2		
31. Ethylbenzene (EB)	100-41-4	µg/L						0/ 3	0.0%	-- D	7E+02	7E+02
32. Ethylene dibromide (EDB)	106-93-4	µg/L						0/ 1	0.0%	-- B2	4E-04	5E-02
33. Styrene	100-42-5	µg/L						0/ 1	0.0%	-- C	1E+02	1E+02

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the Woe is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	Woe HBGL	MCL
Water Sample										
Sample Site: SMW2	Usage:									
ORGANIC										
34. 1,1,1,2-Tetrachloroethane	630-20-6	µg/L						0/ 1	0.0%	-- C 1E+00
35. 1,1,2,2-Tetrachloroethane (TET)	79-34-5	µg/L						0/ 3	0.0%	-- C 2E-01
36. Tetrachloroethylene (PCE)	127-18-4	µg/L						0/ 4	0.0%	-- B2 7E-01 5E+00
37. Toluene (TOL)	108-88-3	µg/L						0/ 3	0.0%	-- D 1E+03 1E+03
38. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L						0/ 4	0.0%	-- D 6E+02 2E+02
39. 1,1,2-Trichloroethane (TCA2)	79-00-5	µg/L						0/ 3	0.0%	-- C 6E-01 5E+00
40. Trichloroethylene (TCE)	79-01-6	µg/L						0/ 4	0.0%	-- B2 3E+00 5E+00
41. Trichlorofluoromethane (TCFM)	75-69-4	µg/L						0/ 2	0.0%	-- D 2E+03
42. 1,2,3-Trichloropropane	96-18-4	µg/L						0/ 1	0.0%	-- D 5E-03
43. Vinyl chloride (VC)	75-01-4	µg/L						0/ 3	0.0%	-- A 2E-02 2E+00
44. Xylenes (total) (XYL)	1330-20-7	µg/L						0/ 2	0.0%	-- D 1E+04 1E+04

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the Woe is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detected	Det %	WoE HRGL	MCL
Water Sample										
Sample Site: SMW3	Usage:									
O R G A N I C										
1. Benzene (BNZ)	71-43-2	µg/L	0.34	0.58	0.15	0.6	1/4	25.0%	A	1E+00 5E+00
2. Bromodichloromethane (THM) (BDCM)	75-27-4	µg/L	0.44	0.95	0.32	1	1/4	25.0%	B2	6E-01 1E+02
3. Bromoform (THM) (BRFM)	75-25-2	µg/L					0/4	0.0%	-- B2	4E+00 1E+02
4. Bromomethane (BMM)	74-83-9	µg/L					0/4	0.0%	-- D	1E+01
5. Carbon tetrachloride (CCl4)	56-23-5	µg/L					0/4	0.0%	-- B2	3E-01 5E+00
6. Chlorobenzene (monochlorobenzene) (MCB)	108-90-7	µg/L					0/4	0.0%	-- D	1E+02 1E+02
7. Chloroethane (CE)	75-00-3	µg/L					0/4	0.0%	-- ND	
8. 2-Chloroethylvinyl ether (CEVE)	110-75-8	µg/L					0/4	0.0%	-- ND	
9. Chloroform (THM) (CLFM)	67-66-3	µg/L	1.9	4.5	1.6	3.2	2/4	50.0%	B2	6E+00 1E+02
10. Chloromethane (CM)	74-87-3	µg/L					0/4	0.0%	-- C	3E+00
11. Dibromochloromethane (THM) (DBCM)	124-48-1	µg/L	0.36	0.67	0.19	0.7	1/4	25.0%	-- C	4E-01 1E+02
12. 1,2-Dichlorobenzene (DCB2)	95-50-1	µg/L					0/4	0.0%	-- D	6E+02
13. 1,3-Dichlorobenzene (DCB3)	541-73-1	µg/L					0/4	0.0%	-- C	2E+00 8E+01
14. 1,4-Dichlorobenzene (DCB4)	106-46-7	µg/L					0/4	0.0%	-- D	1E+03
15. Dichlorodifluoromethane (DCDFM)	75-71-8	µg/L					0/4	0.0%	-- C	7E+01
16. 1,1-Dichloroethane (DCA)	75-34-3	µg/L					0/4	0.0%	-- B2	4E-01 5E+00
17. 1,2-Dichloroethane (DCA2)	107-06-2	µg/L					0/4	0.0%	-- C	6E-02 7E+00
18. 1,1-Dichloroethylene (DCE)	75-35-4	µg/L					0/4	0.0%	-- D	7E+01 7E+01
19. cis-1,2-Dichloroethylene	156-59-2	µg/L					0/4	0.0%	-- D	1E+02 1E+02
20. trans-1,2-Dichloroethylene	156-60-5	µg/L					0/4	0.0%	-- B2	5E+00 5E+00
21. Dichloromethane (DCM)	75-09-2	µg/L					0/4	0.0%	-- B2	5E-01 5E+00
22. 1,2-Dichloropropane (DCP2)	78-87-5	µg/L					0/4	0.0%	-- B2	
23. cis-1,3-Dichloropropene (cDCP3)	10061-01-5	µg/L					0/4	0.0%	-- D	7E+02 7E+02
24. trans-1,3-Dichloropropene (tDCP3)	10061-02-6	µg/L					0/4	0.0%	-- C	2E-01
25. Ethylbenzene (ETB)	100-41-4	µg/L					0/4	0.0%	-- C	2E-01
26. 1,1,2,2-Tetrachloroethane (TET)	79-34-5	µg/L	2.1	7.1	3.1	7.5	1/4	25.0%	B2	7E-01 5E+00
27. Tetrachloroethylene (PCE)	127-18-4	µg/L	0.36	0.67	0.19	0.7	1/4	25.0%	-- D	1E+03 1E+03
28. Toluene (TOL)	108-88-3	µg/L					0/4	0.0%	-- D	6E+02 2E+02
29. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L					0/4	0.0%	-- C	6E-01 5E+00
30. 1,1,2-Trichloroethane (TCA2)	79-00-5	µg/L					0/4	0.0%	-- C	6E-01 5E+00
31. Trichloroethylene (TCE)	79-01-6	µg/L					0/4	0.0%	-- B2	3E+00 5E+00
32. Trichlorofluoromethane (TCFM)	75-69-4	µg/L					0/4	0.0%	-- D	2E+03
33. Vinyl chloride (VC)	75-01-4	µg/L					0/4	0.0%	-- A	2E-02 2E+00

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HRGL or was less than the MCL and the WoE is not "A", "B1" or "B2".

Appendix Table A - Summary for all chemicals by sampling site,
Sandy's Cleaners Risk Assessment

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest Detects	Det %	Woe HBGL	MCL
Water Sample										
Sample Site: SMW3										
Usage:										
ORGANIC										
34. Xylenes (total) (XYL)	1330-20-7	µg/L					0/ 4	0.0%	-- D	1E+04 1E+04

-- Chemical removed from risk analysis because there were no positive detections in the data set or the highest detected value was less than the HBGL or was less than the MCL and the Woe is not "A", "B1" or "B2".

Appendix Table B - Worksheet for calculations of potential CDI, carcinogenic risks, and non-cancer hazard quotients by sampling site, Sandy's Cleaners Risk Assessment

MEAN Concen.	AVERAGE EXPOSURE (MEAN)						95% UCL Concen.	REASONABLE MAXIMUM EXPOSURE (RME)					
	CARCINOGENIC			NON-CARCINOGENIC				CARCINOGENIC			NON-CARCINOGENIC		
	CDI**	RISK	% HZD INDX	CDI**	HZD QTNT	HZD INDX		CDI**	RISK	% HZD INDX	CDI**	HZD QTNT	HZD INDX
Units ug/L							Units ug/L						
0.32	1.1E-06	5.6E-08	41	8.8E-06	8.8E-04		0.47	5.5E-06	2.8E-07	39	1.3E-05	1.3E-03	
2.1	7.4E-06	8.1E-08	59	5.8E-05			3.3	3.9E-05	4.3E-07	61	9.0E-05		
TOTALS w/o Arsenic	--	1E-07	--	--	8.8E-04		TOTALS	--	7E-07	--	--	1.3E-03	
	--	1E-07	--	--	--		w/o Arsenic	--	7E-07	--	--	--	

Water Sample

SITE: 18.5E-7.5N

ORGANIC

Tetrachloroethylene (PCE)

Trichloroethylene (TCE)

* One-half of the reported detection limit or sample quantitation limit was used in calculations for the mean and 95% UCL. ** Units equal mg/kg-day
 * Highest detected concentration was used because the Mean or 95% UCL exceeded the highest detected concentration.

Appendix Table B - Worksheet for calculations of potential CDI, carcinogenic risks, and non-cancer hazard quotients by sampling site.
Sandy's Cleaners Risk Assessment

Water Sample	AVERAGE EXPOSURE (MEAN)						REASONABLE MAXIMUM EXPOSURE (RME)									
	CARCINOGENIC			NON-CARCINOGENIC			CARCINOGENIC			NON-CARCINOGENIC						
	MEAN Concen.	CDI**	RISK	%	HZD INDX	HZD QTNT	CDI**	RISK	%	HZD INDX	HZD QTNT	95% UCL Concen.	CDI**	RISK	HZD QTNT	
ORGANIC Trichloroethylene (TCE)	0.33	1.2E-06	1.3E-08	100			9.0E-06					Units ug/L 0.5	5.9E-06	6.5E-08	100	1.4E-05
TOTALS w/o Arsenic		--	1E-08		--	--	--	--	--	--	--	TOTALS w/o Arsenic	--	6E-08	--	--
		--	1E-08		--	--	--	--	--	--	--		--	6E-08	--	--

SITE: 19E-7.6N

ORGANIC
Trichloroethylene (TCE)

* One-half of the reported detection limit or sample quantitation limit was used in calculations for the mean and 95% UCL.
* Highest detected concentration was used because the Mean or 95% UCL exceeded the highest detected concentration.
** Units equal mg/kg-day

Appendix Table B - Worksheet for calculations of potential CDI, carcinogenic risks, and non-cancer hazard quotients by sampling site, Sandy's Cleaners Risk Assessment

MEAN Concen.	AVERAGE EXPOSURE (MEAN)						95% UCL Concen.	REASONABLE MAXIMUM EXPOSURE (RME)					
	CARCINOGENIC			NON-CARCINOGENIC				CARCINOGENIC			NON-CARCINOGENIC		
	CDI**	RISK	HZD INDX	HZD QTNF	CDI**	HZD QTNF		CDI**	RISK	HZD INDX	CDI**	HZD QTNF	
Units ug/L 5.6	2.0E-05	9.9E-07	100	1.5E-02	1.5E-04	1.5E-02	10.9*	1.3E-04	6.4E-06	100	3.0E-04	3.0E-02	
TOTALS w/o Arsenic	--	1E-06	--	1.5E-02	--	1.5E-02	TOTALS w/o Arsenic	--	6E-06	--	--	3.0E-02	--
	--	1E-06	--	--	--	--		--	6E-06	--	--	--	--

Water Sample

SITE: AR-2

ORGANIC
Tetrachloroethylene (PCE)

* One-half of the reported detection limit or sample quantitation limit was used in calculations for the mean and 95% UCL.
 * Highest detected concentration was used because the Mean or 95% UCL exceeded the highest detected concentration.
 ** Units equal mg/kg-day

Appendix Table B - Worksheet for calculations of potential CDI, carcinogenic risks, and non-cancer hazard quotients by sampling site.
Sandy's Cleaners Risk Assessment

MEAN Concen.	AVERAGE EXPOSURE (MEAN)						REASONABLE MAXIMUM EXPOSURE (RME)					
	CARCINOGENIC			NON-CARCINOGENIC			CARCINOGENIC			NON-CARCINOGENIC		
	CDI**	RISK	HZD INDX	CDI**	HZD QTNT	95% UCL Concen.	CDI**	RISK	HZD INDX	CDI**	HZD QTNT	
Units ug/L 3.8	1.3E-05	6.7E-07	100	1.0E-04	1.0E-02	7.4*	8.7E-05	4.3E-06	100	2.0E-04	2.0E-02	
TOTALS w/o Arsenic	-	7E-07	-	-	1.0E-02	TOTALS w/o Arsenic	-	4E-06	-	-	2.0E-02	
	-	7E-07	-	-	-		-	4E-06	-	-	-	

Water Sample

SITE: AB-4

ORGANIC
Tetrachloroethylene (PCE)

* One-half of the reported detection limit or sample quantitation limit was used in calculations for the mean and 95% UCL.
* Highest detected concentration was used because the Mean or 95% UCL exceeded the highest detected concentration.
** Units equal mg/kg-day

Appendix Table B - Worksheet for calculations of potential CDI, carcinogenic risks, and non-cancer hazard quotients by sampling site, Sandy's Cleaners Risk Assessment

MEAN Concen.	AVERAGE EXPOSURE (MEAN)						95% UCL Concen.	REASONABLE MAXIMUM EXPOSURE (RME)					
	CARCINOGENIC			NON-CARCINOGENIC				CARCINOGENIC			NON-CARCINOGENIC		
	CDI**	RISK	% HZD INDX	HZD QNT	CDI**	HZD QNT		CDI**	RISK	% HZD INDX	CDI**	HZD QNT	
Units ug/L 6.8	2.4E-05	1.2E-06	100	1.9E-02	1.9E-04	1.9E-02	Units ug/L 13.4*	1.6E-04	7.9E-06	100	3.7E-04	3.7E-02	
TOTALS w/o Arsenic	-	1E-06	-	1.9E-02	-	1.9E-02	TOTALS w/o Arsenic	-	8E-06	-	-	3.7E-02	
	-	1E-06	-	-	-	-		-	8E-06	-	-	-	

Water Sample

SITE: AE-5

ORGANIC
Tetrachloroethylene (PCE)

* One-half of the reported detection limit or sample quantitation limit was used in calculations for the mean and 95% UCL.
 * Highest detected concentration was used because the Mean or 95% UCL exceeded the highest detected concentration.
 ** Units equal mg/kg-day

Appendix Table B - Worksheet for calculations of potential CDI, carcinogenic risks, and non-cancer hazard quotients by sampling site, Sandy's Cleaners Risk Assessment

		AVERAGE EXPOSURE (MEAN)					REASONABLE MAXIMUM EXPOSURE (RME)				
		CARCINOGENIC			NON-CARCINOGENIC		CARCINOGENIC			NON-CARCINOGENIC	
MEAN Concn.		CDI**	RISK	HZD INDX	CDI**	HZD QTNT	CDI**	RISK	HZD INDX	CDI**	HZD QTNT
Units ug/L											
11		3.9E-05	1.9E-06	100	3.0E-04	3.0E-02	2.5E-04	1.2E-05	100	5.8E-04	5.8E-02
TOTALS		- -	2E-06	- -	- -	3.0E-02	- -	1E-05	- -	- -	5.8E-02
w/o Arsenic		- -	2E-06	- -	- -	w/o Arsenic	- -	1E-05	- -	- -	- -

Water Sample

SITE: AM-6

ORGANIC
Tetrachloroethylene (PCE)

* One-half of the reported detection limit or sample quantitation limit was used in calculations for the mean and 95% UCL.
 * Highest detected concentration was used because the Mean or 95% UCL exceeded the highest detected concentration.
 ** Units equal mg/kg-day

Appendix Table B - Worksheet for calculations of potential CDI, carcinogenic risks, and non-cancer hazard quotients by sampling site, Sandy's Cleaners Risk Assessment

MEAN Concen.	AVERAGE EXPOSURE (MEAN)						95% UCL Concen.	REASONABLE MAXIMUM EXPOSURE (RME)									
	CARCINOGENIC			NON-CARCINOGENIC				CARCINOGENIC			NON-CARCINOGENIC						
	CDI**	RISK	HZD INDX	CDI**	HZD QTNT	HZD QNTT		CDI**	RISK	HZD INDX	CDI**	HZD QNTT	HZD QNTT				
Units ug/L							Units ug/L										
10	3.5E-05	1.8E-06	100	2.7E-04	2.7E-02	2.7E-02	20.2*	2.4E-04	1.2E-05	100	5.5E-04	5.5E-02					
TOTALS w/o Arsenic	- -	2E-06	- -	- -	2.7E-02	- -	TOTALS w/o Arsenic	- -	1E-05	- -	- -	5.5E-02	- -	1E-05	- -	- -	- -

Water Sample

SITE: AE-7

ORGANIC
Tetrachloroethylene (PCE)

* One-half of the reported detection limit or sample quantitation limit was used in calculations for the mean and 95% UCL.
 * Highest detected concentration was used because the Mean or 95% UCL exceeded the highest detected concentration. ** Units equal mg/kg-day

Appendix Table B - Worksheet for calculations of potential CDI, carcinogenic risks, and non-cancer hazard quotients by sampling site, Sandy's Cleaners Risk Assessment

MEAN Concen.	AVERAGE EXPOSURE (MEAN)						REASONABLE MAXIMUM EXPOSURE (RME)					
	CARCINOGENIC			NON-CARCINOGENIC			CARCINOGENIC			NON-CARCINOGENIC		
	CDI**	RISK	% HZD INDX	CDI**	HZD QTNT	95% UCL Concen.	CDI**	RISK	% HZD INDX	CDI**	HZD QTNT	
Units ug/L						Units ug/L						
1.9	6.7E-06	4.1E-08	4	5.2E-05	5.2E-03	1.9	2.2E-05	1.4E-07	1	5.2E-05	5.2E-03	
5.6	2.0E-05	9.9E-07	96	1.5E-04	1.5E-02	20	2.3E-04	1.2E-05	99	5.5E-04	5.5E-02	
TOTALS w/o Arsenic	- -	1E-06	- -	- -	2.1E-02	TOTALS w/o Arsenic	- -	1E-05	- -	- -	6.0E-02	
	- -	1E-06	- -	- -	- -		- -	1E-05	- -	- -	- -	

Water Sample

SITE: AE-8

ORGANIC

Chloroform (THM) (CLFM)

Tetrachloroethylene (PCE)

* One-half of the reported detection limit or sample quantitation limit was used in calculations for the mean and 95% UCL. ** Units equal mg/kg-day
 * Highest detected concentration was used because the Mean or 95% UCL exceeded the highest detected concentration.

Appendix Table B - Worksheet for calculations of potential CDI, carcinogenic risks, and non-cancer hazard quotients by sampling site, Sandy's Cleaners Risk Assessment

MEAN Concen.	AVERAGE EXPOSURE (MEAN)						REASONABLE MAXIMUM EXPOSURE (RME)					
	CARCINOGENIC			NON-CARCINOGENIC			CARCINOGENIC			NON-CARCINOGENIC		
	CDI**	RISK	% HZD INDX	CDI**	HZD QNT	95% UCL Concen.	CDI**	RISK	% HZD INDX	CDI**	HZD QNT	
Units ug/L						Units ug/L						
8	2.8E-05	1.7E-05	5	2.2E-04	2.4E-02	9*	1.1E-04	6.3E-05	3	2.5E-04	2.7E-02	
9.7	3.4E-05	2.3E-06	1	2.7E-04		23	2.7E-04	1.8E-05	1	6.3E-04		
1800	6.3E-03	3.2E-04	94	4.9E-02	4.9E+00	3100	3.6E-02	1.8E-03	95	8.5E-02	8.5E+00	
18	6.3E-05	7.0E-07		4.9E-04		35*	4.1E-04	4.5E-06		9.6E-04		
TOTALS w/o Arsenic	--	3E-04	--	--	5.0E+00	TOTALS w/o Arsenic	--	2E-03	--	--	9.5E+00	
	--	3E-04	--	--	--		--	2E-03	--	--	--	

Water Sample

SITE: SMW1

ORGANIC
1,1-Dichloroethylene (DCE)
1,2-Dichloropropane (DCP2)
Tetrachloroethylene (PCE)
Trichloroethylene (TCE)

* One-half of the reported detection limit or sample quantitation limit was used in calculations for the mean and 95% UCL.
* Highest detected concentration was used because the Mean or 95% UCL exceeded the highest detected concentration.
** Units equal mg/kg-day

Summary of 1996 Soil Vapor Sampling
Samples for 5 Meters in Depth

Chemical	DP-1-5	DP-2-5	DP-3-5	DP-4-5	DP-5-5D	DP-6-5	DP-7-5	DP-8-5	DP-9-5	DP-10-5	freq	avg (ug/l)	std (ug/l)	95uci (ug/l)
PCE (ug/l)	990	700	270	1200	4200	3200	2300	0.5	2700	2300	10/11	2114.59	1694.30	3252.77
TCE (ug/l)	6.1	2.5	2	12.5	12.5	12.5	12.5	0.5	12.5	12.5	2/11	8.96	5.08	12.37
DCE (ug/l)	16	51	13	12.5	12.5	12.5	12.5	2.7	12.5	12.5	4/11	15.47	12.22	23.68
TCA (ug/l)	1	2.5	0.5	12.5	12.5	12.5	12.5	0.5	12.5	12.5	1/11	8.36	5.76	12.23

Summary of 1996 Soil Vapor Sampling
Samples for 10 Meters in Depth

Chemical	DP-1-10	DP-2-10	DP-3-10	DP-4-10	DP-5-10	DP-6-10D	DP-7-10	DP-8-10	DP-9-10	DP-9-10D	DP-10-5	freq	avg (ug/l)	std (ug/l)	max (ug/l)	95uci (ug/l)
PCE (ug/l)	540	540	260	2100	1600	3600	340	0.5	1100	880	1100	15060.5	1255.04	1127.72	2011.84	1971.56
TCE (ug/l)	2.5	5	2.5	12.5	5	12.5	3.8	0.5	5	5	5	71.8	5.98	4.17	8.78	8.63
DCE (ug/l)	58	69	71	12.5	5	12.5	0.5	4.1	5	5	5	260.1	21.68	27.16	39.90	38.93
TCA (ug/l)	2.5	0.5	2.5	12.5	5	12.5	0.5	5	5	5	5	68.5	5.71	4.43	8.68	8.52

Summary of 1996 Soil Vapor Sampling
Samples for 16 Meters in Depth

Chemical	DP-1-15	DP-2-15	DP-3-15	DP-4-15	DP-4-15D	DP-5-15	DP-6-15	DP-7-15	DP-8-15	DP-9-15	DP-10-15	freq	avg (ug/l)	std (ug/l)	max (ug/l)	95uci (ug/l)
PCE	1600	400	820	11	13	1700	2000	0.5	3100	1500	16744.5	1522.23	1673.73	2645.44	2646.59	
TCE	5	2.5	5	0.5	0.5	11	12.5	0.5	12.5	12.5	75	6.82	5.40	10.44	10.45	
DCE	5	5	86	0.5	0.5	2.5	12.5	1.2	12.5	12.5	150.7	13.70	24.52	30.16	30.17	
TCA	5	2.5	5	0.5	0.5	6.6	12.5	0.5	12.5	12.5	97.1	8.83	11.10	16.28	16.29	

Summary of 1996 Soil Sampling Results
Samples for 7 Meters in Depth

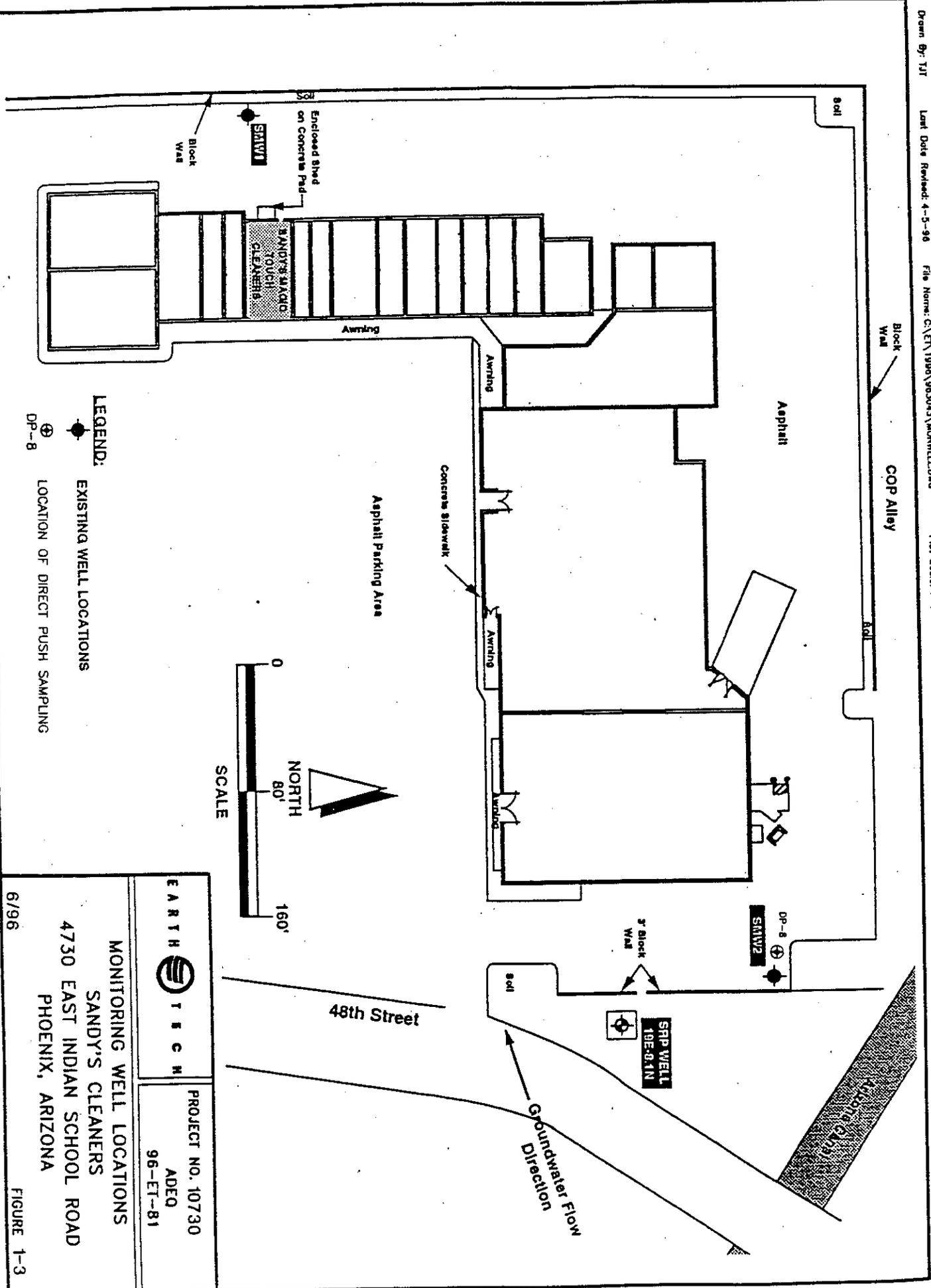
Chemical	DP-1-7	DP-2-7	DP-4-7	DP-4-7D	DP-7-7	DP-9-7	DP-10-7	Totals	freq	avg (MG/KG)	std (MG/KG)	max (MG/KG)	95uci (MG/KG)
PCE	0.008	0.001	0.007	0.012	0.050	0.021	0.002	0.101	7/7	0.014	0.017	0.050	0.030
TCE	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.004	0/7	0.001	0.000	0.001	0.001
DCE	0.001	4.200	0.001	0.001	0.001	0.001	0.001	4.203	1/7	0.600	1.587	4.200	2.266
TCA	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.004	0/7	0.001	0.000	0.001	0.001

Summary of 1996 Soil Sampling Results
Samples for 12 Meters in Depth

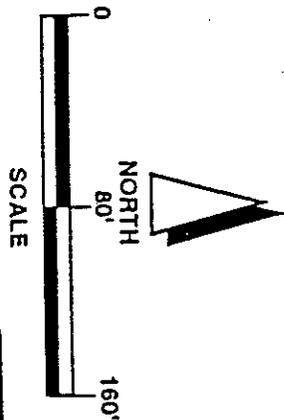
Chemical	DP-3-12	DP-6-12	Totals	freq	avg (MG/KG)	std (MG/KG)	max (MG/KG)	95uci (MG/KG)
PCE	0.010	0.058	0.068	2/2	0.034	0.034	0.058	0.339
TCE	0.001	0.001	0.001	0/2	0.001	0.000	0.001	0.001
DCE	0.001	0.001	0.001	0/2	0.001	0.000	0.001	0.001
TCA	0.001	0.001	0.001	0/2	0.001	0.000	0.001	0.001

Summary of 1996 Soil Sampling Results
Samples for 17 Meters in Depth

Chemical	DP-1-17	DP-2-17	DP-5-17	DP-8-17	Totals	freq	avg (MG/KG)	std (MG/KG)	max (MG/KG)	95uci (MG/KG)
PCE	0.042	0.200	0.045	0.001	0.288	4/4	0.072	0.088	0.200	0.212
TCE	0.001	0.001	0.001	0.001	0.003	1/4	0.001	0.000	0.001	0.001
DCE	0.001	0.006	0.001	0.001	0.007	1/4	0.002	0.003	0.006	0.006
TCA	0.001	0.001	0.001	0.001	0.002	0/4	0.001	0.000	0.001	0.001



LEGEND:
 ● EXISTING WELL LOCATIONS
 ⊕ DP-8 LOCATION OF DIRECT PUSH SAMPLING



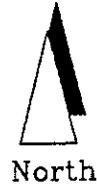
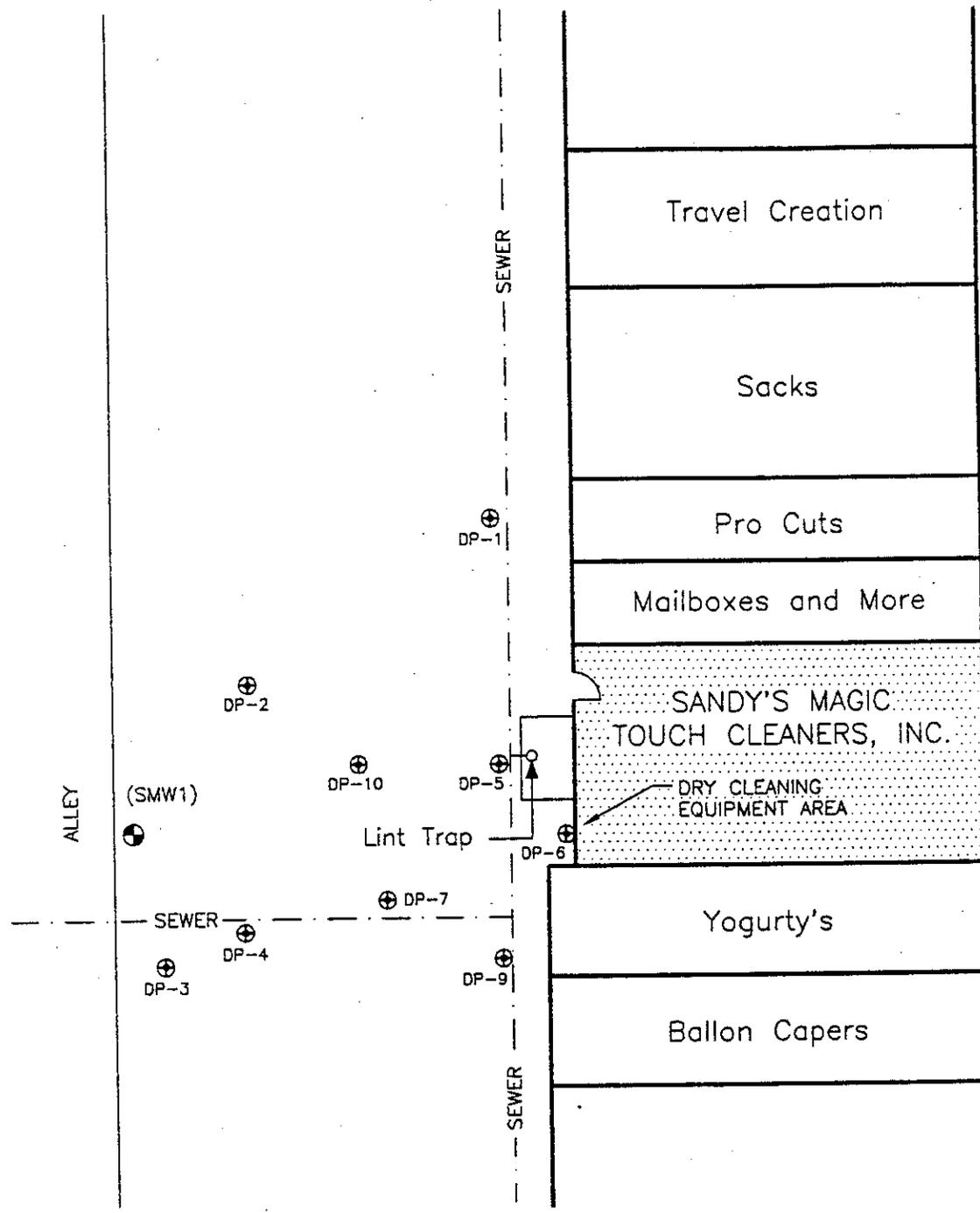
	PROJECT NO. 10730 ADEQ 96-ET-81
	MONITORING WELL LOCATIONS SANDY'S CLEANERS 4730 EAST INDIAN SCHOOL ROAD PHOENIX, ARIZONA

6/96

FIGURE 1-3

Plot Scale: 1"=30'

Drawn By: BDE Last Data Revised: 6-26-96 File Name: C:\ET\10730\BORE_ACT.DWG



LEGEND

-  EXISTING BUILDING
-  EXISTING GROUNDWATER MONITOR WELL LOCATION
-  LOCATION OF DIRECT PUSH SAMPLING

	PROJECT NO. 10730
	ADEQ 96-ET-81

DIRECT PUSH SAMPLING LOCATION MAP
 SANDY'S CLEANERS
 4730 EAST INDIAN SCHOOL ROAD
 PHOENIX, ARIZONA

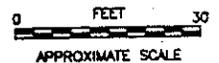
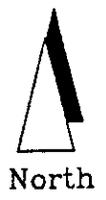
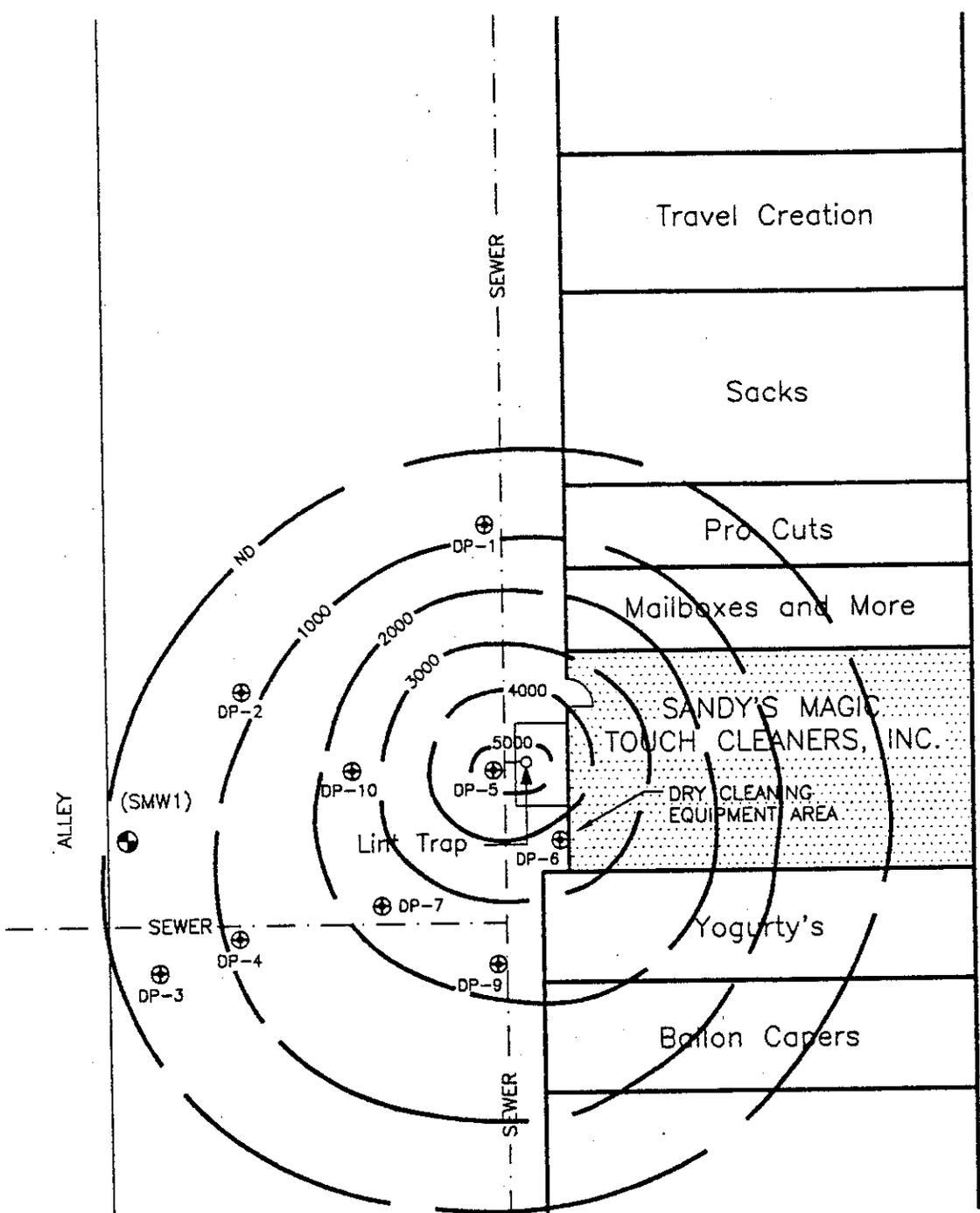
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FIGURE 1-2

Plot Scale: 1"=30'

File Name: C:\ET\10730\BORE_ACT.DWG

Last Date Revised: 6-26-86
Drawn By: BDE

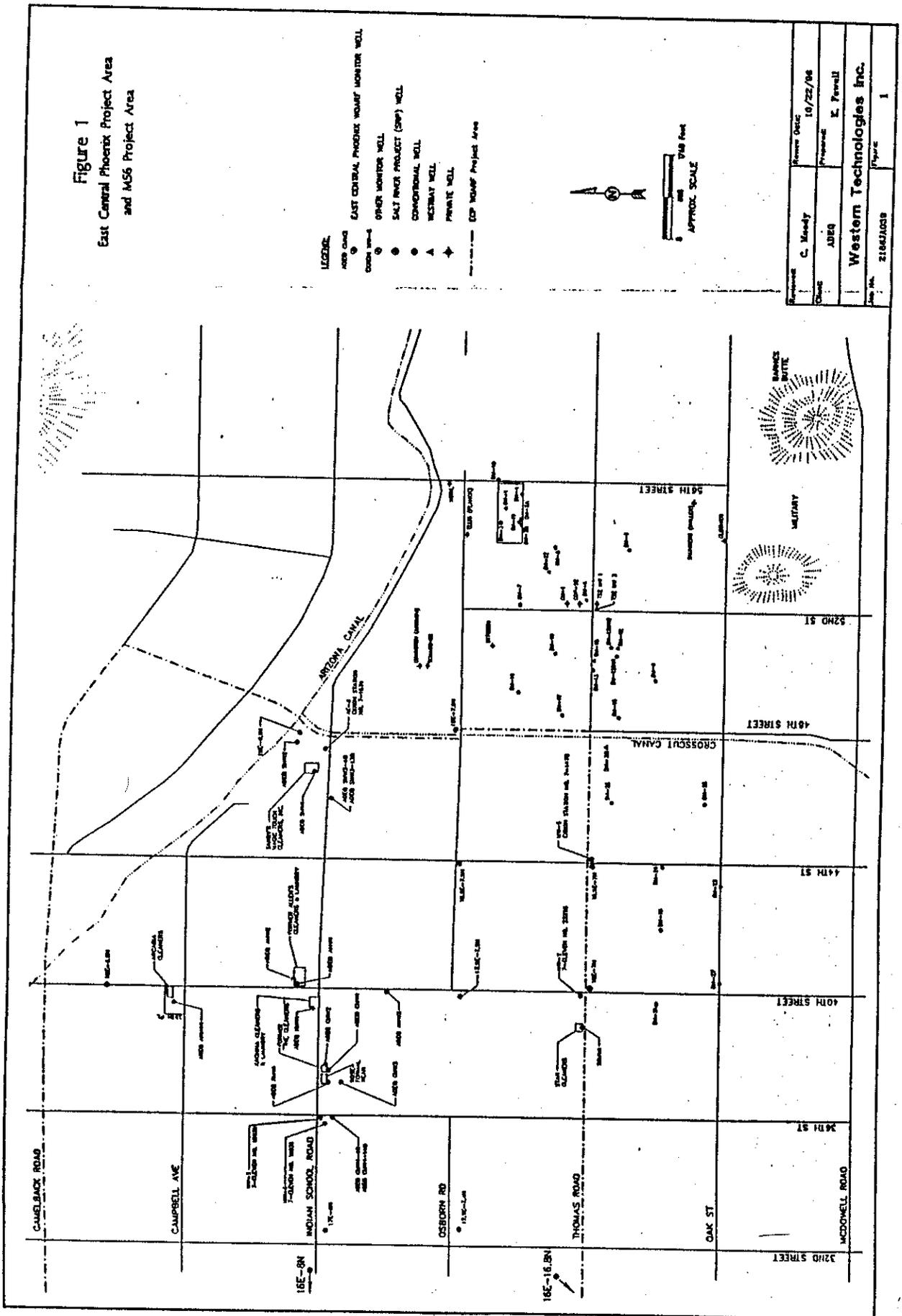


LEGEND

- 1000 APPROXIMATE PCE CONCENTRATION CONTOUR (ug/L)
- EXISTING BUILDING
- EXISTING GROUNDWATER MONITOR WELL LOCATION
- LOCATION OF DIRECT PUSH SAMPLING

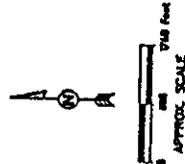
	PROJECT NO. 10730 ADEQ 96-ET-81
PCE CONCENTRATION CONTOUR MAP @ 5' bgs SANDY'S CLEANERS 4730 EAST INDIAN SCHOOL ROAD	
6/96	FIGURE 2-1

Figure 1
 East Central Phoenix Project Area
 and MS6 Project Area



LEGEND

- MS6 CHAD
- EAST CENTRAL PHOENIX WOLF MONITOR WELL
- OTHER MONITOR WELL
- SALT RIVER PROJECT (SRP) WELL
- CONVENTIONAL WELL
- ▲ WESTBAY WELL
- ◆ PRIVATE WELL
- MS6 Project Area



Prepared by	C. Meedy	Review Date	10/22/98
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Job No.	21867A008	Page	1

Toxicity Profiles

Benzene (BNZ)

Benzene (CAS No. 71-43-2, C_6H_6) is an aromatic hydrocarbon which occurs naturally in the environment and in the man-made form. Synonyms include benzol, coal naphtha, phenyl hydride, and pyrobenzol. Benzene is utilized mainly in the manufacture of ethylbenzene (intermediate in synthesis of styrene for plastics), cumene (for the manufacture of phenol and acetone), and cyclohexane (for nylon resins). Environmental emissions of benzene, which are mainly airborne, arise from gasoline vapors, auto exhaust, and industrial production and applications. Benzene is discharged into water and soil from industry, landfills, and underground storage tank leaks. Emissions from motor vehicles, tobacco smoke, hazardous waste sites, industry, and consumer use of products such as paints and adhesives are the main sources for human exposure. The highest exposure concentrations of benzene are found in industries utilizing benzene and benzene-containing products.

Benzene taken in by mouth may irritate the stomach causing gastritis. Inhalation of benzene at a low concentration (0.5 ppm) for a period of 1 to as long as 21 years has not caused any abnormal effects in the blood. Skin irritation may result if benzene is applied to the skin.

Routes of exposure include inhalation, ingestion, or dermal contact with human absorption of benzene occurring by these three routes. Less benzene is absorbed by dermal contact than with inhalation and ingestion exposures. Benzene has been determined to be distributed in the bile, blood, brain, fat (abdominal), kidney, liver, stomach, and urine of humans following inhalation exposure and in the adipose tissue, blood, bone marrow, kidney, liver, and mammary gland of animals with ingestion. In addition, dermal exposure studies in animals have demonstrated distribution in the kidney, liver, and skin. No evidence was found to indicate that the route of exposure influences benzene metabolism. In humans and animals, benzene is metabolized mainly by the liver's cytochrome P-450 system with toxicity believed due to benzene metabolites (e.g. hydroquinone, phenol, and muconic dialdehyde). Following inhalation in humans, benzene may be excreted unchanged by exhalation or through urinary output of conjugated derivatives (sulfates and glucuronides). Human dermal exposures have also resulted in urinary excretion of benzene. With ingestion exposures in animals, exhalation and urinary excretion have also been reported.

Documented research and reports on health effects from ingestion exposure are sparse compared to the documentation on inhalation exposure. Effects have been reported in humans, often without exposure concentration levels attributed to the specific health effect. For example, gastrointestinal effects, in the form of gastritis and later pyloric stenosis, have been reported in an individual who swallowed an unspecified amount of benzene. In another case, an accidental poisoning by ingestion resulted in an odd skin condition in the patient in which swelling and edema of the skin was observed. Symptoms of central nervous system toxicity (giddiness, vertigo, muscular incoordination, and unconsciousness) have been documented after a single ingestion dose of benzene at 125 mg/kg. Before 1913, leukemia was treated with oral benzene in gelatin capsules. The dosage began at 43 mg/kg/day and rose to 71 mg/kg/day for unspecified durations. These patients manifested a decrease in white blood cell counts and multiple hemorrhages resulting in anemia. It was not clear if the benzene treatment caused the effects or if they were due to the leukemic condition. In addition, fatal oral doses of benzene have been estimated to be 125 mg/kg for a 70-kg weight person. Death has been caused by respiratory arrests, central nervous system depression, or collapse of the heart. Accidental ingestion or attempted suicide have resulted in manifestations of staggering gait, vomiting, shallow and rapid pulse, somnolence, and loss of consciousness with delirium, pneumonitis, collapse, central nervous system depression, coma, and death. Visual disturbances and/or feelings of excitement or euphoria which may suddenly reverse to weariness, fatigue, sleepiness, convulsion, coma, and death have also been observed with lethal doses. No other effects have been documented for humans following ingestion exposure.

The hematological system is a major site of benzene toxicity from inhalation exposure. Exposure to benzene for a duration of several months to several years may result in a progressive sequence of diseases of greater severity, dependent upon concentration of exposure. Exposure to benzene for several months to

several years may produce abnormal numbers of circulating blood cells and pancytopenia (decrease in red and white blood cells and platelets due to pathology in the red bone marrow). Aplastic anemia is a more serious effect observed when the bone marrow ceases to function. With further progression, preleukemia (myeloblastic dysplasia) or acute leukemia may occur. Aplastic anemia is often a precursor of the more serious condition of acute myelogenous leukemia.

Health effects have been studied in a number of studies of workers with inhalation exposure to benzene for intermediate and chronic periods. Occupational studies may lack controls and adequate exposure data and may have multiple chemicals present, but they have helped to demonstrate the gross effect of dose in the development of anomalies.

In one investigation of refinery workers, no adverse health effects to the blood were observed at low levels of exposure to benzene (0.5 ppm) for a duration of from 1-21 years. Ten workers of a chemical factory, however, who were exposed over a duration of less than 10 years to benzene levels of 25 ppm or greater, were found to have a rise in the mean corpuscular volume at the termination of the high exposure period. At 75 ppm, a significant reduction in the red and white cell counts and hemoglobin was reported in workers of the rubber industry after an exposure period of 10 years. These changes disappeared when the exposure concentrations subsequently fell to 15-20 ppm in later years.

With a rise in the concentration levels or duration of exposure, more severe effects have been reported in a study of Turkish shoe manufacturing workers exposed to a maximum of 210 ppm of benzene between 4 months and 17 years. Hematological abnormalities were seen in 51 out of the 217 Turkish male workers and included loss of leukocytes, leukopenia, thrombocytopenia, pancytopenia, and eosinophilia. In another shoe industry cohort with exposure concentrations of 210-640 ppm for 4 months to 15 years, thirty-two workers, demonstrated pancytopenia with abnormal bone marrow function. In one other investigation, intensive studies of the blood were done on 102 out of 332 workers exposed to 11-1,060 ppm of benzene for 6 months to 5 years in a study of printing industry workers. Of the 102, 22 were found to have pancytopenia or other clinical manifestations. A positive correlation has also been reported between the prevalence of adverse health effect and concentration of exposure to benzene in shoe factory workers in a Chinese study. Four out of 211 workers, who were exposed to a mean concentration of 324 ppm for 8 months, developed aplastic anemia. In addition, preleukemia or acute leukemia was reported in 26 out of 28,500 workers exposed to benzene at 210-650 ppm for a period of from 1 to 15 years. Further indications of a dose-response effect were observed in a follow-up study done in 44 pancytopenic patients who received benzene exposure (150-650 ppm) from adhesives for a period of from 4 months to 15 years. Of the 44, 23 experienced complete remission, 14 died of complications from the pancytopenia, 1 died of myeloid metaplasia, and 6 developed leukemia. With the reduction in concentrations of benzene, less serious effects were noted.

Benzene exposure by inhalation has also been shown to influence acquired immunity, both humoral and cellular. The alteration of the antibody levels in the blood (humoral immunity) and the modification of circulating leukocytes (cellular immunity) are examples of the effects to the immunological system from benzene exposure. A study of benzene exposure at 3-7 ppm in painters, who had simultaneous exposures to toluene and xylene for 1-21 years, resulted in findings of a rise in serum immunoglobulin levels for IgM and a reduction for the IgG and IgA levels (humoral immunity). Due to multiple chemical exposures received by the painters, the changes could not be attributed to benzene alone. Reports of reduced circulating leukocytes (cellular immunity) have been documented in a number of benzene exposure studies which were previously described in the hematological effects section. In addition, the leukocyte alkaline phosphatase activity rose in workers with chronic benzene exposure at around 31 ppm.

Acute inhalation exposure to benzene has been shown to produce neurological effects (drowsiness, dizziness, headache, vertigo, tremor, delirium, and loss of consciousness) at levels from 300 to 3,000 ppm. At a higher concentration (approximately 20,000 ppm), an acute exposure of 5-10 minutes can produce death. In nonlethal cases, individuals have manifested headaches, nausea, staggering gait, paralysis, convulsions, and unconsciousness at levels of 700-3,000 ppm. Chronic benzene exposures have also been associated with

neurological deficits. Eight patients (six with aplastic anemia and two with preleukemia) with prior work-related exposure to adhesives and solutions containing 9-88% benzene were evaluated. Four of the six with aplastic anemia were found to have neurological abnormalities (global atrophy of the lower extremities and distal neuropathy of the upper extremities). The concentration of benzene in air at this site was 210 ppm or higher. Effects of chronic benzene and toluene exposure were also investigated in 121 workers. The exposure duration to benzene was 2-9 years with four of these years at concentrations of from 6-15.6 ppm (20-50 mg/m³). Toluene concentrations at this time did not exceed 5-mg/m³. Seventy-four of the workers had complaints of frequent headaches (generally at the completion of a work day), tiring easily, sleep problems, and memory loss. Developmental effects from inhalation exposure to benzene have been inconclusive due to study limitations (e.g. multiple chemical exposure, insufficient exposure data). One study reported higher occurrences of chromatid and isochromatid breaks and sister chromatid exchange in lymphocytes for the children of mothers, who had received inhalation exposure to benzene and other organic solvents during pregnancy.

Reproductive effects have been noted with inhalation exposure to benzene. Thirty women, with symptoms of toxic effects from benzene exposure, were studied. Twelve of the women were found to have menstrual disorders. Of the 12, 10 were married with two of these women having experienced spontaneous abortions. Benzene concentrations in air were presumed to be greater than 1 ppm. Menstrual cycle abnormalities have also been shown in other studies. These studies were limited due to the presence of other chemicals besides benzene and lack of exposure data.

In addition, genotoxic effects have been reported with benzene inhalation exposure, especially at levels causing abnormalities to the blood. Fifty-two workers were exposed to benzene concentrations less than 10 ppm for 1 month to 26 years. A significant rise in the frequency of chromosomal abnormalities were found in peripheral lymphocytes of workers when compared to 44 controls. Other factors such as radiation or chemicals may have facilitated the development of the chromosomal deviations. In addition, the study lacked a baseline blood workup in the workers. In one case study, a worker was reported exposed for 18 months to benzene at levels of 200-1640 mg/m³ (62.6-513.4 ppm). The individual developed severe anemia, neutropenia, and thrombocytopenia and subsequently was diagnosed as having leukemia. Bone marrow specimens showed an excess number of D-group chromosomes. In another example, metaphase chromosome spreads were examined in 48 out of 66 benzene-exposed persons and 29 out of 33 controls. The incidence of metaphase chromosome spreads was found to be slightly higher in the workers exposed to benzene at levels of 10-100 ppm as compared to a group of controls.

Studies of the association between exposure from benzene and the development of neoplastic disease have had deficiencies in the study methodology (e.g. inappropriate sampling techniques) and with the inadequate exposure data. However, a cause-effect relationship has been established between benzene and the development of acute myeloid leukemia (AML), as evidenced by the consistently higher incidence of AML in workers with excess benzene exposure. With AML, a decline is seen in the production of normal erythrocytes, granulocytes, and platelets which subsequently causes death by anemia, infection, or hemorrhage. Other leukemias and lymphomas have been associated with benzene exposure, but only the association between excess benzene exposure and AML has been consistently observed. Research was done on 3,536 male chemical workers with cumulative benzene exposures of less than 180 ppm (1,809 workers), 180-719 (1,047 workers) or equal to or greater than 720 ppm (680 workers). The 680 workers in the highest category of exposure were four times more likely to develop leukemia or other lymphopietic cancers. None of the leukemias were of the myelogenous type. The research demonstrates the dose-response effect of benzene exposure. In another study, mortality was examined in 594 white males with occupational exposure to benzene in a chemical manufacturing facility with varying employment periods between 1940 to 1970. The time-weighted average exposure to benzene was 0.1 to 35 ppm. Three cases of myelogenous leukemia (as compared to an expected of 0.8 cases) developed, with one of these cases having an exposure level to benzene of less than 2 ppm. This study demonstrated that a risk of leukemia is present even at a low exposure

concentration. In a retrospective study of 26,319 benzene factory workers in China, thirty cases of leukemia (23 of them AML) were discovered. Workers with leukemia had mean benzene exposure levels between 3 to 313 ppm, with most workers exposed at levels between 16 to 157 ppm. And, finally, in another epidemiologic study, no leukemia deaths were observed at benzene exposure concentrations of less than 1 ppm. The study cohort included 454 workers of a Texas refinery, employed in the period between 1952 and 1978. The refinery workers had a median benzene exposure of 0.14 ppm while employees of benzene-related units had 0.53 ppm levels of exposure. The relative risk for all cancers within this group was not significantly different from non-exposed comparison groups. Other epidemiologic studies for inhalation exposure, which have not been discussed, have supported the causal association between benzene exposure and the development of neoplastic disease, particularly AML. Benzene is one of the few substances in which human evidence is sufficient to categorize it as a human carcinogen. Benzene has an USEPA WoE classification of A (human carcinogen).

Human studies of human health effects from dermal exposure to benzene are even rarer than for ingestion exposure. Benzene is a skin irritant for humans, causing erythema, vesiculation, and dry and scaly dermatitis due to the defatting of the keratin layer. In addition, genotoxic effects have been observed with inhalation exposures which may have had a simultaneous dermal exposure.

A number of substances are known to interact with benzene and, therefore, influence its metabolic activity and toxicity. Ethanol has been shown to intensify the metabolism of benzene and the toxic effects of anemia, lymphocytopenia, and atypical cell morphology in animals. In addition, when animals have been pretreated with phenobarbital, benzene hydroxylation has also been shown to be activated. In contrast, toluene inhibits the breakdown of benzene to phenol, one of benzene's toxic metabolites. In-vitro experiments of mouse liver microsomes have demonstrated that carbon monoxide, aniline, aminopyrine, cytochrome C, and metyrapone have also been shown to inhibit benzene metabolism.

Bromodichloromethane (BDCM)

Bromodichloromethane (CAS No. 75-27-4, CHBrCl_2) is a volatile halogenated hydrocarbon (trihalomethane) which is formed as a by-product from chlorination of water. BDCM is generally used as an intermediate in the synthesis of other chemicals and as a laboratory reagent. Domestic water supplies contaminated with organic material require added chlorination resulting in elevated levels of BDCM and other trihalomethanes. Higher concentrations of exposure to BDCM are seen in individuals consuming or exposed dermally to this water. Even under normal conditions, individuals with health problems who consume a large quantity of water (diabetics) or who are exposed by inhalation and dermal contact in swimming pools will have potentially higher exposures to BDCM than others.

The routes of exposure for BDCM include inhalation, ingestion, or dermal contact. No studies were available which dealt with human absorption, distribution, and excretion of BDCM following inhalation, ingestion, or dermal contact. With ingestion exposure, examination of female monkeys demonstrated almost complete gastrointestinal absorption. In rodents, BDCM was administered by gavage and remained in the stomach for a period of time before being distributed to the fat, liver, muscle, and other tissues. The metabolic pathways for BDCM in humans have not been established. However, animal studies have shown that carbon dioxide is the major end product in mice. In rats, mice, and monkeys, excretion was by exhalation following ingestion and, to a lesser degree, through the urinary and fecal routes. In rats, 42% of BDCM was expired unchanged with 14% expired as carbon dioxide.

Only animal studies were available for examining the consequences to health from ingestion exposure to BDCM. A number of body systems have been shown to be affected by oral exposure dependent upon the concentration of exposure.

In one study, rats who received 130 mg/kg/day of BDCM in their diet for two years had no hematological changes when compared to control animals. Similarly, a dose of 213 mg/kg/day of BDCM in

drinking water for a period of 90 days produced no adverse effect to lymphocyte levels in male or female rats. Hematological effects have been observed at relatively high levels of oral exposure. After a single dose of BDCM at 390 mg/kg, hemoglobin and hematocrit levels decreased significantly in male rats.

The liver has been shown to be sensitive to the harmful effects of BDCM at various concentrations. In one rare finding, a low dose of 7 mg/kg/day resulted in significant effects to the liver. In subchronic rodent studies of 10 to 14 days, slight effects (mild increases in weight and minimal microscopic changes in the liver) have been seen at 37 mg/kg/day and 50 mg/kg/day. These effects become more noticeable at doses of 125 to 300 mg/kg/day. Liver damage in rodents have also been observed at doses ranging from 50 to 200 mg/kg/day for long-term studies. At acute, single doses around 1,250 mg/kg or higher, characteristic signs (increased liver weight, pale discoloration of the liver, rise in levels of hepatic tissue enzymes in serum, reduction in secreted hepatic proteins in blood, and focal inflammation or degeneration of the liver) have been observed. At this concentration level, death has resulted within two weeks.

The kidney is also an organ sensitive to BDCM oral exposure. In long-term studies, focal necrosis of the proximal tubular epithelium was observed in male mice exposed for 13 weeks to doses of 100 mg/kg/day with cytomegaly following chronic exposure of 25 mg/kg/day. Cytomegaly and nephrosis were reported in rats chronically exposed at 50 to 100 mg/kg/day. Doses of from 74 to 148 mg/kg/day resulted in a reduction in the uptake of p-aminohippurate into kidney slices from mice, indicating a decrease in the kidney excretory flow. A dose of 200 mg/kg/day for 10 day resulted in a rise in the renal weight of rats. In subchronic studies (14-day), a rise in the blood urea nitrogen has been noted in mice receiving 250 mg/kg/day of BDCM.

Immunological effects have not been examined in detail, but effects have been reported with oral exposure. In one study, mice received BDCM for 14 days. Females were found to have a reduction in the number of antibody-forming cells in the spleen and in the hemagglutination titer at doses of 125 to 250 mg/kg/day.

Neurological outcomes have also been observed. One study explored the long-term effects on behavior from BDCM ingestion exposure. Mice were examined two days after the final doses of BDCM. No effect was observed on tests of coordination, strength, endurance, or exploratory activity at doses of 1.2 to 11.6 mg/kg/day for 14 to 90 days. Passive-avoidance learning was not influenced by a 90 day exposure to BDCM at 100 mg/kg/day. Acute effect on operant behavior (decreased pressing of a lever that delivers food) was seen at 100 or 400 mg/kg/day for 90 days, but these effects were not lasting. Oral doses for rodents of 150 to 600 mg/kg frequently result in signs of acute CNS depression (lethargy, labored breathing, sedation, and flaccid muscle tone) with reverse of these signs after several hours.

Developmental effects (sternebral anomalies) were observed in fetuses of female mice exposed orally to 50 to 200 mg/kg/day on days 6 to 15 of gestation (when organogenesis occurs). Maternal toxicity (40% reduction in body weight gain) was also observed.

Genotoxic effects of a rise in occurrence of sister chromatid exchange (SCE) in mice have been detected. A significant rise in SCE s have been demonstrated in animals receiving doses of 50 or 100 mg/kg/day for four days of BDCM. Doses of 200 mg/kg/day for four days resulted in death.

The acute dose, which is lethal to 50% of the exposed rodents, ranges between 400 and 1000 mg/kg. Pathological changes detected in acutely poisoned animals included fatty infiltration of liver and hemorrhagic lesion in the kidney, adrenals, lung, and brain. In a two week, repeated-dose study, a dose of 150 mg/kg/day was lethal to all the animals in the study. Male rodents appeared more sensitive to the lethal effects of BDCM.

No human studies were available which documented the effect of BDCM exposure and the development of cancer with ingestion exposure. However, epidemiologic studies have been done on the frequency of cancer with ingestion of chlorinated water. Because other trihalomethanes are present in chlorinated water, difficulties arise in determining the specific effect of BDCM on the development of cancer. Chronic oral studies of animals have, however, given persuasive evidence that BDCM is carcinogenic. Tumors of the large intestine were reported in male rats exposed to 50 and 100 mg/kg/day and in females at 100 mg/kg/day. Male and female rats exposed to 100 mg/kg/day had a rise in the frequency of liver tumors.

Female rats exposed orally to 150 mg/kg/day of BDCM had a rise in the frequency of liver tumors. In mice, renal tumors were detected with oral exposure at 50 mg/kg/day, and hepatic tumors were reported in females exposed at 75 or 150 mg/kg/day. BDCM has an USEPA WoE classification of B2 (probable human carcinogen).

No studies were available in animals or humans for inhalation or dermal exposures.

A study of rats demonstrated BDCM's interaction with acetone. The toxic effects on liver and kidneys were enhanced when rats were given oral BDCM following the ingestion of acetone.

Chloroform (CLFM)

Chloroform (CAS No. 67-66-3, CHCl_3) is a halogenated hydrocarbon (trihalomethane) which occurs naturally in the environment and is also man-made. Synonyms include trichloromethane, methenyl chloride, methane trichloride, methyl trichloride, and formyl trichloride. Chloroform is used mainly for the manufacture of fluoropolymers and as a coolant in air conditioners. In the past, chloroform was also used as an anesthetic. Environmental discharge of chloroform arises primarily from its manufacture and use, and from chlorination of wastewater and drinking water. The greatest release occurs to the air and secondarily to the groundwater. Occupational exposures take place in industries which manufacture or utilize chloroform. Exposure to the public occurs from consumption of contaminated food and water, inhaling contaminated air, and dermal contact with water which contains chloroform (e.g. shower) with high exposures for persons residing in areas with natural background levels of chloroform (e.g. proximity to water treatment plants).

Chemical interactions have been observed between chloroform and a number of other substances. When the drug, morphine, was utilized as a premedication with chloroform as an anesthetic, severe respiratory depression was observed. Animal studies have also demonstrated interaction of chloroform with other substances. When chloroform was administered together with dicophane (DDT), phenobarbital, ketonic solvents and chemicals, carbon tetrachloride, or ethanol, the hepatotoxicity of chloroform was enhanced. In experiments with rat hepatocytes, cadmium and chloroform have been observed to act synergistically to increase the cytotoxicity of each. When disulfiram, diethyldithiocarbamate, or carbon disulfide was given simultaneous with chloroform, the hepatotoxicity of chloroform was diminished.

The routes of exposure for chloroform include inhalation, ingestion, or dermal contact. Of the inhaled dose of chloroform, the amount of absorption by the body is related to factors such as concentration of chloroform in inhaled air. With oral exposure in humans, 100% of the chloroform was shown to be absorbed from the gastrointestinal tract. Following death from chloroform anesthesia, the organs of seven patients were examined for concentrations of chloroform. Highest levels were distributed in the brain, followed by the lungs and liver. In one human study, half of an oral dose of chloroform was shown to be metabolized into CO_2 . In another study, around 38% of the chloroform received orally was metabolized in the liver with approximately 17% exhaled unchanged. Chloroform was excreted by exhalation following inhalation exposure and mainly by exhalation and secondarily by urinary excretion following ingestion exposure in humans.

Health effects from ingestion exposure of chloroform are frequently limited to case studies of humans. Animal data are presented in the absence of human studies. Data on respiratory effects from ingestion of chloroform are limited. In a case report of an accidental ingestion of chloroform, the patient was shown to have a respiratory tract obstruction due to muscular relaxation. The oral dose for this patient was estimated at 2,500 mg/kg of chloroform. In another case, a patient committed suicide by ingesting 3,755 mg/kg of chloroform. On autopsy, the lungs were found to be congested with scattered patches of pneumonic consolidation.

Data on cardiovascular effects after ingestion exposure are also limited. A patient who accidentally ingested 2,500 mg/kg of chloroform was found to have some electrocardiographic changes (occasional extrasystoles and a slight S-T segment depression). The patient's blood pressure was 140/90 with a pulse of 70 beats per minute.

Gastrointestinal effects have also been observed in case studies of accidental or intentional ingestion of chloroform. Retrosternal soreness, pain on swallowing, and gastric distress with vomiting have been documented. Congestion with patchy necrosis of the mucosa have been found in the stomach and duodenum of a male who ingested approximately 3,755 mg/kg of chloroform and subsequently died. These findings were noted on autopsy.

Hematological effects were documented from a case study of one subject who took approximately 21 mg/kg/day of chloroform in cough medicine. The intake of this medicine for ten years was associated with a reduction in the erythrocyte count and in the hemoglobin level.

Musculoskeletal effects were also seen in an individual case study of a man who accidentally took 2,500 mg/kg of chloroform. The ingestion was associated with a relaxation of the jaw which resulted in the development of upper respiratory obstruction.

Hepatic effects have been observed with ingestion exposure to chloroform. The liver has been shown to be the target organ in chloroform toxicity. Injury to the liver has been observed in patients within 1-3 days of ingestion exposure. Jaundice, liver enlargement and tenderness manifested in all the patients who were observed. Blood tests revealed a rise in liver enzymes (SGOT, SGPT, LDH) and bilirubin levels. In a single fatal case, fatty degeneration and extensive centrilobular necrosis were detected on autopsy. Impaired liver function was seen in a person who took cough medicine containing 21 mg/kg/day of chloroform for 10 years. These changes reversed when the cough medicine was stopped. In another example, liver function was not altered in humans using mouthwash with a chloroform concentration of 2.46 mg/kg/day of chloroform for a period of five years or less.

Renal effects have also been observed with ingestion exposure to chloroform. The kidney has also been shown to be a target organ in chloroform toxicity. Following chloroform ingestion (2,500 mg/kg or approximately 3,755 mg/kg), oliguria was reported the day following the oral intake. Renal injury was also manifested by a rise in blood urea nitrogen and creatinine levels. Albumin and casts were also found in urine. In one fatal case, histopathological tests showed epithelial swelling and hyaline and fatty degeneration in the convoluted tubules of the kidney. Albumin and casts were also detected in the urine of a subject who took cough medicine for 10 years which had a concentration of 21 mg/kg/day of chloroform.

Dermal/ocular effects have been observed in animals with ingestion exposure. Mice developed rough coats at exposure levels of 100 mg/kg/day of chloroform in oil for 14 days. Alopecia was reported in pregnant rats exposed to 126 mg/kg/day of chloroform in oil.

Immunological effects have been reported in animals following ingestion exposures. Depression of the humoral immunity (antibody-forming cells) was observed in mice receiving 50 mg/kg/day of chloroform for 14 days. Cell-mediated immunity (delayed type hypersensitivity) was influenced by a high dose of 250 mg/kg/day of chloroform administered to female mice for a 14 day period and a 90 day period. Reduced lymphocyte counts were seen in female rats receiving a single gavage dose of 1,071 mg/kg, but no health effects were detected in the group receiving 765 mg/kg.

Neurological effects have additionally been reported in case reports of humans following oral exposure. Levels of 2,500 or 3,755 mg/kg produced deep coma immediately following intentional or accidental ingestion. Reflexes were absent and the size of the pupil changed. With the exception of one individual, all patients lived. One person died several days later of extensive liver necrosis. Another patient was reported to have mild cerebellar damage (unstable gait and intentional tremor) which later reversed itself.

Developmental effects have also been documented in animals. No effects were reported in rats from oral exposures of 50 mg/kg/day. A rise in the rate of resorptions was seen in rabbits exposed to 100 mg/kg/day during gestation. Reduced birth weight was detected in offspring of mothers receiving 126 mg/kg/day of chloroform exposure. Decreased fetal weight was not seen in offspring of mothers receiving 200 mg/kg/day but was observed in offspring of mothers who were treated by gavage with 400 mg/kg/day of chloroform.

Reproductive effects were seen in some studies but not in others. Rabbits exposed to 63 mg/kg/day of chloroform during gestation experienced abortions. Intermediate duration of exposure to 160 mg/kg/day of chloroform in drinking water resulted in no histopathology in rabbits. A rise in the rate of resorptions was detected in rats exposed to 316 mg/kg/day of chloroform by ingestion. Gonadal atrophy was reported in male and female rats treated by gavage with 410 mg/kg/day of chloroform in toothpaste. Chronic exposures at concentrations of 200 and 477 mg/kg/day of chloroform by gavage did not result in histopathological changes in the reproductive organs of male and female rats.

Additionally, genotoxic effects of increased sister chromatid exchange in bone marrow cells of mice were observed with ingestion exposure of 200 mg/kg/day of chloroform in oil by gavage for four days.

Death has been observed in accidental or intentional ingestion of chloroform. A dose of 212 mg/kg may be fatal to humans. An ingested dose of 3,755 mg/kg of chloroform proved fatal to a man whose death was due to severe hepatic injury. Chloroform has an EPA Weight-of-Evidence Classification of B2 (probable human carcinogen).

1,1-Dichloroethylene (DCE)

1,1-Dichloroethylene (CAS No. 75-35-4, $C_2H_2Cl_2$) is a halogenated hydrocarbon made by man. Synonyms include 1,1-dichloroethene; 1,1-DCE; and vinylidene chloride. DCE is used to manufacture packing wrap (Saran™) and flame-retardant fabrics. DCE is released primarily into air and water from industrial emissions, hazardous waste sites, and accidental spills. The highest potential exposure levels are seen in occupations utilizing DCE and in populations residing near hazardous waste sites.

Toxic intermediates from the metabolism of DCE are responsible for its adverse health effects. A number of substances act to increase or decrease the development of these intermediates. SKF-525-A, disulfiram, and other dithiocarbamates (thiram, diethyldithiocarbamate) are thought to inhibit the enzymes responsible for the formation of the DCE toxic intermediates. Administration of amino acids (cysteine, methionine) also has a protective effect against DCE toxicity. On the contrary, substances such as 1,1,1-trichloropropane and other inhibitors of epoxide hydrolase enhance DCE toxicity as does phenobarbital with high levels of DCE by inhalation. In addition, replacement therapy of thyroxine following removal of the thyroid in rats intensifies the liver damage from subsequent DCE exposure. In addition, diethyl maleate also increases liver damage by depleting glutathione (reducing agent in the body).

The routes of exposure for DCE include inhalation, ingestion, or dermal contact. No human studies were available for the absorption, distribution, metabolism, and excretion of DCE. In animal studies, DCE was readily absorbed following inhalation and ingestion exposures and was distributed to the kidneys, liver, and lungs on inhalation and to the kidneys and liver on ingestion. The metabolic pathway of DCE in rats has been extensively studied with formation in the initial stages of an epoxide intermediate. With inhalation exposure, the majority of the DCE metabolites was excreted in the urine with very little eliminated unchanged in the expired air. In an ingestion study of rats, the greatest portion of the DCE was excreted in the urine (44-80%) and recovered as CO_2 (5-14%) with 1% unchanged in expired air and a small amount in the feces.

Upper airway irritation, a high incidence of liver toxicity in workers of a DCE polymerization plant, and CNS depression (convulsions, spasms, unconsciousness) have been demonstrated in humans with inhaled DCE. In addition, animal research has demonstrated that DCE is a weak teratogen and also causes reproductive effects and DNA damage with inhalation. Toxic effects in humans were not available for ingestion exposure. However, oral animal studies produced adverse outcomes to the gastrointestinal (forestomach edema) and respiratory (pulmonary edema) systems, to the liver (necrosis, hemorrhage), and to fetal development (increase in mean fetal crown-rump length in pups). With human dermal exposure, local irritant effects were observed.

Three human studies investigated the association of inhalation exposure to DCE and the development of cancer. No association was discovered, but the studies had real limitations such as small sample sizes.

Animal studies have reported an increase in kidney and mammary cancers and lung tumors with inhalation exposures. Liver cancer was seen in oral animal studies. Dermal application of DCE in mice demonstrated its tumor initiator effect. DCE has an EPA Weight-of-Evidence Classification of C (possible human carcinogen).

1,2-Dichloropropane (DCP2)

1,2-Dichloropropane (CAS No. 78-87-5) is an organic chemical which is present in the environment due to human activities. Synonyms include propylene dichloride; propylene chloride; 2,3-dichloropropane; and 1,2-D. It's main uses are as a chemical intermediate in the production of tetrachloroethylene and other chlorinated products and as an industrial solvent. Environmental emissions occur as a result of its production and uses as described above and, and its evaporation from wastewater streams. No specific groups have been reported to have greater susceptibility to the health effects of 1,2-dichloropropane than others.

When 1,2-dichloropropane has been mixed with other substances, chemical interactions have been observed. When administered orally or by inhalation with 1,1,2-trichloroethane and when administered with both ethylene dichloride and tetrachloroethylene, a toxic effect which was additive was observed for the dose lethal to 50% of the animals. An additive health effect to the lung, liver, and nervous system was seen when 1,2-dichloropropane was administered to rodents in combination with 1,2,3-trichloropropane and tetrachloroethylene.

Routes of exposure include ingestion, inhalation, and dermal contact. Following animal ingestion and inhalation exposure, absorption is considered to occur as evidenced by the presence of 1,2-dichloropropane in urine and expired radioactive carbon-labeled CO₂ in air, 1,2-dichloropropane was also found in excreta. Following application to the skin of rabbits, absorption was assumed to occur due to the death of the exposed animals. The liver, kidney, lung, and blood were the areas of distribution for 1,2-dichloropropane following inhalation exposure. The brain and cerebellar tissue, adipose tissue, and liver had the highest concentration following ingestion exposure. When radioactive carbon-labeled 1,2-dichloropropane was metabolized by rats following ingestion exposure, over 40% of the dose was measured in expired air. Rats exposed by gavage or inhalation to 1,2-dichloropropane were reported to have three major metabolites in their urine: 1) N-acetyl-S-(2-hydroxypropyl)-L-cysteine, 2) N-acetyl-S-(2-oxopropyl)-L-cysteine, and 3) N-acetyl-S-(1-carboxyethyl)-L-cysteine. These metabolites formed after oxidation of 1,2-dichloropropane and also before or after conjugation with glutathione. It may also conjugate with lactate, producing CO₂ and Acetyl Co-A, which may eventually breakdown into CO₂ or used in other biosynthetic pathways. With radioactive-labeled 1,2-dichloropropane, it was found that the urine and expired air were major excretory routes for ingestion and inhalation exposures and also the feces for ingestion exposure.

Health effects have been documented in some studies of humans and animals following ingestion exposure. No respiratory effect (histopathological lesions to the lungs, bronchi, and trachea) were reported in rats receiving gavage exposures of up to 1,000 mg/kg/day of 1,2-dichloropropane for 13 weeks, mice receiving up to 500 mg/kg/day for 13 weeks, or rats and mice receiving up to 250 mg/kg/day for 103 weeks.

Cardiovascular effects have, however, been documented with ingestion exposure to 1,2-dichloropropane. A single ingestion dose of unknown quantity resulted in the death by cardiac failure of two individuals 30 to 36 hours following exposure.

Gastrointestinal effects have also been noted with ingestion exposure. A case report documented an overexposure by ingestion concerning a 59 year old man. He suffered a burning feeling in the oropharynx, esophagus, and stomach with vomiting for an extended duration. In another case of unknown dosage, a man drank 1,2-dichloropropane in a suicide attempt and developed esophagitis and esophageal varices which eventually reversed itself.

Hematological effects have also been observed with ingestion exposure. Anemia, leukopenia, and disseminated intravascular coagulation was observed following accidental ingestion of an unknown dose of 1,2-

dichloropropane. One of the patients recuperated while two died, one from septic shock and the other from cardiac arrest.

Hepatic effects have been documented with ingestion exposure. Liver damage in the form of hepatic necrosis and histological changes have been reported in persons who intentionally ingested 1,2-dichloropropane.

Renal failure has also been documented in three humans following ingestion of 1,2-dichloropropane. Two of the persons died but death was not attributed to renal failure.

Neurological effects (dizziness, headache, disorientation, and coma) have been documented in patients who received lethal exposures to 1,2-dichloropropane. The concentration of the single doses were unknown.

The developmental effects of delayed ossification of the skull bones was seen in the fetuses of female rats receiving 125 mg/kg/day of 1,2-dichloropropane by gavage during gestation days 6-21.

Reproductive effects were reported in female and male rodents receiving gavage doses of 1,2-dichloropropane. Female mice administered doses of 125 and 250 mg/kg/day for 103 weeks had a rise in the incidence of infections to the ovary, uterus, or the other organs. With male rats, testicular degeneration was observed at gavage doses of 500 mg/kg/day for 1, 5, or 10 consecutive days of for 13 weeks (5 days/week).

When the occurrence of cancer was examined, a marginal but statistically significant rise in the incidence of adenocarcinomas of the mammary gland was reported for female rats who received 250 mg/kg/day of 1,2-dichloropropane for 103 weeks. In addition, a dose-related rise in liver adenoma was reported in male and female mice receiving gavage doses of 125 or 250 mg/kg/day of 1,2-dichloropropane.

Inhalation exposure to 1,2-dichloropropane has been found to produce respiratory effects to humans. An accidental spill of 2,000 gallons produced chest discomfort, dyspnea, and cough in some exposed individuals indicating respiratory irritation. No measurements of air concentrations were made. No adverse cardiovascular effects were seen on histological examination of the heart and aorta of rodents receiving exposures \leq 150 ppm and rabbits exposed to \leq 1000 ppm, 6 hours/day, 5 days/week for 13 weeks. However, fatty degeneration of the heart was observed in dogs receiving exposures of 1,000 ppm for 7 hours/day, 5 days/week for 27-128 exposures.

Gastrointestinal effects (vomiting, abdominal pain) were documented in a young female who sniffed stain remover composed mainly of 98% 1,2-dichloropropane. Inhalation exposure to 1,2-dichloropropane in two individuals resulted in hematological effects (epistaxis, hemolytic anemia, and disseminated intravascular coagulation).

Musculoskeletal effects were not observed in rodents exposed to \leq 150 ppm and rabbits exposed to \leq 1000 ppm, 6 hours/day, 5 days/week for 13 weeks. Hepatic effects have been observed in humans following inhalation exposure. Severe hepatic failure, as evidenced by the presence of liver enzymes, was documented in the case of a woman who inhaled cleaning solution containing 60% 1,2-dichloropropane. In another case a female inhaled trielina which was 98% 1,2-dichloropropane. Subsequent tests demonstrated liver damage. Severe renal failure was also observed in this woman. The concentrations of 1,2-dichloropropane were not documented in the two cases.

Ocular hemorrhages were also observed in a patient who had inhaled vapors of 1,2-dichloropropane. No information of the concentration was reported.

No immune system effects were documented in rodents and rabbits exposed to 150 ppm and 1,000 ppm respectively for an exposure lasting 13 weeks, 6 hours/day, 5 days/week. This was also true for an exposure at 1,000 ppm, 6 hours a day, 4-5 days/week for 2 weeks. In contrast, mice exposed to 300 ppm of 1,2-dichloropropane for 2 weeks, 6 hours/day, 4-5 days/week, manifested a reduction in the absolute and relative thymus weight and a decrease in cortical lymphoid cells.

Fatigue, which was possibly a neurological effect due to central nervous system depression, occurred following exposure to a leak of 2,000 gallons of 1,2-dichloropropane from a tank truck. The concentration of the leaked chemical was not known.

The reproductive effect of uterine bleeding between menstrual periods occurred in a woman following an acute inhalation exposure to 1,2-dichloropropane. The lethal concentration for 50% of the exposed mice was 480 ppm for a single 10 hour exposure and up to 3,029 ppm for a single 8 hour exposure in rats.

The hepatocarcinogenic effect of 1,2-dichloropropane inhalation has also been examined. Hepatomas were reported in 3 out of 80 mice exposed 37 times to 400 ppm for 4-7 hours. High mortality was also reported. Dermatitis was the main health effect from dermal exposure to 1,2-dichloropropane. For animals, 8.75 ml/kg was the dermal dose calculated as being fatal to 50% of the exposed rabbits.

No EPA WoE classification has been established for 1,2-dichloropropane.

Tetrachloroethylene (PCE)

Tetrachloroethylene (CAS No. 127-18-4, C_2Cl_4) is a halogenated hydrocarbon which is man made. Synonyms include carbon tetrachloride; carbon dichloride; ethylene tetrachloride; perchloroethylene; tetrachloroethene; and 1,1,2,2-tetrachloroethylene. PCE is commonly used as an industrial solvent and degreaser, as an intermediate for manufacturing other chemicals, and is used extensively in the dry cleaning and textile industries. Although PCE is liquid at room temperature, it tends to evaporate into the atmosphere which accounts for most of its environmental emissions, especially from the industrial and dry-cleaning operations. Exposure to PCE results from employment in certain industries (e.g. dry cleaning), residence near emission sites, and ingestion of contaminated food and water.

The routes of exposure for PCE include inhalation, ingestion, or dermal contact. Absorption following inhalation or ingestion is extensive but poor with dermal exposure. Following absorption, much of the inhaled and ingested PCE is deposited in the fatty tissue. PCE was reported to be distributed in the liver, kidney, brain, and lung of a dry cleaner who received a fatal inhalation exposure to PCE. The metabolism of PCE in the human body has been established by the detection of known metabolites (trichloroacetic and trichloroethanol) in the urine and blood of humans. In humans, PCE is excreted primarily through exhalation with urinary excretion playing only a secondary role in inhalation and ingestion exposures. With dermal exposure, excretion occurs by exhalation. With inhalation and ingestion exposure in humans, metabolites of PCE have been identified in urine and blood.

Data on human health effects from ingestion exposure to PCE are limited. Research on the health effects to animals are more commonly found. Hematological effects, including the relative reduction of bone marrow erythropoiesis, have been observed in mice receiving PCE in drinking water at a concentration of 0.1 mg/kg/day for seven weeks. At a concentration of 0.05 mg/kg/day, slight or no hematological effects were observed. A recovery period of two months resulted in the disappearance of all hematological effects.

Cardiovascular outcomes were reported in the Woburn Massachusetts study, in which health outcomes (particularly childhood leukemia) associated with contaminated drinking water were investigated. One of the solvents contaminating the drinking water was PCE. Family members (14/25 - 14 persons out of 25) of leukemia cases manifested cardiac symptoms of tachycardia at rest, palpitations, or near syncope at 21 ppb of PCE. Detailed testing was done on 11 of the individuals with some findings of serious ventricular dysfunctions and premature ventricular beats diagnosed in a number of those tested. Hepatic effects to humans from ingestion exposure to PCE have been rare. In one case study, an infant developed obstructive jaundice and hepatomegaly following exposure to PCE from the mother's breast milk. In rodents, PCE delivered by gavage at 1,000 mg/kg/day for 10 days produced evidence of peroxisomal proliferation. Gavage doses of PCE at 0, 20, 100, 200, 500, 1,000, 1,500, or 2,000 mg/kg/day for six weeks, produced a rise in relative liver weight and triglycerides starting at 100 mg/kg/day; reduction in glucose-6-phosphate and rise in alanine aminotransferase at 500 mg/kg; and hepatocellular lesions, including centrilobular hepatocellular hypertrophy and centrilobular necrosis. These changes were observed histologically at 200 and 1,000 mg/kg/day. In another study, the liver weights rose, relative to body weight in rats receiving concentrations of PCE at doses of 1,400 mg/kg/day in drinking water for 13 weeks. Gross examination of the animals during

autopsy did not show any abnormalities in the liver or other selected body organs. Most biochemical parameters examined did not indicate hepatotoxicity.

Toxic nephropathy and increased mortality were reported at all dose levels in rats and mice administered PCE in corn oil by gavage for 78 weeks, followed by a period of observation. Time-weighted-average (TWA) dose of 536 and 1,072 mg/kg/day for male mice, 386 and 772 mg/kg/day for female mice, 471 and 941 mg/kg/day for male rats, and 474 and 949 mg/kg/day for female rats were included in the study which compared exposed to controls. Degenerative changes in the kidney tubules with swelling, fatty degeneration, and necrosis of the tubular epithelium and hyaline intraluminal casts were detected. Following a gavage administered dose of 1,000 mg/kg of PCE to male rats for 10 days, compound-induced renal damage (i.e. rise in protein droplet accumulation and cell proliferation in a specific segment of the kidney) was shown. Renal pathology involving the α -2 μ -globulin was observed in male rats exposed to concentrations of 1,500 mg/kg of PCE by gavage for 42 days.

Dermal effects have also been detected in a study of humans with multiple chemical exposures. The population of Woburn, Massachusetts, was examined to determine health outcomes (particularly childhood leukemia) from ingestion of drinking water contaminated with solvents. Two of the solvents, trichloroethylene and PCE, were detected at higher concentrations than other substances. Family members (13/25) of leukemia cases, with lengthy exposure history to the contaminated water, were found to have skin rashes occurring twice a year for 2-4 weeks. The skin problems disappeared within 1-2 years after termination of exposure to the contaminated water.

Furthermore, immunological effects were observed in the Woburn study. Twenty-three adults, family members of children with leukemia, who were exposed to contaminated water, were found to have persistent lymphocytosis, rise in the number of T lymphocytes, and depressed helper:suppressor T cell ratio. Subsequent testing 18 months later demonstrated a decrease in lymphocytic counts and numbers of suppressor T cells, and a rise in the helper:suppressor ratio. Auto-antibodies were found in 48% of the adults tested (11/23).

Neurological effects, similar to those observed in inhalation exposure, have been reported in humans with oral exposures. PCE was used orally at one time for the treatment of worms. Doses ranging from 2.8 to 4 ml (around 4.2-6 g) produced narcotic effects, inebriation, perceptual distortion, exhilaration, but not death. A case study of a 6-year-old child who received a 12-16 g dose of PCE, resulted in the development of somnolence and coma. In addition, drowsiness, vertigo, agitation, and hallucinations were also manifested in the child.

Developmental effects have been reported in the Woburn study. An association was reported between eye/ear anomalies and central nervous system/chromosomal/oral cleft anomalies and ingestion of contaminated water. In addition to other solvents in the water, PCE was detected at 21 ppb. Scientists have disputed the biological relevance of the groupings of these anomalies.

A death following a 3 ml oral dose of PCE for treatment of hookworm was reported in a severely undernourished individual. The cause of death was unknown due to the pre-existent state of chronic malnutrition and septic cholecystitis.

The Woburn study also demonstrated a potential association between ingestion of contaminated water and a rise in the risk of childhood cancer (leukemia). Because of the presence of multiple chemicals in the drinking water, it was not possible to attribute the development of childhood leukemia to any specific substance.

Inhalation exposure to PCE has also affected a number of body systems. Respiratory irritation was documented in volunteers exposed to a concentration of 216 ppm for 45 minutes to 2 hours workers and in workers exposed to PCE at inhalation levels of 232-385 ppm. Human volunteers exposed to concentrations as high as 1,060 ppm could only endure an exposure period of one to two minutes before leaving the study chamber.

Cardiovascular effects (cardiac arrhythmia) were detected in a worker employed for seven months in a dry cleaning facility where his job required the use of PCE for cleaning clothes. The worker was without symptoms a month after finding new employment.

Hematological effects have also been observed with inhalation exposure. Polycythemia vera (disorder of bone marrow) was diagnosed in a male with a history of 22 years of exposure, including transient exposure concentrations above 300 ppm for five minutes out of any three hours. Because genetic and other environmental factors may increase the chances of developing the disease, PCE could not be singly associated with this disease outcome.

The liver has been a target organ in PCE exposure. Hepatocellular damage was diagnosed in a woman worker inhaling PCE fumes. In addition, a dry cleaner, who was exposed to PCE fumes, was found to have diffuse fatty liver and died shortly after the exposure. This condition may have existed prior to employment. No effect to the liver (as measured by the presence of liver enzymes - alanine aminotransferase) was observed in 22 dry cleaning workers who received a TWA exposure to PCE of 21 ppm.

Renal effects have additionally been observed based upon the inhalation dose. An examination of the workers with an estimated TWA of 10 ppm of PCE for 14 years demonstrated a rise in urinary levels of lysozyme and β -glucuronidase indicating mild tubular damage of the kidney. Serum creatinine and urinary albumin, β - μ -globulin and retinol-binding protein levels were found to be within normal limits in dry cleaning workers exposed to a TWA of 21 ppm of PCE for six years.

In addition, dermal/ocular effects (mild ocular irritation) were documented in four subjects at an exposure levels of 106 ppm or 216 ppm. Burning and stinging of the eyes was noted with exposures of 280 ppm or 600 ppm. An acute exposure of greater than 1,000 ppm resulted in intense eye irritation in humans.

Data to evaluate immunological effects from inhalation exposure are not firm. However, in one study, mice were found to be more sensitive to pulmonary bacterial infection following a three hour inhalation exposure to PCE at 50 ppm.

Neurological effects have additionally been observed in humans. The brain is the target organ for exposure by inhalation. Impaired perceptual and intellectual function and attention were detected in dry cleaning workers exposed to a TWA of 12 ppm (for 141 days) or 54 ppm (for 127 days) of PCE when compared to controls. In a separate study of dry cleaning workers, no significant changes in neurological symptoms or psychomotor performance were seen in the workers who received a TWA exposure to PCE of 21 ppm over an average of six years. However, in 17 of 22 subjects, neurologic symptoms (memory loss and difficulty sleeping) were more widespread in the exposed compared to the control group. In one study, headache, dizziness, difficulty speaking, and sleepiness occurred after inhalation exposure to 100 ppm of PCE for seven hours. After a one hour exposure to 106 ppm, volunteers of one exposure study manifested no symptoms of neurological impairment. At 216 ppm for an exposure period of 45 minutes to 2 hours, symptoms of dizziness and drowsiness developed. In a separate study, motor coordination was lost at an inhalation exposure concentration of 280 ppm for two hours or 600 ppm for 10 minutes. Additionally, mood change, slight ataxia, faintness, and dizziness were observed in four volunteer subjects receiving inhalation exposure to PCE at concentrations of 1,000-1,500 ppm for less than two hours. The subjects had the sense of impending collapse at exposure to 2,000 ppm for 5-7 minutes.

Reproductive effects have also been seen with occupational inhalation exposure to PCE in the dry cleaning business. Menstrual disorders and spontaneous abortions have been observed in female dry cleaning workers.

Genotoxic effects have not been consistent from inhalation exposure to PCE. Inhalation exposure concentrations of from 10-220 ppm for three months to 18 years did not result in genotoxic effects (sister chromatid exchanges and chromosome aberrations) to 10 workers with occupational exposure to PCE. In a separate study, sister chromatid exchange was significantly higher in exposed workers who smoked when compared to a group of controls who were non-smokers. The workers received a TWA of 10 ppm for eight hours. Epidemiologic research has shown a potential association between chronic PCE exposure and an

increased cancer risk. The findings are limited due to the simultaneous exposure to a number of chemicals. In one investigation, a subcohort (consisting of 615 dry cleaning workers employed in shops where PCE was the main solvent) was examined. The workers had no history of exposure to petroleum solvents. Excess cancer risk was not observed in this subcohort. However, the main cohort, consisting of 1,690 workers with a number having petroleum solvent exposure, had a significant increase in mortality from kidney, bladder, and cervical cancer. In two other epidemiological studies of laundry and dry cleaning workers, an increased risk of bladder cancer was not observed in workers who were compared with controls. Additional studies of dry cleaning and laundry workers have demonstrated significant increases in mortality from lung, cervical, esophageal, renal, dermal, lymphatic/hematopoietic system, and/or colon cancers. In one study, an increased risk of primary liver cancer was found in male workers with classifications of craftsman or operators of laundry or dry cleaning operations. Additionally, a retrospective cohort study of 14,457 aircraft maintenance workers examined mortality in relation to occupational exposure to over 20 solvents which included PCE. Female workers who were exposed to PCE for at least one year were found to have elevated death rates due to multiple myeloma or on-Hodgkin's lymphoma. In contrast, a cohort of white male chemical workers, having multiple chemical exposures which included PCE, was examined and found not to have an increased risk for total mortality or cancer. PCE has an EPA Weight-of-Evidence Classification of B2 (probable human carcinogen). EPA is presently reviewing PCE's Weight-of-Evidence classification and slope factor. Pending EPA's final report, this study utilizes the existing information on classification and slope factor.

Very little information is available for health effects from dermal exposures. Chemical burns (redness, blistering, sloughing of skin) have been observed with extended exposure (greater than five hours) in dry cleaning operations. Intense ocular irritation have been documented in humans following acute exposure to PCE vapors at concentrations greater than 1000 ppm.

The interactive effect of certain chemicals in the presence of PCE has resulted in conflicting outcomes. An epoxide intermediate is produced from PCE metabolism and is believed to be the toxic agent in the development of adverse health effects such as liver tumors in rodents. Any substance (e.g. ethanol, phenobarbital, polychlorinated biphenyls) which stimulates PCE metabolism would be expected to increase PCE's toxicity. Animal experiments have demonstrated that pretreatment with PCBS did stimulate metabolism as evidenced by the increase in hepatotoxicity and the presence of urinary metabolites for PCE. However, ethanol and phenobarbital failed to increase PCE toxicity. Urinary metabolites were reduced when Chinese dry cleaning workers were exposed to both PCE and TCE and not TCE alone.

Trichloroethylene (TCE)

Trichloroethylene (CAS No. 79-01-6, C_2HCl_3) is a halogenated hydrocarbon. Synonyms include 1-chloro-2,2-dichloroethylene; 1,1-dichloro-2-chloroethylene; ethylene trichloride; and 1,1,2 trichloroethylene. TCE is used as an industrial solvent and degreaser, an intermediate for manufacturing other chemicals, and is commonly used in the automotive, metal, and textile industries. In the past, it has also been used as a general and obstetrical anesthetic, surgical disinfectant, and extractant of caffeine for decaffeinated coffee. Although TCE is liquid at a room temperature, evaporation does occur in industrial processes resulting in exposure by inhalation for workers and the general public residing in areas of industry and waste disposal sites. The degreasing operation in industry is the primary cause of TCE emissions into the environment with releases also occurring from other industries and disposal of waste. Due to the ease with which it travels through soil, groundwater contamination with TCE is common. Since vaporization does not occur in subsurface areas, TCE's persistence in groundwater is evidenced by its detection in a large number of monitoring studies. Exposure may also result from contact or ingestion of food and water contaminated with TCE.

Routes of exposure for TCE include inhalation, ingestion, or dermal contact. Human absorption following inhalation or ingestion is extensive, but poor with dermal exposure. Studies on the distribution of TCE have been done on humans but primarily in animals and have demonstrated deposition in the blood and

fat. TCE has been found in the blood of babies at birth following TCE anesthesia in the mother. With oral exposure, TCE has been observed in fatty tissue in animals while dermal exposure resulted in the detection of TCE in the blood of humans. In animals and humans, TCE metabolism occurs primarily in the liver following inhalation exposure. In addition to the liver, metabolism of TCE following inhalation also appears to occur in the kidneys and lungs of animals. Major metabolites are common to animals and humans. In humans, excretion of TCE occurs in the urine and by exhalation through the lungs following inhalation exposure. The same excretory pathway is also found in animals following ingestion exposure. With dermal exposure, excretion of TCE was by exhalation in subjects whose hand was submerged in a solution of TCE.

Neurological effects have been observed in animal ingestion studies with TCE. Ataxia, lethargy, convulsions, and hind limb paralysis were observed in rats administered gavage doses of 500 or 1,000 mg/kg for five days a week up to 103-104 weeks. In a separate study, mice were observed to experience a period of excitation with a subsequent subanesthetic state following exposure to TCE by gavage at doses of 1,800 mg/kg (females) or 2,400 mg/kg (males) for a period of five days a week for 35 weeks.

Hepatic lesions were not detected in persons accidentally ingesting TCE. However, rapid death following ingestion of TCE may have prevented the development of these lesions. Animal studies, however, have shown hepatic changes, including increase in the weight of the liver, at gavage doses of 240 mg/kg or greater for periods of two weeks or more. Yet, another study of rats demonstrated no non-neoplastic effects to the liver at gavage doses of 500 or 1,000 mg/kg for a period of five days a week for 103 to 104 weeks.

Renal effects have also been noted in animals with ingestion exposure. Kidney effects, which included a rise in ketone and protein levels in urine, have been documented in rodents receiving TCE in drinking water at a dose of 393 mg/kg/day or greater for a period of six months. This condition may suggest renal dysfunction. Treatment-related nephropathy has also been detected at gavage doses of 500 mg/kg or greater for a five day a week period of 103 weeks or more of exposure. In contrast, a separate study revealed that kidney weights and histology were not out of the ordinary for mice with gavage doses of 250, 500, 1,200, or 2,400 mg/kg of TCE for an exposure period of five days a week for three weeks.

Data on the immunotoxic effects in humans were unavailable and inconclusive in animals. Effects to the hematological system have also been observed in animals. A 5% lower hematocrit was reported at a gavage exposure level of 240 mg/kg/day for 14 days in male mice. In addition, a reduction in erythrocyte count was seen in male mice at 660.2 mg/kg/day of TCE administered in drinking water after four and six months.

Case reports of humans indicate that TCE can produce cardiac arrhythmias at an ingestion dose of 350 to 500 ml.

Additionally, intestinal lesions have been seen in gavage-treated mice at doses of 216.7 or 660.2 mg/kg/day for six months.

Developmental effects, which included perinatal deaths and congenital anomalies, have been observed in humans with exposure to well water contaminated with a number of chlorinated hydrocarbons, including TCE. TCE was detected at 267 ppb while other substances were detected at much lower levels (e.g. tetrachloroethylene at 21 ppb). Due to the study's limitations which included multiple chemical exposures, the association between TCE and disease outcome could not be established.

Reproductive effects of impaired copulatory behavior were observed in male rats after a week of TCE exposure by gavage at 1,000 mg/kg for five days a week for a period of six weeks.

Human death has been documented following an accidental ingestion of TCE (dose unknown) with cause of death documented as hepatorenal failure.

Potential carcinogenic effects from ingestion exposure to TCE have also been observed. An elevated rate of childhood leukemia was observed in a study conducted in Woburn, Massachusetts. In a follow-up investigation of the same community, the leukemia cases were found to be significantly associated with access to wells with drinking water contaminated with TCE and other chlorinated organic compounds. Due to the limitations of the study (e.g. multiple chemicals in the drinking water), the cause of the leukemia was not

established. Significant increases have also been observed in the incidence of hepatocellular carcinoma in mice treated by gavage with TCE at 1,000 mg/kg for a period of five days a week for 103 weeks. In addition, a dose-related increase has been seen in the incidence of leukemia in male rats receiving 50 or 250 mg/kg of TCE for a period of four to five days a week for one year.

Inhalation exposure has also resulted in health effects. Neurological outcomes have been observed in humans following exposure by inhalation. Concentrations of 0, 27, 81, or 201 ppm of TCE were administered to groups of three human subjects for four hours. Irritation of the eyes and throat and drowsiness were observed at 27 ppm or greater, headache at 81 ppm or greater, and dizziness and anorexia at 201 ppm. In another study, subjects were found to have a significant reduction in performance on a perception test, the Wechsler Memory Scale, a complex reaction time test, and manual dexterity tests with inhalation exposure of 110 ppm of TCE for two four hour exposures with an interval of 1.5 hours. In contrast, no significant treatment-related effects on behavioral task performance were reported in subjects receiving 95 ppm or 150 ppm or 300 ppm of exposure to TCE for 2.5 hours. Subjects were tested for reaction time, hand steadiness, hand tapping, and pursuit tracking. In a separate study, however, eight male human volunteers were found to have a compromise in visual-motor skills. Subjects were exposed to 0, 100, 300, or 1,000 ppm of TCE for two hour periods with an interval of three days between exposure sessions. A significant reduction in visual-motor performance was seen at an exposure of 1,000 ppm. Subjective symptom, neurological, and psychiatric evaluations have been done in yet another study. Workers who had been employed an average of 3.75 years in industrial operations utilizing TCE as a solvent were found to have a greater likelihood of complaints which included vertigo, fatigue, and headaches with test results showing short-term memory loss, fewer word associations, and increase misunderstanding at higher mean concentrations of TCE (85 ppm). The high-dose workers were compared to those exposed to lower mean concentrations of TCE (14 or 34 ppm).

Severe liver damage in the form of necrosis has been observed in acute occupational inhalation exposure to lethal concentrations of TCE.

Kidney dysfunction and failure have also been seen with inhalation exposures in acute occupational and intentional exposure.

Immune system effects as manifested by the change in weight of the thymus gland have been observed in rats receiving continuous exposure at 800 ppm of TCE.

Hematological effects in the form of depression of delta-aminolevulinic acid dehydratase activity was reported in rats with continuous inhalation exposure for 10 days. The reduced activity was detected in liver and bone marrow cells at 50 ppm or greater and in erythrocytes at 398 ppm or greater.

Male mice experienced a reduction in the weight of the spleen with continuous exposure to TCE at 150 ppm for 120 days. The toxicological significance of this weight reduction is unknown.

The respiratory system has also been affected as manifested by a rise in the rate of respiration and a reduced alveolar ventilatory amplitude when TCE was administered in anesthetic concentrations to humans.

When used as an anesthetic in humans, an association has been observed between TCE and the development of cardiac arrhythmia (i.e. bradycardia, atrial and ventricular premature contractions, and ventricular extrasystole).

Chronic gastrointestinal system effects (anorexia, nausea, vomiting, and intolerance to fatty foods) have been detected in occupational inhalation exposure to TCE. In addition, a large percentage of workers with primary pneumatosis cystoides intestinalis (PCI) had a history of occupational exposure to TCE. PCI manifests as thin-walled, gas-containing cysts in the intestinal wall.

Developmental toxicity studies have shown that no malformed babies were born to mothers from a cohort of 2,117 Finnish workers, who were exposed to TCE between 1963 and 1976. In an animal study, however, litter resorption, reduced fetal body weight, and skeletal ossification anomalies were observed in the fetuses of rats, who were exposed by distillation to TCE. The exposure concentration was 100 ppm for four hours a day on days 8 to 21 of gestation.

Reproductive toxicity has also been observed with inhalation exposure. A significant rise in sperm morphology abnormalities was seen in mice, exposed to 2,000 ppm of TCE for a period of four hours a day for five days.

Potential genotoxic effects have been observed with TCE inhalation exposure of workers. Workers exposed to TCE up to 75 ppm for 1 to 21 years were observed to have a rise in hypoploid cells. In yet another study, six workers who were exposed to TCE were reported to have an increase in sister chromatid exchanges. Since multiple chemical exposures were involved, these studies were not conclusive in associating genotoxic effects to TCE.

TCE exposure has proven fatal in a number of industrial operations. Four men died suddenly after being exposed to TCE by inhalation in degreasing operations. The cause of death was believed to be ventricular fibrillation. The TCE exposure concentrations were 200 to 8,000 ppm at the working area of one of the workers who died. In another case, a man died approximately 17 hours after cleaning a vat containing TCE at an electroplating shop. A post-mortem examination revealed fatty degeneration of the liver and old and new lung hemorrhages. Air samples obtained after the death of a male dry cleaning operator revealed TCE breathing zone concentrations of 2,900 ppm.

A number of epidemiologic studies have been done examining the effect on workers from inhalation exposure to TCE. A significant increase in bladder cancer and lymphomas was detected in a cohort of 1,424 men with unspecified exposure to TCE. In another study, a significant rise was also discovered in the incidence of lung/bronchus/trachea, cervix, and skin cancers in over 330 deceased cleaning and/or laundry workers. Exposures were mainly to tetrachloroethylene. The TCE exposure was not well documented. In contrast, fewer deaths from non-respiratory cancer were found in white males from a group of 2,646 employees of a manufacturing plant that used TCE. The workers were employed for three months or longer during 1957 to 1983. However, an association was observed between cancer of the naso- and oropharynx and exposure to TCE and cutting oil. The researchers believed that the cutting oil was more likely than TCE to be involved in the cancer formation.

Studies of the health effects from dermal exposure are sparse. In one study, dermal application of TCE made on the skin of rabbits resulted in the death of 50% of the animals at a concentration of 29g/kg. Purified TCE (1 mg in acetone) was spread on the shaved skin of female mice. A significant rise in the incidence of tumor formation was not observed in this study. TCE has an EPA Weight-of-Evidence Classification of B2 (probable human carcinogen). EPA is presently reviewing TCE's Weight-of-Evidence classification and slope factor. Pending EPA's final report, this study utilizes the past information on classification and slope factor.

TCE interacts with a number of substances which either increase or inhibit its effect. At low concentrations of ingested alcohol, inhaled TCE metabolism is enhanced while high does of alcohol restrict the metabolism. TCE causes the heart to be more susceptible to epinephrine-induced cardiac arrhythmia in animals. Phenobarbital and 3-methylcholanthrene promoted the injury to the liver caused by TCE metabolites. The liver toxicity of carbon tetrachloride in rats is also known to be enhanced by TCE. In addition, a TCE metabolite enhances the anti-clotting effect of warfarin.