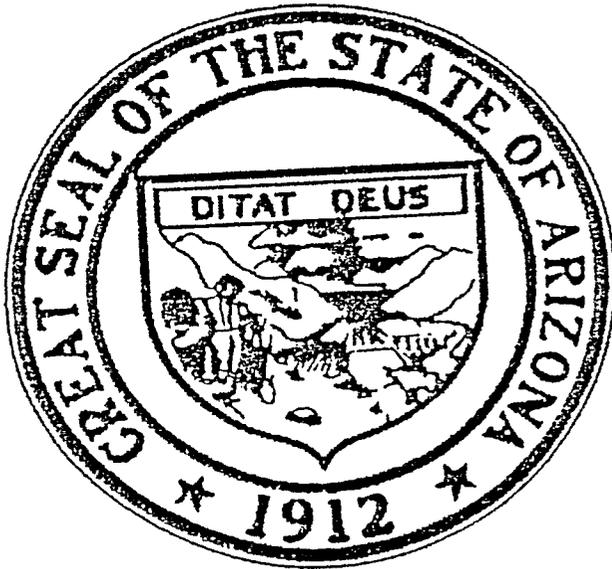


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BASELINE RISK ASSESSMENT

MOTOROLA, INC. 52nd STREET FACILITY
Phoenix, Arizona



Prepared For

ARIZONA DEPARTMENT OF ENVIRONMENTAL QUALITY
under the
Water Quality Assurance Revolving Fund

Prepared By

ARIZONA DEPARTMENT OF HEALTH SERVICES
Division of Disease Prevention
Office of Risk Assessment and Investigation

November, 1992

FINAL



ARIZONA DEPARTMENT OF HEALTH SERVICES

Division of Disease Prevention

FIFE SYMINGTON, GOVERNOR
DOUG CAMPOS-OUTCALT, MD, MPA,
ACTING DIRECTOR

February 17, 1993

Mr. Michael Montgomery
Superfund Program
Environmental Protection Agency
75 Hawthorne Street
San Francisco, California 94105

Dear Mr. Montgomery:

A recent review of the Baseline Risk Assessment, Motorola 52nd Street Facility revealed inconsistencies in the use of the terms "hazard quotient (HQ)" and "hazard index (HI)" for both systemic and carcinogenic toxicants. I am providing copies of the risk assessment that include the changes. An errata page showing the changes and locations is provided with each manuscript.

Please withdraw any older versions from circulation and replace with these.

I am also providing copies to Jeffery Kulon at DEQ. If you have any questions, please call me at (602) 230-5874.

Sincerely,

Win Chromec, Ph.D.
Manager, Risk Assessment and
Environmental Epidemiology

WC:tr

Enclosures

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Motorola 52nd Street Risk Assessment

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LIST OF ABBREVIATIONS, ACRONYMS AND UNITS

ADEQ	Arizona Department of Environment Quality
ADHS	Arizona Department of Health Services
ADWR	Arizona Department of Water Resources
Ag	Silver
ARS	Arizona Revised Statutes
As	Arsenic
AT	Averaging Time (days)
ATP	Acid Treatment Plant (Motorola 52nd Street)
B	Boron
Ba	Barium
BDCM	Bromodichloromethane
Be	Beryllium
BMM	Bromomethane
BNZ	Benzene
BRFM	Bromoform
BW	Body Weight (kg)
Ca	Calcium
CA	Chemical Concentration in Air (mg/m ³)
CASRN	Chemical Abstract Service Registry Number
CCL4	Carbon Tetrachloride
Cd	Cadmium
cDCP3	cis-1,3-Dichloropropene
CDI	Chronic Daily Intake
CERCLA	Comprehensive Environmental Response Compensation and Liability Act of 1980 (Superfund)
CEVE	2-Chloroethylvinyl Ether
CF	Conversion Factor
CFS	Cubic Feet per Second
CHI	Cancer Hazard Index
CHQ	Cancer Hazard Quotient
Cl	Chloride
CM	Chloromethane
Cn	Cyanide
CNS	Central Nervous System
CO ₂	Carbon Dioxide
COPC	Contaminants of Potential Concern
Cr	Chromium
CT	Census Tract
Cu	Copper
CW	Chemical Concentration in Water (ug/L)
DBCM	Dibromochloromethane
DCA	1,1-Dichloroethane
DCA2	1,2-Dichloroethane
DCB2/4	1,2/1,4-Dichlorobenzene

List of Abbreviations, Acronyms and Units--continued

DCB2	1,2-Dichlorobenzene
DCB3	1,3-Dichlorobenzene
DCB4	1,4-Dichlorobenzene
DCDFM	Dichlorodifluoromethane
DCE	1,1-Dichloroethylene
DCE2	1,2-Dichloroethylene
DCM	Dichloromethane
DCP2	1,2-Dichloropropane
EC	Ethyl Chloride
ED	Exposure Duration
EF	Exposure Frequency
ELCR	Excess Lifetime Cancer Risk
EKG	Electrocardiogram
EPA	U.S. Environmental Protection Agency
ET	Exposure time (hours/day)
ETB	Ethylbenzene
F	Fluoride
F113	Trichlorotrifluoroethane
Fe	Iron
GI	Gastrointestinal Tract
HBGL	Health-Based Guidance Level
HEAST	Health Effects Assessment Summary Tables
Hg	Mercury
HI	Hazard Index (Systemic)
HQ	Hazard Quotient (Systemic)
IQ	Intelligence Quotient
IR	Ingestion Rate
IRIS	Integrated Risk Information System
K	Potassium
kg	kilograms
MCB	Chlorobenzene
MCL	Maximum Contaminant Level
MF	Modifying Factor
mg/L	milligrams per Liter
mg/m ³	milligrams per meter ³
mg/kg	milligrams per kilogram
Mg	Magnesium
Mn	Manganese
Na	Sodium
NCI	National Cancer Institute
NH ₃	Ammonia
Ni	Nickel
NO ₃	Nitrate
NOAEL	No-observed Adverse Effects Level
NPL	National Priority List

List of Abbreviations, Acronyms and Units--continued

Pb	Lead
PCE	Tetrachloroethylene
ppb	Parts Per Billion
ppm	Parts Per Million
PVC	Polyvinyl Chloride
RAGS	Risk Assessment Guidance for Superfund
RAP	Remedial Action Plan
RfC	Reference Concentration (inhalation)
RfD	Reference Dose (ingestion)
RI/FS	Remedial Investigation/Feasibility Study
RME	Reasonable Maximum Exposure
Sb	Antimony
Se	Selenium
SF	Slope Factor
SiO ₂	Silica
SO ₄	Sulfate
SQL	Sample Quantification Limit
SRP	Salt River Project
SWPL	Southwest Parking Lot (Motorola 52nd Street)
TCA	1,1,1-Trichloroethane
TCA2	1,1,2-Trichloroethane
TCE	Trichloroethylene
tDCP3	trans-1,3-Dichloropropene
TDS	Totally Dissolved Solids
TE	1,1,2,2-Tetrachloroethane
TFM	Trichlorofluoromethane
Tl	Thallium
TOL	Toluene
UCL	Upper Confidence Limit
UF	Uncertainty Factor
ug/L	micrograms per Liter
ug/kg	micrograms per kilogram
USEPA	U.S. Environmental Protection Agency
VC	Vinyl Chloride
VOC	Volatile Organic Compound
WoE	Weight of Evidence
WQARF	Water Quality Assurance Revolving Fund
XYL	Xylene (total)
Zn	Zinc

EXECUTIVE SUMMARY

The purpose of this risk assessment is: to evaluate the potential and current public health risks associated with chemical contamination at the Motorola 52nd Street Facility; to provide a basis for determining additional alternative response actions; and to provide information for developing preliminary environmental media-specific remediation goals.

Contaminants that have been used, stored, or disposed at the facility include trichloroethylene, tetrachloroethylene, and trichloroethane. Other volatile organic compounds (VOCs) have been detected in groundwater which may be a result of impurities in the released compounds, degradation, or undocumented releases from unknown sources. These VOC's include vinyl chloride, chloroform, 1,2-dichloroethane, and dichloroethylene. Inorganics observed in the sample analysis include arsenic, boron, fluoride, lead, nitrate, sulfate, and thallium. All compounds detected at the Motorola 52nd Street site are reported in Section 2.0. Table 2.1 lists detected organic compounds, and Table 2.2 lists detected inorganic compounds.

Fifty-four wells were sampled. These included specially installed monitor wells, both on- and off-site; SRP owned irrigation wells, state-owned wells, and private domestic wells. Thirty-six (36) chemicals of potential concern (COPC) were identified. The list of COPC is reported in Table 2.5.

The primary uses of groundwater within the investigation area are irrigation and industrial. There are no municipal supply wells in the area. A private well located on the northern perimeter of the study area (well 4626G), with a history of use for irrigation, swimming and short-term domestic use, was sampled. The analyses conducted on the private well from 1987 through 1992 indicate that only boron, fluoride, and lead were in excess of HBGLs or MCLs.

Complete, or potentially complete, exposure pathways, for this risk assessment, include ingestion, inhalation and dermal contact exposures to soil, soil gas, and groundwater for current and future residential and occupational receptor populations. Table 3.2 provides a detailed summary of the exposure pathways evaluated in this risk assessment.

Current federal and state guidance emphasizes the use of health-conservative assumptions in the risk assessment process. The potential for adverse health effects is defined using conditions that produce upper bound estimates of risk. Consequently, final health risk estimates are unlikely to under-estimate, and may significantly over-estimate, risks. This risk assessment should not be construed as presenting an absolute estimate of potential risk to human health. It is a conservative analysis intended to provide an estimate of potential and current human health risks resulting from uncontrolled chemical releases at the Motorola 52nd Street facility. It is intended for use in the risk management process. The risk values developed for this study are presented in Chapter 5.0.

This risk assessment is an evaluation of the *no-action alternative*. The existence of baseline risks which do not meet the protectiveness criterion only indicate that remediation may be required. The following observations and recommendations are made based on the results of the baseline risk assessment:

- 1) Significant groundwater contamination exists within the Motorola 52nd Street study area. Estimated risk from potential domestic exposures to groundwater reach a RME maximum of $9E-01$ at one on-site monitor well. However, the impacted groundwater is not used in a public drinking water system. The risk of public exposure to groundwater is considered limited, causing no imminent health hazard. Refer to Table 5.3 and Figures 5.1 through 5.9.
- 2) Increased monitoring of the private domestic well (2646G) is recommended to better define the extent of exposure and subsequent risk. Current data do not indicate excessive risk associated with the use of the well for irrigation, or swimming. Over 98% of the current risk from domestic use of the well is from arsenic, present at levels below the MCL. Refer to Tables 5.3, 5.4, 5.7 and 5.8.
- 3) Risks due to ingestion, inhalation, and dermal exposures to surface soil, on-site, could not be quantitatively assessed due to a lack of recent data.
- 4) Continued monitoring of the area's groundwater should be maintained for plume definition and migration tracking.
- 5) The 1985 and 1992 soil gas data indicate no excess risk to residents to the west of the facility. A single 1985 on-site sampling location had a risk greater than $1E-06$ for occupational indoor exposures. None of the 1989 or 1991 on-site soil gas samplings had associated occupational risk values above $1E-06$. One 1989 outdoor air sample had an associated risk of $1E-06$. It is impossible to determine if this was due to soil gas releases or other sources. Refer to Tables 5.9 through 5.18.

1.0 INTRODUCTION

The purpose of this human health risk assessment is to determine the extent and likelihood of adverse health effects that may result from exposure to chemical contamination of groundwater, soil, and soil gas resulting from uncontrolled releases of potentially toxic substances, as documented, from the Motorola Inc., 52nd Street site.

1.1 AUTHORITY

Pursuant to Chapter 7, Article 1 of the Arizona Revised Statutes, Water Quality Assurance Revolving Fund (WQARF), A.R.S. 49-282; and the Comprehensive Environmental Response Compensation and Liability Act of 1980 (CERCLA/Superfund Act, 42 U.S.C. 9601 et. seq.), this human health risk assessment has been prepared in accordance with the requirements of Contract Number 2207-000000-3-3-DR-8074 for the Arizona Department of Environmental Quality (ADEQ) in accomplishment of Task Assignment 26, Motorola Inc., 52nd Street, Phoenix, Arizona. Compliance with Arizona Administrative Code (A.A.C.) R18-7-108, part B.3 of the Remedial Action Plan is hereby provided, conducted under the guidelines prescribed by the U.S. Environmental Protection Agency (USEPA) Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual: Part A (1989a). The components of the remedial action are described in detail in the following Dames & Moore reports: Draft Remedial Investigation and Feasibility Study Reports (1987a); Draft Remedial Action Plan (1988); and Draft Final Remedy Remedial Investigation Report (1991a). Satisfaction of A.A.C. part B.4, a health effects study, was previously submitted to ADEQ by Arizona Department of Health Services (ADHS) under Task Assignment 13, contract number 2207-000000-3-3-DR-8074, August 1990.

1.2 OVERVIEW

A routine audit of Motorola records in November 1982, revealed a discrepancy in the chemical inventory. Subsequent investigation indicated a leak of 1,1,1-Trichloroethane (TCA) had occurred from a 5,000 gallon underground tank. Hydrogeological studies conducted in response to the preliminary investigation, determined significant levels of TCA and other chemicals in soil and groundwater on and near the Motorola site and the migration of contamination in groundwater (Gutierrez-Palmenberg, Inc. Preliminary Report, Chemical Leak Project, 1983, Dames & Moore Water Quality Data Usability Report, Sampling Rounds 8 through 14, Final Remedy RI/FS Studies, 1991b, and Final Remedy RI Report, 1992).

In the following report the term study area will be used to denote the area surrounding the Motorola facility, extending from 52nd Street to 24th Street and from Palm Lane on the north to Washington Street on the south. This area corresponds to that for which groundwater has been sampled for the RI/FS.

1.3 GOALS AND OBJECTIVES

The scope of this baseline risk assessment is to assess the risks that are associated with the uncontrolled (non-permitted) releases that have occurred at the Motorola 52nd Street facility. The goal is to provide risk information necessary to assist decision-making within the risk management process. The specific objectives of this assessment are:

- A. Provide an evaluation of baseline risks (current or potential risks if no remedial action is taken) associated with the uncontrolled releases that have occurred at the facility and assist in determining site-specific actions.
- B. Provide a basis for determining cleanup levels protective of human health.
- C. Provide information to facilitate the comparison of potential health impacts of remedial alternatives (to be completed at a later date).

1.4 SITE BACKGROUND

The Motorola 52nd Street facility is an electronic components manufacturer that commenced operations in 1956 and has remained in production for 35 years without interruption. Numerous expansions have taken place during this time. The facility occupies approximately 90 acres of industrial property with more than 20 permanent production and administrative buildings (Figure 1.1). The facility, located in the eastern part of the City of Phoenix, is about one mile east of the Old Crosscut Canal, and the Hohokam and Papago Freeway interchange. The facility boundaries are McDowell Road to the north, 52nd Street to the east, Garfield Street to the south, and 50th Street to the west. Legal description of Motorola 52nd Street site is contained in Table 1.2 of Dames & Moore Draft Remedial Action Plan (1988). Major geographic features in the area include the Papago Buttes to the east of the facility, the Salt River flowing westerly about one mile to the south, and the Grand Canal, which flows northwesterly to the west of 40th Street and Van Buren Street. Phoenix Sky Harbor Airport is located approximately 1.5 to the southwest. The Phoenix Military Reservation, a 0.75 square mile area used by the Arizona National Guard, is located northeast and east of the site (Figure 1.2).

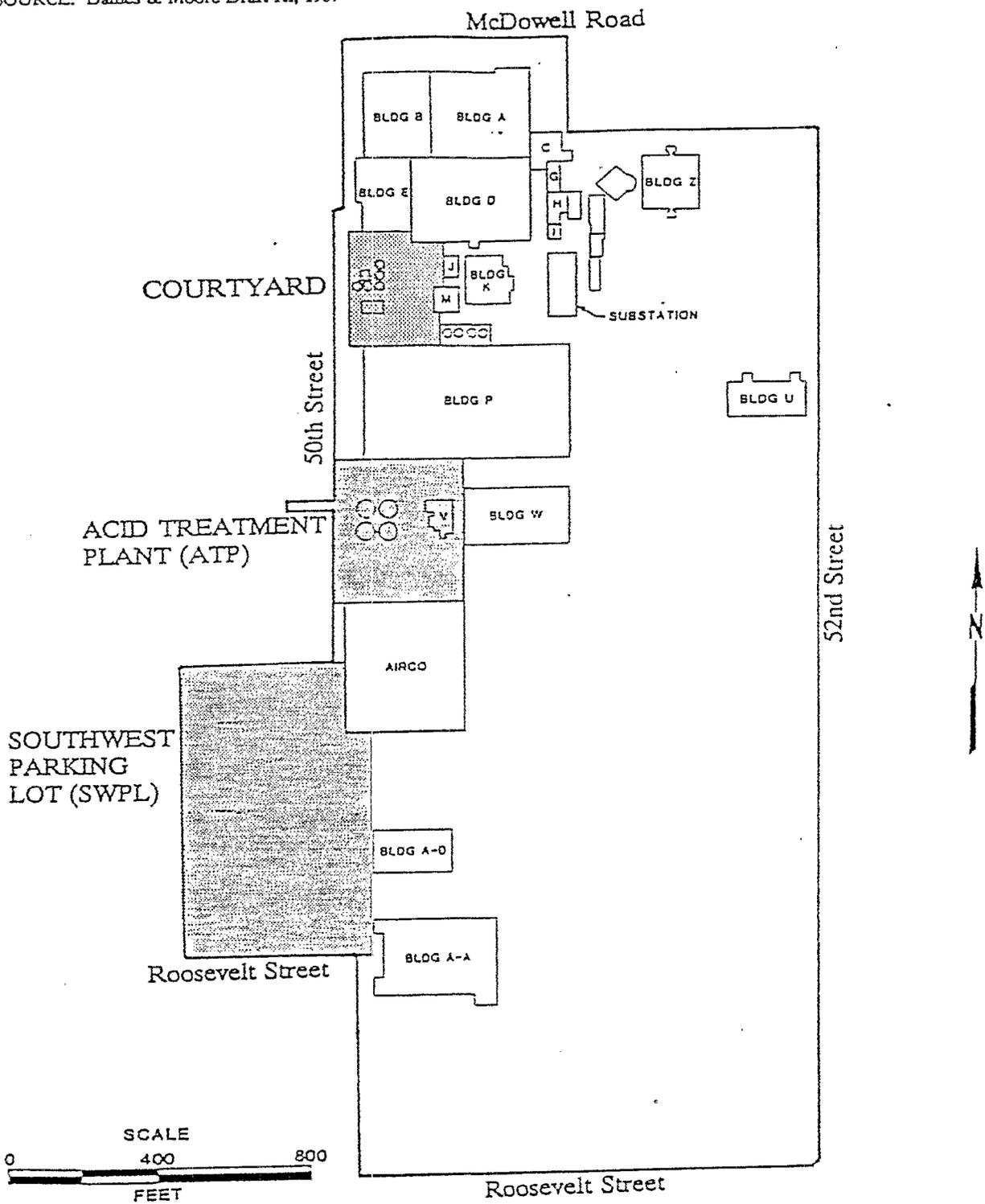
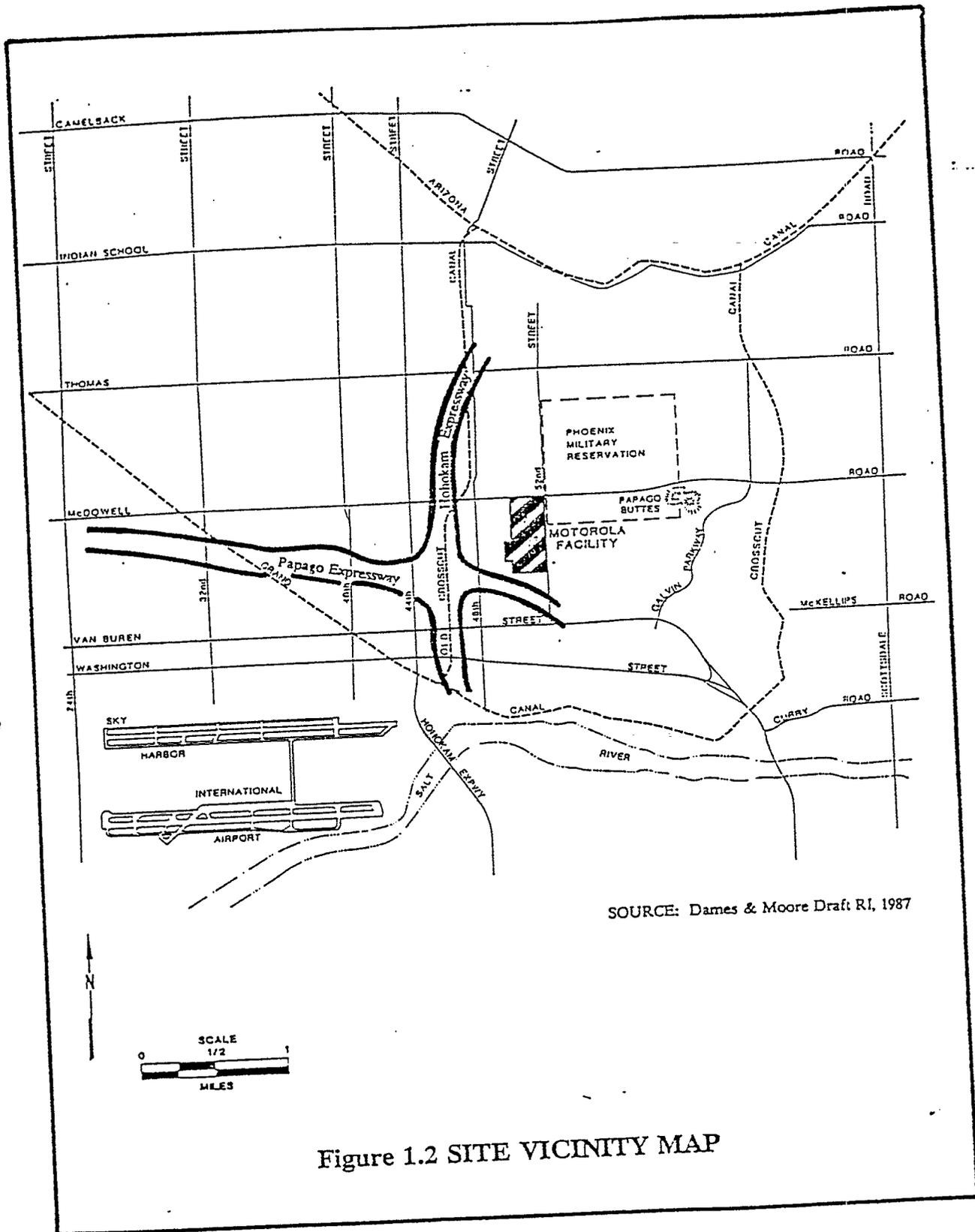


Figure 1.1 MOTOROLA 52ND STREET FACILITY MAP



SOURCE: Dames & Moore Draft RI, 1987

Figure 1.2 SITE VICINITY MAP

Hydrogeologic studies began in January, 1983, in response to reported leaking underground storage tanks. Results of initial investigations indicated that TCA and several other volatile organic compounds (VOCs) were present at concentrations in excess of state action levels at the time. In December 1983, Motorola Inc. entered into verbal agreement with the U.S. Environmental Protection Agency (EPA) Region IX, the Arizona Department of Water Resources (ADWR) and ADHS to define the nature and extent of contamination and develop remediation actions. Motorola initiated a pilot groundwater treatment plant at the facility in 1986. Contaminated groundwater has been treated from two on-site extraction wells and used in the manufacturing process to replace potable water supplied from the City of Phoenix.

Studies since 1983 have focused on defining of the distribution of VOCs in groundwater beneath the facility, determination of probable sources, and the extent of the off-site migration in groundwater. Aquifer monitoring began in 1983. Organic compounds, particularly VOCs, were detected in high concentrations in source areas. Off-site migration has been extensive. Significant soil contamination by VOCs has also been found at the Acid Treatment Plant (ATP) and Southwest Parking Lot (SWPL).

A Technical Subcommittee that included representatives from EPA, ADHS, ADWR, City of Phoenix, City of Scottsdale, Salt River Project (SRP), and Motorola was organized in 1983 to provide review and guidance for the technical aspects of the RI/FS. In 1984, the Motorola 52nd Street facility was proposed for listing on the National Priority List (NPL, Federal Superfund). Based on hydrogeologic and water quality studies described in guidelines for Superfund sites, the Motorola 52nd Street facility was listed on the NPL in 1989. Also, in 1989, the State of Arizona and Motorola Inc. signed the Motorola 52nd Street Consent Order, which implemented the Operable Unit, and required completion of the Remedial Investigation and Feasibility Study (RI/FS) describing the activities and schedules for completion of the final remedy. Remediation of the site will continue under the provisions of CERCLA and WQARF, administered by EPA Region IX and ADEQ respectively. The Draft RI was submitted in June, 1987; a Draft Remedial Action Plan (RAP) in June, 1988; a Draft Work Plan for the final remedy RI/FS in September, 1990; and a Draft Final Remedial Investigation Report was completed in September, 1991. A Final Remedy Remedial Investigation Report has been submitted in February, 1992.

1.5 SCOPE OF RISK ASSESSMENT

The baseline risk assessment evaluates risks associated with exposures of both human and ecological receptors, under present land-use conditions, to chemicals from sources addressed in the consent decree. Alternative land-use options, other than industrial, are not considered reasonable for the

Motorola Inc. site within the next 50 years, and therefore, are addressed equally under present land-use. The urban setting and nature of the contamination indicates that no biological resources are likely to be impacted. At present there are two known potential groundwater exposure points; (1) the private well, 4625G, is known to have drawn ground-water for irrigation, swimming pool, and domestic use since initial detection of contamination; and (2) SRP well 18E-5N which pumps into the Grand Canal when in use. These are addressed in Section 3.3.3 of this risk assessment. These are the only known complete or potentially complete exposure routes to groundwater. Other private well exposures may exist due to the lack of statutory barriers, although none have been documented.

This risk assessment is based on groundwater and soil data collected during the RI monitoring by Dames and Moore. Data collected from 1988 through 1991, inclusive, are utilized for the assessment of potential risks associated with groundwater use. Pre-1988 data were not considered due to the dynamic nature of the groundwater contaminant plume (Dames and Moore, 1991c), and abandonment of monitor wells within the right-of-way of the Hohokam and Papago expressways. Risks associated with exposure to VOC vapors released from the soil are based on data collected in 1984 and 1985, limited data for the on-site area collected in 1989 and 1991, and a survey of off-site areas completed in March 1992. The 1984 - 1985 soil gas data are included for comparison to the 1992 data. It is recognized that concentrations may have changed since the 1984 - 1985 sampling.

1.6 REPORT APPROACH AND ORGANIZATION

This baseline risk assessment is prepared in accordance with guidelines for risk assessments of Superfund sites published by the EPA (1988a,b, 1989a,b, 1990b, 1991a). The following four step approach was used for this risk assessment: 1) identification of chemicals of potential concern; 2) exposure assessment; 3) toxicity assessment; and 4) risk characterization. Organizational format of the risk assessment is:

(Chapter 1.0) **Introduction** -- Description of the contamination problem and overview of the site.

(Chapter 2.0) **Identification of Chemicals of Potential Concern** -- Description of the process and selection of chemicals of potential concern based on RI data.

(Chapter 3.0) **Exposure Assessment** -- Identification of complete or potential exposure pathways and quantification of human intakes.

(Chapter 4.0) **Toxicity Assessment** -- Identification of hazard and dose response data for chemicals of concern.

(Chapter 5.0) **Risk Characterization** -- Presentation and discussion of current and potential human health risks associated with the site.

(Appendices) -- Supporting data, informational displays, worksheets, etc.

2.0 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

This section identifies the chemicals of potential concern (COPC) for the Motorola Inc. 52nd Street Superfund site. The discussion addresses the data evaluated from the Remedial Investigation (RI); the methodology used in the identification process; the criteria for selection; and determination of the extent and levels of contamination. The above components are described under their respective sections:

- * Section 2.1 - Summarizes possible sources of the contamination.
- * Section 2.2 - Evaluates RI data available for use in the risk assessment.
- * Section 2.3 - Presents methodology used to select chemicals of potential concern.
- * Section 2.4 - Summarizes data used in the risk assessment.
- * Section 2.5 - Identifies uncertainties related to the data collection and evaluation.

2.1 SOURCES OF CONTAMINATION

On-site disposal locations historically included leaching fields, dry wells, pits, sumps and surface disposal areas. Potential sources of contamination also include past surface discharges, spills, and tank and pipe leaks. Figure 2.1 identifies twenty-five potential source areas determined during the Preliminary Investigation and subsequent Source Characterization Study conducted by Dames & Moore (1987a).

When the Motorola Inc., 52nd Street facility was first constructed, no municipal sewer was available, requiring on-site disposal of domestic and industrial waste in a septic tank and leaching field. These disposal practices continued from 1956 until 1963, when utility service became available from the City of Phoenix. Waste solvents were collected in underground tanks or smaller containers, then packaged into 55-gallon drums and stored on-site for salvage or contract disposal. Dry wells were designed primarily to handle area storm water runoff; however, discharges of chemicals into these wells from incidental spills and waste disposal have been documented.

Many locations were identified as potential chemical sources in the draft RI (1987a). Volume II of the 1987 RI/FS offers an in-depth analysis of locations and types of potential sources identified at the Motorola Inc., 52nd Street facility. Three primary areas have been identified with the greatest potential for contaminating soil and groundwater (Figure 1.1): the courtyard, Acid Treatment Plant (ATP), and Southwest Parking Lot (SWPL). The courtyard was the site of a leaking TCA storage tank which initiated the Preliminary Investigation. A dry well located in the courtyard has been reported to have received over 100,000 gal of waste solvents from 1963 to 1974. The ATP was built on a buried waste solvent line suspected of leaking approximately 4,000 gallons before repair. Solvent spills totaling an

SOURCE: Dames & Moore Draft RI, 1987

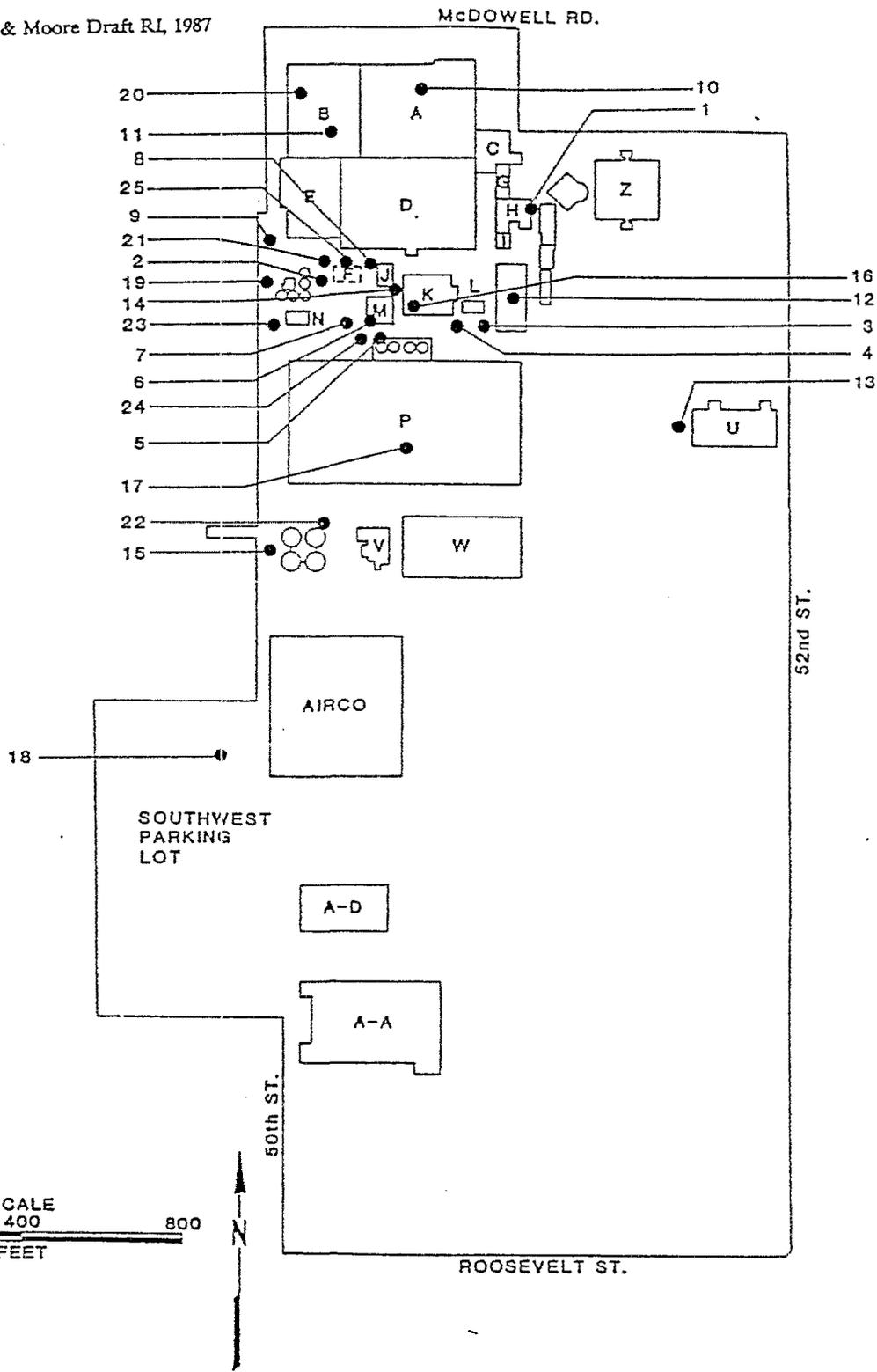


Figure 2.1 POTENTIAL SOURCE AREAS

estimated 5,000 gal occurred in the ATP area from 1964 to 1973. The SWPL was used extensively as a main staging area from 1974 through 1976, with 3,700 gallons of waste chemicals estimated to have leaked from stored 55-gallon barrels.

2.2 DATA COLLECTION AND EVALUATION

Groundwater, soil gas, and soil were sampled during the RI/FS. Samples were collected from 51 monitor wells placed in the alluvium and/or bedrock (Figure 2.2). Many were multi-port wells with samples collected at more than one depth per event. A total of 366 soil gas samples were collected at 324 locations. The majority of the soil gas data were collected in 1984-1985, smaller samplings of only on-site locations were done in 1990 and 1991. A soil gas survey of 41 sites in the residential neighborhoods surrounding the Motorola facility was performed in March 1992. A total of 87 soil samples were collected, consisting of 46 samples for VOCs, and 41 samples for inorganics. Analyses were performed by Analytical Technologies Inc., Hydro Geo Chem, Inc., Tracer Research Corp., and Western Technologies Inc.

Groundwater sampling data for the years 1988 to 1991 were used in this risk assessment. The data were submitted to ADHS by Dames & Moore in database format on 3.5" diskettes. Data for 1988 to Jan 1991 were submitted in May, 1991; data collected from January, 1991 to August, 1991 were received in December, 1991. Data included in the first submittal were contained in the Water Quality Data Usability Report (Dames & Moore, 1991b). Soil gas and soil data were received in hard copy format and entered into electronic format by ADHS staff.

Mean and the 95% upper confidence limit of the mean (UCL) concentrations of individual VOCs and inorganic chemicals were calculated for the entire area and for each well for determination of COPC. The UCL associated with each mean value was used to provide a measure of data reliability. The UCL was selected according to EPA methodology (USEPA 1989a) and calculated as described by Sanders et al. (1985). Means were calculated using reported concentrations or one-half the Sample Quantification Limit (SQL) for each sample. The SQL is the lowest estimate of the concentration of a chemical that is possible in a particular sample. It may vary greatly between samples due to interferences from other chemicals that are present in high concentrations. The concentration of the chemical in the sample may be considerably lower than the reported SQL, or may not be present. The use of one-half the SQL for calculation of the means conforms to current USEPA guidance (USEPA, 1989a). If there were no detects (a detect is a reading greater than the method detection limit or the SQL) for a chemical in a

particular well over the period from 1988 to 1991, then one-half the SQL or the UCL of one-half the SQLs was used for calculation of risks and hazard quotients.

Most groundwater samples were taken from the alluvium, but some monitor wells have sampling ports at the bedrock interface. Some of the chemicals are heavier than water and sink through the aquifer and concentrate in the bedrock. These are referred to as dense nonaqueous phase liquids (DNAPLs). Therefore samples from alluvial and bedrock locations were separated for calculation of mean and UCL concentrations, and potential risk.

Specific sample collection techniques, laboratory analysis, and quality control procedures are discussed in detail in the 1987 and 1991 RI/FS. Sampling was performed in accordance with the Task Specification for Sampling and Analysis of Ground Water (Dames & Moore, 1987c) and the Draft Sample Collection and Analysis Plan (Dames & Moore, 1990b).

2.2.1 Groundwater

Comprehensive organic priority pollutant and inorganic chemical analyses were performed on groundwater samples collected from alluvium and/or bedrock levels at 54 monitor wells. Groundwater samples were collected from soil borings where groundwater was encountered and analyzed for VOCs by EPA Method 624. Water samples were analyzed for inorganic content by the appropriate EPA method (Dames & Moore, 1987c).

The data were examined for uniformity of chemical designations and names were standardized. Water quality analyses that were not of interest in this risk assessment were removed; these included alkalinity, bicarbonate, carbonate, coliforms, conductivity, hardness, hydroxide, Ph, biological oxygen demand (BOD), total nitrogen, surfactants, and total organic carbon. Only data collected after evacuation of two well casing volumes were used for this assessment in order to maintain consistency of sampling. Samples designated as having fewer or a greater number of evacuations were eliminated from the data set (Dames and Moore, 1991b). Appendix Table 1 shows a summary of the data by well.

Tables 2.1 and 2.2 show the organic and inorganic species for which analyses were available. The mean, standard deviation, 95% upper confidence limit, and frequency of detection for all samples were then developed for each chemical detected (Table 2.3). Chemicals were removed from the risk assessment if there were no positive detections in the data set; or the highest detected value was less than the MCL or HBGL and the chemical is not recognized by IRIS as a possible (C), potential (B2), or human (A) carcinogen; or the chemical is not recognized as a potential health threat. Species not meeting

Table 2.1. Detected inorganic compounds in groundwater and bedrock, Motorola 52nd Street.

Chemical Name	Abbrev.	CASRN	WoE	Det#
Inorganic				
1. Ammonia	NH3	7664-41-7	D	48
2. Arsenic	As	7440-38-2	A	163
3. Barium	Ba	7440-39-3	D	56
4. Boron	B	7440-42-8	D	74
5. Cadmium	Cd	7440-43-9	D	2
6. Calcium	Ca	7440-70-2	ND	181
7. Chloride	Cl		ND	181
8. Chromium (VI)	CrVI	7440-47-3	A	2
9. Chromium (Tot)	Cr		D	10
10. Copper	Cu	7440-50-8	D	11
11. Cyanide, free	Cn	57-12-5	D	6
12. Fluoride	F	7782-41-4	D	82
13. Iron	Fe	7439-89-6	ND	76
14. Lead	Pb	7439-92-1	B2	69
15. Magnesium	Mg	7439-95-4	ND	180
16. Manganese	Mn	7439-96-5	D	100
17. Mercury	Hg	7439-97-6	D	1
18. Nickel	Ni	7440-02-0	D	43
19. Nitrate	NO3	14797-55-8	D	81
20. Potassium	K	7440-09-7	ND	26
21. Selenium	Se	7782-49-2	D	24
22. Silica	SiO2	112945-52-5	ND	96
23. Silver	Ag	7440-22-4	D	1
24. Sodium	Na	7440-23-5	ND	182
25. Sulfate	SO4	14808-79-8	D	180
26. Thallium	Tl	7440-28-0	ND	12
27. Total Dissolved Solids	TDS		ND	181
28. Zinc	Zn	7440-66-6	ND	61

Table 2.2. Detected organic compounds in groundwater
and bedrock, Motorola 52nd Street.

Chemical Name	Abbrev.	CASRN	WoE	Det#
Organic				
1. Benzene	BNZ	71-43-2	A	1
2. Bromodichloromethane	BDCM	75-27-4	B2	32
3. Carbon Tetrachloride	CCL4	56-23-5	B2	2
4. Chlorobenzene	MCB	108-90-7	D	71
5. Chloroform	CLFM	67-66-3	B2	117
6. Chloromethane	CM	74-87-3	C	3
7. Dibromochloromethane	DBCM	124-48-1	C	16
8. 1,2-Dichlorobenzene	DCB2	95-50-1	D	16
9. 1,4 Dichlorobenzene	DCB4	106-46-7	C	1
10. 1,2 & 1,4 Dichlorobenzene	DCB2/4		ND	114
11. Dichlorodifluoromethane	DCDFM	75-71-8	D	3
12. 1,1-Dichloroethane	DCA	75-34-3	C	96
13. 1,2-Dichloroethane	DCA2	107-06-2	B2	17
14. 1,1-Dichloroethylene	DCE	75-35-4	C	187
15. 1,2-Dichloroethylene	DCE2	540-59-0	D	228
16. Dichloromethane	DCM	75-09-2	B2	12
17. trans-1,3-Dichloropropene	tDCP3	10061-02-6	B2	1
18. Ethyl Chloride	EC	75-00-3	ND	1
19. Tetrachloroethylene	PCE	127-18-4	B2	240
20. 1,1,1-Trichloroethane	TCA	71-55-6	D	159
21. 1,1,2-Trichloroethane	TCA2	79-00-5	C	1
22. Trichloroethylene	TCE	79-01-6	B2	437
23. Trichlorofluoromethane	TCFM	75-69-4	D	18
24. Trichlorotrifluoroethane	F113	76-13-1	D	53
25. Vinyl Chloride	VC	75-01-4	A	41

Table 2.3.-Data summary by chemical for Motorola 52nd street risk assessment.

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest	Detects	Det %	WoE	HDGL	MCL
Inorganic												
1. Ammonia (NH3)	7664-41-7	mg/L	4.3	6.5	9.5	0.03	49	48/ 74	64.9%	--	D	
2. Antimony (Sb)	7440-36-0	mg/L					0/126		0.0%	--	D	3E-03
3. Arsenic (As)	7440-38-2	mg/L	0.10	0.15	0.32	0.005	2.6	163/181	90.1%		A	5E-02 5E-02
4. Barium (Ba)	7440-39-3	mg/L	0.12	0.18	0.24	0.014	1.2	56/ 74	75.7%	--	D	2E+00 2E+00
5. Beryllium (Be)	7440-41-7	mg/L					0/126		0.0%	--	B2	8E-06 4E-03
6. Boron (B)	7440-42-8	mg/L	2.2	2.6	1.6	0.14	7.5	74/ 74	100.0%		D	6E-01
7. Cadmium (Cd)	7440-43-9	mg/L	0.003	0.003	0.002	0.005	0.024	2/181	1.1%		D	4E-03 5E-03
8. Calcium (Ca)	7440-70-2	mg/L	122	141	126	0.9	596	181/181	100.0%	--	ND	
9. Chloride (Cl)		mg/L	487	552	449	8.3	2800	181/181	100.0%	--	ND	
10. Chromium (VI) (CrVI)	7440-47-3	mg/L	0.02	0.03	0.02	0.07	0.15	2/ 57	3.5%		A	4E-02
11. Chromium (Tot) (Cr)		mg/L	0.01	0.01	0.02	0.01	0.24	10/181	5.5%		D	1E-01 1E-01
12. Copper (Cu)	7440-50-8	mg/L	0.01	0.01	0.01	0.011	0.1	11/181	6.1%	--	D	1E+00
13. Cyanide, free (Cn)	57-12-5	mg/L	0.01	0.02	0.03	0.01	0.21	6/ 59	10.2%		D	2E-01
14. Fluoride (F)	7782-41-4	mg/L	7.6	8.7	4.8	0.2	25	82/ 82	100.0%		D	4E-01 4E+00
15. Iron (Fe)	7439-89-6	mg/L	0.29	0.46	1.15	0.011	10.5	76/181	42.0%	--	ND	
16. Lead (Pb)	7439-92-1	mg/L	0.006	0.007	0.01	0.002	0.08	69/181	38.1%		B2	5E-03
17. Magnesium (Mg)	7439-95-4	mg/L	54	62	55	0.2	275	180/181	99.4%	--	ND	
18. Manganese (Mn)	7439-96-5	mg/L	0.68	0.9	1.5	0.01	8.13	100/181	55.2%		D	7E-01
19. Mercury (Hg)	7439-97-6	mg/L	0.0002	0.0003	0.0004	0.0003	0.0003	1/181	0.6%	--	D	2E-03 2E-03
20. Nickel (Ni)	7440-02-0	mg/L	0.03	0.04	0.04	0.02	0.22	43/126	34.1%		D	1E-01
21. Nitrate (NO3)	14797-55-8	mg/L	19	24	25	0.37	92	81/ 82	98.8%		D	1E+01 1E+01
22. Potassium (K)	7440-09-7	mg/L	10	13	6.3	2	23.3	26/ 26	100.0%	--	ND	
23. Selenium (Se)	7782-49-2	mg/L	0.004	0.004	0.003	0.006	0.024	24/181	13.3%	--	D	5E-02 5E-02
24. Silica (SiO2)	112945-52-5	mg/L	41	46	24	3	190	96/ 96	100.0%	--	ND	
25. Silver (Ag)	7440-22-4	mg/L	0.01	0.01	0.01	0.1	0.1	1/181	0.6%		D	5E-02 5E-02
26. Sodium (Na)	7440-23-5	mg/L	482	531	339	22.5	2920	182/182	100.0%	--	ND	
27. Sulfate (SO4)	14808-79-8	mg/L	528	597	474	9	3400	180/181	99.4%		D	4E+02
28. Thallium (Tl)	7440-28-0	mg/L	0.003	0.003	0.002	0.0009	0.014	12/126	9.5%		ND	5E-04
29. Total Dissolved Solids (TDS)		mg/L	2009	2219	1440	320	9230	181/181	100.0%	--	ND	
30. Zinc (Zn)	7440-66-6	mg/L	0.04	0.07	0.18	0.01	2	61/181	33.7%		ND	1E+00
Organic												
1. Benzene (BHZ)	71-43-2	µg/L	4.2	8.2	4.8	2.3	2.3	1/ 8	12.5%		A	1E+00 5E+00
2. Bromodichloromethane (BDCH)	75-27-4	µg/L	19	28	106	0.26	314	32/568	5.6%		B2	3E-01 1E+02
3. Bromoform [TTHM] ² (BRFM)	75-25-2	µg/L						0/568	0.0%	--	B2	4E+00 1E+02
4. Bromomethane (BMH)	74-83-9	µg/L						0/568	0.0%	--	D	1E+01

Table 4.3. Continued.

Chemical Name	CASRN	Units	K	95% UCL	Deviation	Lowest	Highest	Detects	Det %	WoE	HRGL	MCL
5. Carbon tetrachloride (CCL4)	56-23-5	µg/L	19	28	107	0.3	0.6	2/568	0.4%	B2	3E-01	5E+00
6. Chlorobenzene (Monochlorobenzene) (MCB)	108-90-7	µg/L	83	108	305	0.3	1300	71/568	12.5%	D	1E+02	1E+02
7. 2-Chloroethylvinyl Ether (CEVE)	110-75-8	µg/L						0/561	0.0% --	ND		
8. Chloroform [THM] (CLFM)	67-66-3	µg/L	22	32	124	0.2	1500	117/568	20.6%	B2	6E+00	1E+02
9. Chloromethane (CM)	74-87-3	µg/L	21	30	115	2.1	14	3/564	0.5%	C	3E+00	
10. Dibromochloromethane [THM] (DBCM)	124-48-1	µg/L	18	27	106	0.2	1.1	16/568	2.8%	C	1E+01	1E+02
11. 1,2-Dichlorobenzene (DCB2)	95-50-1	µg/L	73	146	475	0.88	5600	16/163	9.8%	D	6E+02	6E+02
12. 1,3-Dichlorobenzene (DCB3)	541-73-1	µg/L						0/568	0.0% --	D	6E+02	
13. 1,4 Dichlorobenzene (DCB4)	106-46-7	µg/L	19	32	82	36.9	36.9	1/163	0.6%	C	7E+01	8E+01
14. 1,2 & 1,4 Dichlorobenzene (DCB2/4)		µg/L	348	674	3330	0.2	65000	114/402	28.4%	ND ¹		
15. Dichlorodifluoromethane (DCDFM)	75-71-8	µg/L	59	88	356	0.3	0.8	3/561	0.5% --	D	1E+03	
16. 1,1-Dichloroethane (DCA)	75-34-3	µg/L	29	41	137	0.09	1300	96/568	16.9%	C	7E+01	
17. 1,2-Dichloroethane (DCA2)	107-06-2	µg/L	20	30	123	0.2	1500	17/568	3.0%	B2	4E-01	5E+00
18. 1,1-Dichloroethylene (DCE)	75-35-4	µg/L	430	650	2670	0.3	26600	187/568	32.9%	C	6E+00	7E+00
19. 1,2-Dichloroethylene (DCE2)	540-59-0	µg/L	264	335	870	0.2	7000	228/568	40.1%	D	1E+02	7E+01
20. Dichloromethane (DCM)	75-09-2	µg/L	591	1234	7810	2.7	170000	12/568	2.1%	B2	5E+00	
21. 1,2-Dichloropropane (DCP2)	78-87-5	µg/L						0/568	0.0% --	B2	5E-01	5E+00
22. cis-1,3-Dichloropropene (cDCP3)	10061-01-5	µg/L						0/568	0.0% --	B2		
23. trans-1,3-Dichloropropene (tDCP3)	10061-02-6	µg/L	24	35	124	17.9	17.9	1/568	0.2%	B2		
24. Ethyl Chloride (EC)	75-00-3	µg/L	29	41	146	0.4	0.4	1/568	0.2% --	ND		
25. Ethylbenzene (ETB)	100-41-4	µg/L						0/8	0.0% --	D	7E+02	7E+02
26. 1,1,2,2-Tetrachloroethane (TET)	79-34-5	µg/L						0/566	0.0% --	C	2E-01	
27. Tetrachloroethylene (PCE)	127-18-4	µg/L	144	251	1302	0.2	30000	240/568	42.3%	B2	7E-01	5E+00
28. Toluene (TOL)	108-88-3	µg/L						0/8	0.0% --	D	1E+03	1E+03
29. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L	2677	4570	23020	0.2	330000	159/568	28.0%	D	2E+02	2E+02
30. 1,1,2-Trichloroethane (TCA2)	79-00-5	µg/L	17	25	105	4	4	1/568	0.2%	C	3E+00	
31. Trichloroethylene (TCE)	79-01-6	µg/L	19994	36900	205574	0.2	4100000	437/568	76.9%	B2	3E+00	5E+00
32. Trichlorofluoromethane (TCFM)	75-69-4	µg/L	63	89	3199	0.2	20	18/568	3.2% --	D	2E+03	
33. Trichlorotrifluoroethane (F113)	76-13-1	µg/L	329	540	2538	0.3	52000	53/557	9.5% --	D	2E+05	
34. Vinyl Chloride (VC)	75-01-4	µg/L	161	249	1066	1.4	20000	41/567	7.2%	A	2E-02	2E+00
35. Xylenes (total) (XYL)	1330-20-7	µg/L						0/5	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 the MCL and the WoE is not "A", "B2", or "C".

1. Unfortunately, 1,2 and 1,4 dichlorobenzene (DCB2 and DCB4) were reported together for a majority of the samples. The WoE for DCB2 is "D", but is "C" for DCB4. To be health protective, the combined samples will be treated as DCB4.
2. Trihalomethanes.

Table 2.4. Compounds eliminated from risk assessment,
Motorola 52nd Street.

Chemical Name	Abbrev.	CASRN	WoE	Det#
Inorganic				
1. Ammonia	NH3	7664-41-7	D	48
2. Antimony	Sb	7440-36-0	D	0
3. Barium	Ba	7440-39-3	D	56
4. Beryllium	Be	7440-41-7	B2	0
5. Calcium	Ca	7440-70-2	ND	181
6. Chloride	Cl		ND	181
7. Copper	Cu	7440-50-8	D	11
8. Iron	Fe	7439-89-6	ND	76
9. Magnesium	Mg	7439-95-4	ND	180
10. Mercury	Hg	7439-97-6	D	1
11. Potassium	K	7440-09-7	ND	26
12. Selenium	Se	7782-49-2	D	24
13. Silica	SiO2	112945-52-5	ND	96
14. Sodium	Na	7440-23-5	ND	182
15. Total Dissolved Solids	TDS		ND	181
Organic				
16. Bromoform	BRFM	75-25-2	B2	0
17. Bromomethane	BMM	74-83-9	D	0
18. 2-Chloroethylvinyl Ether	CEVE	110-75-8	ND	0
19. 1,3-Dichlorobenzene	DCB3	541-73-1	D	0
20. Dichlorodifluoromethane	DCDFM	75-71-8	D	3
21. 1,2-Dichloropropane	DCP2	78-87-5	B2	0
22. cis-1,3-Dichloropropene	cDCP3	10061-01-5	B2	0
23. Ethyl Chloride	EC	75-00-3	ND	1
24. Ethylbenzene	ETB	100-41-4	D	0
25. 1,1,2,2-Tetrachloroethane	TET	79-34-5	C	0
26. Toluene	TOL	108-88-3	D	0
27. Trichlorofluoromethane	TCFM	75-69-4	D	18
28. Trichlorotrifluoroethane	F113	76-13-1	D	53
29. Xylenes (total)	XYL	1330-20-7	D	0

the selection criteria are shown in Table 2.4. The 36 analytes that meet the criteria for inclusion in the risk assessment as chemicals of potential concern are summarized in Table 2.5.

2.2.2 Soil

Details of soil sampling procedures are discussed in the Preliminary Report Chemical Leak Project (Gutierrez-Palmenberg, 1983). Location of sampling sites were based on evaluation of available water-quality data, examination of company records, recollections of waste disposal practices, and observations made during field reconnaissance of the facility. Soil sampling was very limited and confined to source areas on site. ADHS was not provided with any recent soil sampling data for use in this risk assessment. Inorganic analysis and sample preparations were accomplished using a variety of methods appropriate to the particular group of anions or cations. Other sample preparation and analytical procedures were used, depending on the inorganic specie (Dames & Moore, 1987a,b). A summary of chemical concentrations found in soil on-site is listed in Appendix Table 2.

2.2.3 Soil Gas

Soil gas samples were obtained from in situ soil and analyzed at the sampling location by mobile laboratory using gas chromatography. Soil gas surveys completed in 1984 and 1985 (Dames & Moore, 1987a) as part of a source investigation. It encompassed the Motorola 52nd Street Facility and extended to approximately 44th Street to the east, Washington Street to the south, and Palm lane to the north. These results, although dated, have been used for a quantitative risk assessment for the purpose of comparing the results to those for later samplings.

The 1984 and 1985 soil gas samples were collected using a hollow steel probe that was driven into the soil to depths of three to five feet and then evacuated with a vacuum pump. A constant vacuum was maintained by use of a vacuum gauge on the pump. A gas tight syringe was then inserted through the evacuation line into the steel probe. The samples were drawn from within the probe. Syringes were purged three times with soil gas prior to each sampling. The samples were analyzed in a field van using a gas chromatograph (HP 5895 or Varian 3300) with a packed column and an electron capture detector. Instruments were calibrated to NBS standards each morning. Equipment, instrument, and ambient air blanks were run periodically. Three injections were made for each sampling location. Samples were held up to 30 minutes before injection. A prior experiment had determined that results were not affected by holding times of 30 minutes. Sampling was done by Tracer Research Corporation, under the supervision of Dames and Moore (Dames and Moore, 1987a).

Table 4.5.-Summary of chemicals of potential concern.

Chemical Name	CASRN	Units	Mean	95% UCL	Lowest	Highest	Detects	MoE	IRGL	NCL
Inorganic										
1. Arsenic, (As)	7440-38-2	mg/L	0.10	0.15	0.005	2.6	163/181	A	5E-02	5E-02
2. Boron (B)	7440-42-8	mg/L	2.2	2.6	0.14	7.5	74/74	D	6E-01	6E-01
3. Cadmium (Cd)	7440-43-9	mg/L	0.003	0.003	0.005	0.024	2/181	D	4E-03	5E-03
4. Chromium (VI) (CrVI)	7440-47-3	mg/L	0.02	0.03	0.07	0.15	2/57	A	4E-02	4E-02
5. Chromium (Tot) (Cr)		mg/L	0.01	0.01	0.01	0.24	10/181	D	1E-01	1E-01
6. Cyanide, free (Cn)	57-12-5	mg/L	0.01	0.02	0.01	0.21	6/59	D	2E-01	2E-01
7. Fluoride (F)	7782-41-4	mg/L	8	9	0.2	25	82/82	D	4E-01	4E+00
8. Lead (Pb)	7439-92-1	mg/L	0.006	0.007	0.002	0.08	69/181	B2	5E-03	5E-03
9. Manganese (Mn)	7439-96-5	mg/L	0.68	0.9	0.01	8.13	100/181	D	7E-01	7E-01
10. Nickel (Ni)	7440-02-0	mg/L	0.03	0.04	0.02	0.22	43/126	D	1E-01	1E-01
11. Nitrate (NO3)	14797-55-8	mg/L	18	24	0.37	92	81/82	D	1E+01	1E+01
12. Silver (Ag)	7440-22-4	mg/L	0.01	0.01	0.1	0.1	1/181	D	5E-02	5E-02
13. Sulfate (SO4)	14808-79-8	mg/L	528	597	9	3400	180/181	D	4E+02	4E+02
14. Thallium (Tl)	7440-28-0	mg/L	0.003	0.0032	0.0009	0.014	12/126	ND	5E-04	5E-04
15. Zinc (Zn)	7440-66-6	mg/L	0.04	0.07	0.01	2	61/181	ND	1E+00	1E+00
Organic										
16. Benzene (BNZ)	71-43-2	µg/L	4	8	2.3	2.3	1/8	A	1E+00	5E+00
17. Bromodichloromethane (BDCM)	75-27-4	µg/L	19	28	0.26	314	32/568	B2	3E-01	1E+02
18. Carbon tetrachloride (CCL4)	56-23-5	µg/L	19	28	0.3	0.6	2/568	B2	3E-01	5E+00
19. Chlorobenzene (Monochlorobenzene) (MCB)	108-90-7	µg/L	83	108	0.3	1300	71/568	D	1E+02	1E+02
20. Chloroform [THM] (CLFM)	67-66-3	µg/L	22	32	0.2	1500	117/568	B2	6E+00	1E+02
21. Chloromethane (CM)	74-87-3	µg/L	21	30	2.1	14	3/564	C	3E+00	3E+00
22. Dibromochloromethane [THM] (DBCM)	124-48-1	µg/L	18	27	0.2	1.1	16/568	C	1E+01	1E+02
23. 1,2-Dichlorobenzene (DCB2)	95-50-1	µg/L	73	146	0.88	5600	16/163	D	6E+02	6E+02
24. 1,1-Dichloroethane (DCA)	75-34-3	µg/L	29	41	0.09	1300	96/568	C	7E+01	7E+01
25. 1,2-Dichloroethane (DCA2)	107-06-2	µg/L	20	30	0.2	1500	17/568	B2	4E-01	5E+00
26. 1,4-Dichlorobenzene (DCB4)	106-46-7	µg/L	19	32	36.9	36.9	1/163	C	7E+01	8E+01
27. 1,2 & 1,4-Dichlorobenzene (DCB2/4)		µg/L	348	674	0.2	65000	114/402	ND		
28. 1,1-Dichloroethylene (DCE)	75-35-4	µg/L	430	650	0.3	26600	187/568	C	6E+00	7E+00
29. 1,2-Dichloroethylene (DCE2)	540-59-0	µg/L	264	335	0.2	7000	228/568	D	1E+02	7E+01
30. Dichloromethane (DCM)	75-09-2	µg/L	591	1234	2.7	170000	12/568	B2	5E+00	5E+00
31. trans-1,3-Dichloropropene (tDCP3)	10061-02-6	µg/L	24	35	17.9	17.9	1/568	B2		
32. Tetrachloroethylene (PCE)	127-18-4	µg/L	144	251	0.2	30000	240/568	B2	7E-01	5E+00
33. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L	2677	4570	0.2	330000	159/568	D	2E+02	2E+02
34. 1,1,2-Trichloroethane (TCA2)	79-00-5	µg/L	17	25	4	4	1/568	C	3E+00	3E+00
35. Trichloroethylene (TCE)	79-01-6	µg/L	19994	36900	0.2	4100000	437/568	B2	3E+00	5E+00
36. Vinyl Chloride (VC)	75-01-4	µg/L	161	249	1.4	20000	41/567	A	2E-02	2E+00

Soil gas investigations were conducted on the Motorola site in 1989 and 1990 (Dames & Moore, 1990c, 1991a, 1992). In January, 1989 soil gas samples were collected at 19 locations in the Courtyard based upon areas of highest concentrations observed during the 1984-85 sampling. Samples were collected from a depth of four feet (One sample was collected at a depth of 3 feet.) through probes driven through asphalt. Vacuum gauge readings during sampling were 4 to 5 inches of mercury. Concentrations were generally lower than in 1985 with the greatest differences in the central and southern portion of the Courtyard.

Two soil gas sampling rounds were completed in March and October, 1991 to identify the source or sources of ground water contamination observed in the area of the Southwest Parking Lot (SWPL). The March sampling was performed by Hydro Geo Chem, Inc. and the October sampling by Tracer Research. All of the March sampling was done on site while the October sampling included locations to the south and southwest of the SWPL.

Hydro Geo Chem used carbon sampling cartridges coupled with a flow controller down stream of the collector. A mobile field laboratory was used for analysis of samples. Thermal desorption was then used for analysis using a Varian 3400 GC equipped with Hall and PID detectors. Nickel plated pipe was used for sampling. Three (3) volumes were purged before sample collection. Instruments were calibrated at start of analyses and after every 10 samples. Equipment, instrument, and ambient air blanks were also run.

Tracer research used methods explained above for the October sampling.

New rounds of soil gas sampling were completed in March and July, 1992 by Malcolm Pirnie Inc. under the direction of ADEQ. Sampling and analyses were performed by Transwest Geochem. The sampling area extended from the western border of the Motorola facility to approximately 44th Street, but was concentrated in the residential neighborhoods immediately to the west and southwest of the facility (Malcolm Pirnie, 1992).

Forty-one (41) soil gas samples were collected. A six foot steel gas probe with adjustable inlet tip was driven three feet into the soil for in situ sample collection. Five probe volumes of air were evacuated before sampling. Samples were collected in a 10 mL air-tight syringe. Samples were usually analyzed within an hour of collection. Analyses were performed by Transwest Geochem using a gas chromatograph equipped with PID and Hall detectors. Quality assurance procedures included daily calibration, periodic analysis of standards, analysis of a variety of blanks, and duplicate analyses of at least one out of ten samples.

The existing soil gas data for all sampling periods are reported in Appendix Tables 3 through 7.

2.2.4 Private Wells

Well 4626G

The private well designated, 4626G, is located northwest of the Motorola facility. It is a private water supply well registered for domestic use and has been primarily used for filling a residential swimming pool and for grounds irrigation. The well was also used for indoor domestic purposes for a period of about six months in the late 1980's. This well is located near the northern boundary of the groundwater plume emanating from the Motorola 52nd Street facility. A summary of chemicals is reported in Table 2.6. This table reports data from 1987 to 1992. During this period boron, fluoride, and lead were determined to exceed either HBGLs or MCLs. Four (4) organic compounds, chloroform, toluene, TCE, and trichlorofluoromethane were found in the samples, but none of these exceeded either the HBGL or MCL.

Six (6) samples were collected over the five (5) year period. This level of monitoring may have been justified due to the low number and concentrations of chemicals detected. It would be prudent in the future to increase the sampling to biannually, or quarterly, due to continued use of the well and its proximity to the northern reach of the groundwater plume. Risks posed by use of this private well are addressed in Chapter 3.0.

Turnage Well

The Turnage well is located at 1502 N. 46th Street. It was drilled in 1948. The well is cased in eight inch steel from the surface to 117 feet and uncased from 177 to 132 feet. The well was used as the domestic water source for about 20 years, from the 1948 to 1969 or 1970. The well was sampled for VOCs during the period from 1984 to March, 1986. The results of five sampling rounds during this period were delivered to ADHS by ADEQ. Ranges in reported concentrations were: TCE 1,600-12,000 $\mu\text{g/L}$; PCE 14.2-60 $\mu\text{g/L}$; 1,2-DCB <2-45 $\mu\text{g/L}$; 1,2-t-DCE 3.1-98.7 $\mu\text{g/L}$; and methylene chloride <0.5-6350 $\mu\text{g/L}$. Sampling was discontinued in 1986 due to the proximity of a monitor well (DM 106). At that time a locked steel housing was installed to protect the well and prevent its use. Access to the well has been controlled by Motorola, Inc. since the installation of the housing and lock.

The time at which the well became contaminated is not known and can not be established. It is not possible to estimate past risk from domestic use of the well water for a twenty year period, ending about 1970. This well will not be included in the quantitative risk assessment due to the lack of current data and the fact that it is not currently in use and is under lock and key.

Table 2.6. Data summary by chemical for well 4626G.

Chemical Name	CASRN	Units	Me	95% UCL	Deviation	Lowest	Highest	Detects	Det %	Wc	BGL	HCL
Inorganic												
1. Ammonia (NH3)	7664-41-7	mg/L	0.84	**		0.84	0.84	1/1	100.0% --	D		
2. Antimony (Sb) ¹	7440-36-0	mg/L						0/2	0.0% --	D	3E-03	
3. Arsenic (As)	7440-38-2	mg/L	0.008	**	0.001	0.006	0.008	3/4	75.0%	A	5E-02	5E-02
4. Barium (Ba)	7440-39-3	mg/L	0.062	**	0.012	0.074	0.074	1/2	50.0% --	D	2E+00	2E+00
5. Beryllium (Be)	7440-41-7	mg/L						0/2	0.0% --	B2	8E-06	4E-03
6. Boron (B)	7440-42-8	mg/L	1.1	**		1.1	1.1	1/1	100.0%	D	6E-01	
7. Cadmium (Cd)	7440-43-9	mg/L						0/4	0.0% --	D	4E-03	5E-03
8. Calcium (Ca)	7440-70-2	mg/L	118	**	14	100	134	3/3	100.0% --	ND		
9. Chloride (Cl)		mg/L	279	**	22	255	310	4/4	100.0% --	ND		
10. Chromium (VI) (CrVI)	7440-47-3	mg/L						0/1	0.0% --	A	4E-02	
11. Chromium (TOT) (Cr)		mg/L						0/4	0.0% --	D	1E-01	1E-01
12. Copper (Cu)	7440-50-8	mg/L	0.015	**	0.006	0.013	0.013	1/4	25.0% --	D	1E+00	
13. Fluoride (F)	7782-41-4	mg/L	2.27	**	2.07	0.2	4.33	2/2	100.0%	D	4E-01	4E+00
14. Iron (Fe)	7439-89-6	mg/L	0.088	**	0.07	0.043	0.209	3/4	75.0% --	ND		
15. Lead (Pb)	7439-92-1	mg/L	0.019	**	0.027	0.065	0.065	1/4	25.0%	B2	5E-03	
16. Magnesium (Mg)	7439-95-4	mg/L	30	**	1.5	28.5	32	3/3	100.0% --	ND		
17. Manganese (Mn)	7439-96-5	mg/L	0.01	**	0.01	0.01	0.015	2/4	50.0% --	D	7E-01	
18. Mercury (Hg)	7439-97-6	mg/L						0/4	0.0% --	D	2E-03	2E-03
19. Nickel (Ni)	7440-02-0	mg/L						0/2	0.0% --	D	1E-01	
20. Nitrate (NO3)	14797-55-8	mg/L	2.03	**	0.38	1.65	2.4	2/2	100.0% --	D	1E+01	1E+01
21. Selenium (Se)	7782-49-2	mg/L						0/4	0.0% --	D	5E-02	5E-02
22. Silica (SiO2)	112945-52-5	mg/L	27	**	0.5	26.5	27.4	2/2	100.0% --	ND		
23. Silver (Ag)	7440-22-4	mg/L						0/4	0.0% --	D	5E-02	5E-02
24. Sodium (Na)	7440-23-5	mg/L	202	**	16	181	220	3/3	100.0% --	ND		
25. Sulfate (SO4)	14808-79-8	mg/L	168	**	33	130	200	4/4	100.0% --	D	4E+02	

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Table 2.6. (Continued) Data summary by chemical for well 4626G.

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest	Detects	Det %	MoE	MBGL	MCL
Inorganic												
26. Thallium (Tl)	7440-28-0	mg/L						0/ 2	0.0% --	ND	5E-04	
27. Total dissolved solids (TDS)		mg/L	1030	**	72	930	1130	4/ 4	100.0% --	ND		
28. Zinc and Compounds (Zn)	7440-66-6	mg/L	0.01	**	0.01	0.019	0.019	1/ 4	25.0% --	ND	1E+00	
Organic												
1. Benzene (BNZ)	71-43-2	µg/L						0/ 2	0.0% --	A	1E+00	5E+00
2. Bromobenzene	108-86-1	µg/L						0/ 1	0.0% --	ND		
3. Bromodichloromethane (BDCM)	75-27-4	µg/L						0/ 6	0.0% --	B2	3E-01	1E+02
4. Bromochloromethane	74-97-5	µg/L						0/ 1	0.0% --	D		
5. Bromoform [THM] (BRFH)	75-25-2	µg/L						0/ 6	0.0% --	B2	4E+00	1E+02
6. Bromomethane (BHM)	74-83-9	µg/L						0/ 6	0.0% --	D	1E+01	
7. Carbon tetrachloride (CCL4)	56-23-5	µg/L						0/ 6	0.0% --	B2	3E-01	5E+00
8. Chlorobenzene (MCB)	108-90-7	µg/L						0/ 6	0.0% --	D	1E+02	1E+02
9. 2-Chloroethylvinyl ether (CEVE)	110-75-8	µg/L						0/ 5	0.0% --	ND		
10. Chloroform [THM] (CLFM)	67-66-3	µg/L	0.2	**	0.2	0.5	0.5	1/ 6	16.7%	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/ 1	0.0% --	D	1E+02	
13. p-Chlorotoluene	106-43-4	µg/L						0/ 1	0.0% --	ND		
14. Dibromochloromethane [THM] (DBCM)	124-48-1	µg/L						0/ 6	0.0% --	C	1E+01	1E+02
15. Dibromomethane	74-95-3	µg/L						0/ 1	0.0% --	ND		
16. 1,2-Dichlorobenzene (DCB2)	95-50-1	µg/L						0/ 3	0.0% --	D	6E+02	6E+02
17. 1,3-Dichlorobenzene (DCB3)	541-73-1	µg/L						0/ 6	0.0% --	D	6E+02	
18. 1,4-Dichlorobenzene (DCB4)	106-46-7	µg/L						0/ 3	0.0% --	C	7E+01	8E+01
19. 1,2 & 1,4 Dichlorobenzene (DCB2/4)		µg/L						0/ 3	0.0% --	ND		
20. Dichlorodifluoromethane (DCDFM)	75-71-8	µg/L						0/ 6	0.0% --	D	1E+03	
21. 1,1-Dichloroethane (DCA)	75-34-3	µg/L						0/ 6	0.0% --	C		
22. 1,2-Dichloroethane (DCA2)	107-06-2	µg/L						0/ 6	0.0% --	B2	4E-01	5E+00

Table 2.6. (Continued) Data summary by chemical for well 4626G.

Chemical Name	CASRN	Units	Mean	95% UCL	Deviation	Lowest	Highest	Detects	Det %	WoE	HDGL	MCL
<i>Organic</i>												
23. 1,1-Dichloroethylene (DCE)	75-35-4	µg/L						0/ 6	0.0% --	C	6E+00	7E+00
24. 1,2-Dichloroethylene (DCE2)	540-59-0	µg/L						0/ 7	0.0% --	D	1E+02	7E+01
25. 1,2-Dichloropropane (DCP2)	78-87-5	µg/L						0/ 6	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/ 6	0.0% --	B2		
30. trans-1,3-Dichloropropene (tDCP3)	10061-02-6	µg/L						0/ 5	0.0% --	B2		
31. Ethyl chloride (EC)	75-00-3	µg/L						0/ 6	0.0% --	ND		
32. Ethylbenzene (ETB)	100-41-4	µg/L						0/ 2	0.0% --	D	7E+02	7E+02
33. Dichloromethane	75-09-2	µg/L						0/ 6	0.0% --	B2	5E+00	
34. Styrene	100-42-5	µg/L						0/ 1	0.0% --	C	1E+02	1E+02
35. 1,1,1,2-Tetrachloroethane	630-20-6	µg/L						0/ 1	0.0% --	C	2E+01	
36. 1,1,2,2-Tetrachloroethane (TET)	79-34-5	µg/L						0/ 6	0.0% --	C	2E-01	
37. Tetrachloroethylene (PCE)	127-18-4	µg/L						0/ 6	0.0% --	B2	7E-01	5E+00
38. Toluene (TOL)	108-88-3	µg/L	0.6	**	0.4	1	1	1/ 2	50.0% --	D	1E+03	1E+03
39. 1,1,1-Trichloroethane (TCA)	71-55-6	µg/L						0/ 6	0.0% --	D	2E+02	2E+02
40. 1,1,2-Trichloroethane (TCA2)	79-00-5	µg/L						0/ 6	0.0% --	C	3E+00	
41. Trichloroethylene (TCE)	79-01-6	µg/L	0.3	**	0.2	0.5	0.7	2/ 6	33.3%	B2	3E+00	5E+00
42. Trichlorofluoromethane (TCFM)	75-69-4	µg/L	0.3	**	0.1	0.4	0.4	1/ 6	16.7%	D	2E+03	
43. 1,2,3-Trichloropropane	96-18-4	µg/L						0/ 1	0.0% --	D	4E+01	
44. Trichlorotrifluoroethane (F113)	76-13-1	µg/L						0/ 4	0.0% --	D	2E+05	
45. Vinyl Chloride (VC)	75-01-4	µg/L						0/ 6	0.0% --	A	2E-02	2E+00
46. Xylenes (totals) (XYL)	1330-20-7	µg/L						0/ 1	0.0% --	D	1E+04	1E+04

1. Chemicals with no detects were left blank. Data samples are inclusive from 1987 through 1992.

** 95% UCL was not calculated due to small number of samples.

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

2.2.5 Salt River Project (SRP) Well

Well 18E-5N is a high-capacity irrigation well. Owned by the Salt River Project, it is located at the vicinity of 40th Street, Van Buren Street, and the Grand Canal. Well 18E-5N is specifically used to augment irrigation supplies during intermittent periods of surface water shortages. In response to high irrigation demands, groundwater is discharged into the SRP irrigation system which includes the Grand Canal and a system of distribution laterals (Figure 3.7). Table 2.7 lists the mean concentrations and ranges for the chemicals detected during four rounds of sampling performed by SRP from 1988 to 1991. With the exception of three samples of chloroform, all organic chemicals were at concentrations below the detection limit. The concentrations of chloroform were well below the health-based guidance level. Reported concentrations of nitrate, and sulfate are above drinking water MCL. Concentrations of boron and fluoride are above the more conservative Arizona HBGL for drinking water. Although it is likely that these are background levels, or in the case of nitrate, due to former agricultural activities in the area (Dames and Moore 1992), these constituents will be carried through to the next step in the risk assessment process, due to the difficulty in determining if the values represent background levels. The potential of SRP well 18E-5N as a exposure pathway is discussed later in this risk assessment.

2.3 SELECTION METHODOLOGY

2.3.1 Chemical Criteria

Extensive sampling of groundwater has been conducted at the Motorola 52nd Street facility and the surrounding area of investigation during the period from 1983 to the present. A complete soil gas survey was done in 1984 and 1985. A new, less extensive, soil gas sampling round was completed in March, 1992.

This health risk assessment assumes that all forms of contamination at or emanating from the site, that are within the authority of this risk assessment to address, have been detected and that chemicals of potential concern may be determined from chemicals reported in the Remedial Investigation (1987a, 1991a) and associated reports. Chemicals were selected if detected levels were considered greater than background levels; were considered a potential threat to human health; were detected in at least one monitor well; and the highest detected value was greater than the MCL or HBGL or the chemical is recognized by IRIS as a possible (C), potential (B2), or human (A) carcinogen. All chemicals determined to be present in soil gas were evaluated due to public concern over soil gas exposures.

Table 2.7 - Chemicals detected in SRP Well 18E-5N.

Chemical	Units	Mean	Range of Concentrations
Arsenic	mg/L	0.012	0.001 - 0.015
Benzene	μg/L	0.3 ^c	<0.5
Boron ^b	mg/L	1.9	1.8 - 2.3
Bromodichloromethane	μg/L	0.3 ^c	<0.5
Chlorobenzene	μg/L	0.3 ^c	<0.5
Chloroform	μg/L	0.7	<0.5 - 1.0
1,1-Dichloroethane	μg/L	0.3 ^c	<0.5
1,1-Dichloroethylene	μg/L	0.4 ^c	<1.0 - <0.5
1,2-Dichloroethylene	μg/L	0.3 ^c	<0.5
Fluoride ^b	mg/L	4.5	3.6 - 5.0
Lead	mg/L	0.003	<0.002 - 0.004
Nitrate ^a	mg/L	42.8	40.3 - 46.4
Sulfate ^a	mg/L	354	299 - 403
Tetrachloroethylene	μg/L	0.3 ^c	<0.5
Thallium	mg/L	NT ^d	NT
1,1,1-Trichloroethane	μg/L	0.3 ^c	<0.5
Trichloroethylene	μg/L	0.3 ^c	<0.5
Vinyl Chloride	μg/L	NT	NT

- a: Exceeds USEPA Maximum Contaminant Level for drinking water.
- b: Exceeds Arizona Health-Based Guidance Levels for drinking water.
- c: One-half of the detection limit used to calculate the mean.
- d: Not Tested for during sampling.

TABLE 2.8 - Chemicals of potential concern.

Organic	Inorganic
Benzene	Arsenic
Bromodichloromethane	Boron
Carbon tetrachloride	Cadmium
Chlorobenzene	Chromium
Chloroform	Cyanide
Chloromethane	Fluoride
Dibromochloromethane	Lead
1,2-Dichlorobenzene	Manganese
1,4-Dichlorobenzene	Nickle
1,1-Dichloroethane	Nitrate
1,2-Dichloroethane	Silver
1,1-Dichloroethylene	Sulfate
1,2-Dichloroethylene	Thallium
Dichloromethane	Zinc
trans-1,3-Dichloropropene	
Tetrachloroethylene	
1,1,1-Trichloroethane	
1,1,2-Trichloroethane	
Trichloroethylene	
Vinyl Chloride	

2.3.2 Quality Control

The purpose of this section is to introduce the concepts used to develop the list of potential chemicals of concern for groundwater. First, sample concentration data, recording codes, reporting periods, and monitor well identification, and compound names were checked for consistency. Procedures for identifying quality control problems focused on range checks (outliers were verified) and consistency checks (relationships between data elements were examined). All samples that deviated from the normal sampling procedure were eliminated from the data set. The chemicals were broadly divided into **organic** and **inorganic** categories. The mean, 95% upper confidence limit, and frequency of detection was then calculated for each chemical for which an analysis had been performed (Table 2.3).

Chemicals were then removed from the list if they met the following criteria: 1) not considered a potential threat to human health; 2) no positive detections in the data set; and 3) the highest detected value was less than the MCL or HBGL and the chemical is not recognized by IRIS as a possible (C), potential (B2), or human (A) carcinogen. The remaining species are considered to be chemicals of potential concern (Tables 2.5 and 2.8).

The data were then divided into alluvium and bedrock sampling locations. This divisional technique was expected to provide information about the vertical distribution of the contamination. The 29 chemicals satisfying the criteria for elimination from the quantitative risk assessment are identified in Table 2.4.

Private well 4626G was included in the general assessment of wells. A separate assessment was also performed. For the individual assessment, only data from the well were used to determine chemicals of concern (Table 2.6). This procedure was also followed for the SRP well (Table 2.7).

2.4 HEALTH RISK ASSESSMENT DATA SUMMARY

When the procedures described in Section 2.3 are applied to the list of detected chemicals, only those in Table 2.8 meet the requirements for inclusion as chemicals of potential concern. Information concerning carcinogenicity, toxicity, and other relevant physical and chemical properties are discussed in subsequent sections. Chapter 5.0 addresses the development of the final characterization of risk due to contamination originating from the Motorola Inc., 52nd Street facility.

2.5 DATA QUALITY ASSURANCE AND QUALITY CONTROL

The groundwater data were reviewed in the *Water Quality Usability Report* (Dames and Moore, 1991). The report reviews data collected during sampling rounds 8 through 14 (1987 to 1991). This included all but the most recent data used in the risk assessment. Evaluation criteria (acceptance limits for accuracy and precision) were taken from the analytical method or from EPA guidelines. If a deviation from an evaluation criterion occurred, the analytical result was qualified as "A", acceptable for use with qualification, or "R", unusable. Samples with an "A" designation are considered usable for risk assessment purposes and were included for risk calculations. Only two samples were designated with "R", and were dropped from the risk assessment.

Malcolm Pirnie, Inc. also performed data validation of analytical results submitted to ADEQ in February of 1992 by Motorola 52nd Street facility. There were some discrepancies between the two reports. Some samples designated as "estimated" by one set of reviewers that were not qualified by the other reviewers. The samples in question fall into the "A" category used by Dames and Moore. Samples specified as having "estimated" values are included in the risk assessment data set (USEPA, 1989a). Neither Malcolm Pirnie, Inc. or Dames and Moore found any major problems with the quality control.

An extensive soil gas sampling took place in 1984 and 1985 as part of a source location project. Samples were analyzed on-site by a mobile laboratory using gas chromatographs with packed columns

and electron capture detectors. No QA/QC on these samples was made available to ADHS, however all data points are means of three replicated analyses for each sample location. Equipment was calibrated to NBS standard each morning and appropriate blanks were run, including equipment, instrument and ambient air. It is the opinion of the ADHS that the soil gas data set is of sufficient quality and has less associated uncertainty than the risk characterizations it was used to estimate. The data were used to produce a quantitative assessment of risk in the area from soil gas. It should be recognized that the risk figures are of historical interest and are not intended to represent current risk.

Soil gas samples were collected on-site in 1989 and 1991 and off-site from the former high school property to the southwest of the facility. Levels in the Courtyard have tended to drop since the earlier samplings. Calculations using data from these samplings are considered to better represent the present situation.

Two off-site sampling rounds were completed in February and July of 1992. These results are used to calculate current residential risk for the neighborhoods surrounding the facility. Soil sampling also was not as extensive as in 1984 and 1985. This should not affect quantitation of current off-site risks associated with soil gas because areas of greatest concern, based upon concentrations of COPC in groundwater, were sampled. Uncertainties associated with the data collection should have a minimal impact on this risk assessment (see section 2.2.3).

The data from the three media sampled have confirmed the presence of chemicals at the site and in the groundwater.

To facilitate a more complete and reliable characterization of potential risk from groundwater use in the study area, chemical concentrations have been calculated on a well-specific basis. This approach was adapted due to the large area underlain by the groundwater plume and the large range of chemical levels within the plume. A much more detailed characterization of potential risk is possible by this method than by averaging data across wells.

Another source of uncertainty concerns the use of sample quantification limits (SQL). Some sample results have reported extremely high SQLs due to the presence of interferences. One half of the SQL was used in calculations of the means, if compounds had been previously identified as positively present in groundwater (see Section 2.3). This has the effect of increasing mean concentrations and is considered the most conservative treatment of the data. The ADHS considers this approach most protective of human health.

3.0 EXPOSURE ASSESSMENT

This exposure assessment focuses on present and potential human populations living or working on, or in the vicinity of the Motorola Inc. 52nd Street facility. It estimates the types and magnitudes of exposures to chemicals of potential concern and possible exposure pathways associated with contamination detected at the site. An exposure pathway is considered complete when a chemical of concern contacts a receptor (person). The four steps comprising an exposure assessment are: 1) identification of the exposure setting; 2) description of the exposed population and exposure pathways; 3) estimation of exposure concentrations of chemicals; and 4) calculation of intake doses for each pathway. This discussion is divided into the remaining six sections:

- * 3.1 - Exposure Setting Characterization
- * 3.2 - Exposure Pathway Identification
- * 3.3 - Data Modeling
- * 3.4 - Quantification of Exposures
- * 3.5 - Uncertainties in the Exposure Assessment
- * 3.6 - Exposure Assessment Summary

3.1 EXPOSURE SETTING CHARACTERIZATION

This section describes the physical setting of the site, including, location, meteorology, geology, soils, surface and ground water flow, current land use, and human populations in the vicinity of the site.

3.1.1 Physical Setting

Location

Section 1.5 details the location of Motorola Inc., 52nd Street facility.

Meteorology

Motorola Inc., 52nd Street Facility 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. Predominant wind directions are from the east, southeast, or west respectively. Figure 3.1 depicts the frequency of wind velocities and prevailing directions.

Geology

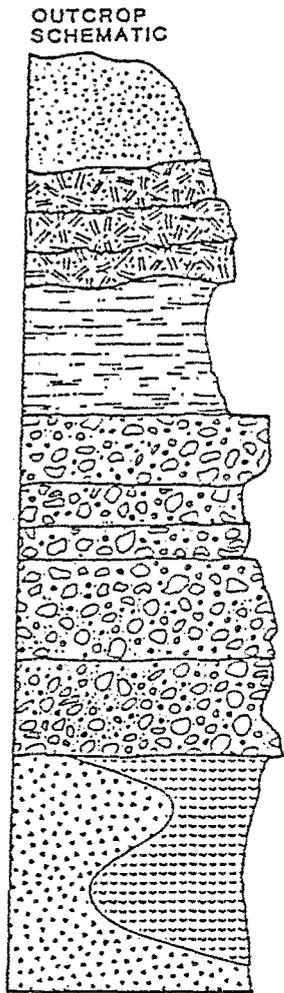
The geology of the study site is a two-layer system of alluvium overlying bedrock. Features of particular interest include a bedrock trough located to the west of the Motorola facility and trending to the northwest; there are well-defined bedrock highs to the west (Papago Buttes) and south; and overall downward slope to the west-southwest. Figure 3.2 illustrates the distribution of hydrostratigraphic units in the bedrock consist of Precambrian bedrock (metarhyolite and granite) and Tertiary bedrock (volcanics, Tempe beds, and Camels Head Formation).

The Salt River Valley is a series of coalesced alluvial basins. The Motorola 52nd Street facility is built on an alluvial fan, situated near the eastern margin of the West Basin of the Salt River Valley (Figure 3.3). This basin is a structural depression bounded by the Papago Buttes. This northwest-trending set of low hills lie about one mile east of the facility and form the structural and topographic divide between the East and West Basins. The geologic cross-section distribution of these units is represented in Figure 3.4.

Unconsolidated quaternary alluvium overlies bedrock throughout most of the area. Thickness of the alluvium generally increases to the west. The alluvium varies in thickness from less than 20 feet on the eastern boundary of the site to more than 60 feet at locations on the western boundary. At the Old Crosscut Canal, the alluvium is about 100 to 125 feet thick. The maximum thickness of alluvium encountered during this investigation is approximately 240 feet at well location DM-126. Physiography of the Basin is discussed more completely in Chapter 3.0 of the Draft RI (Dames & Moore, 1987a).

Soil

The Salt River Valley is commonly filled to depths of more than 1,000 feet by sand, gravel, silt and clay which 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 course-grained sand and gravel.

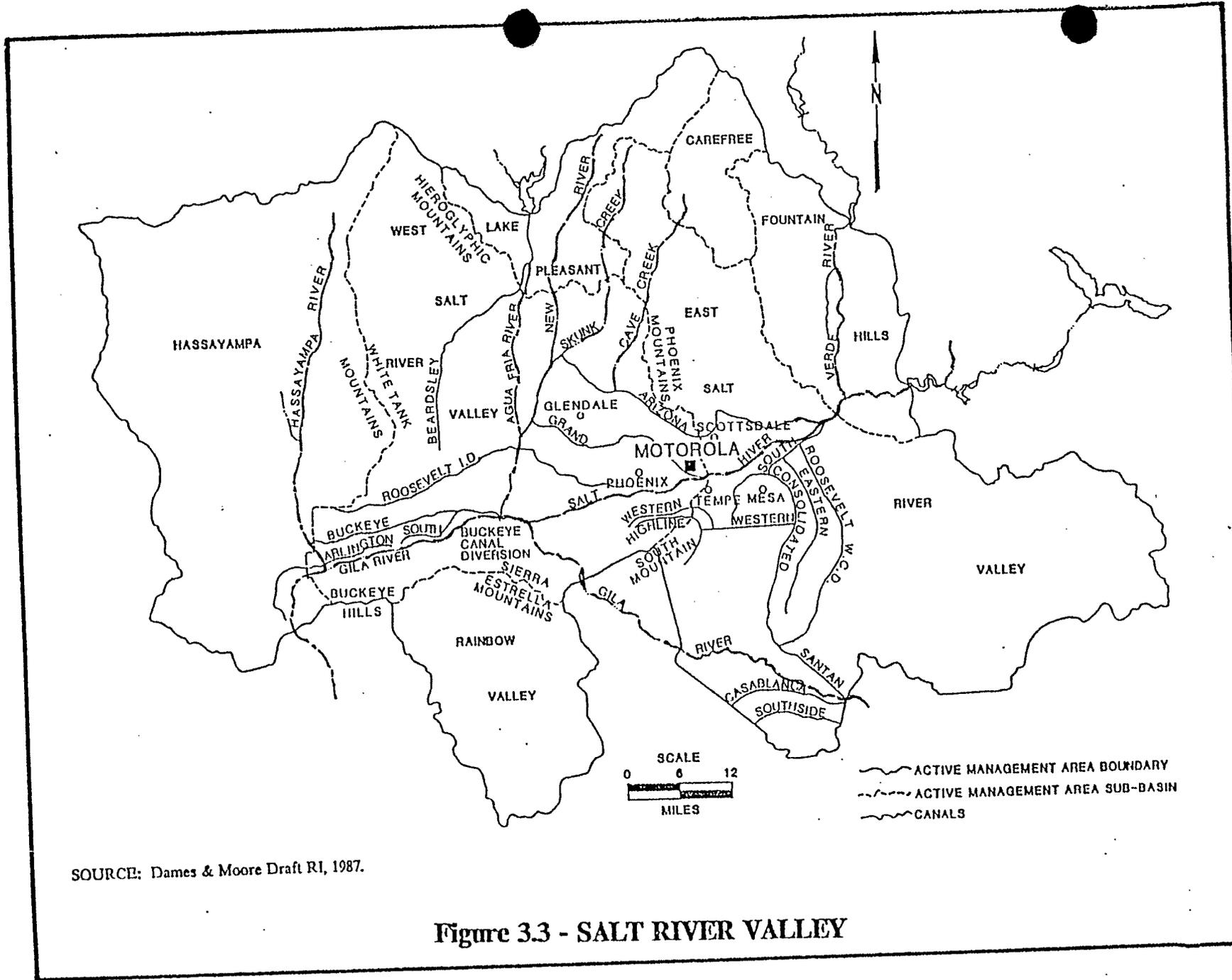


FORMATION OR UNIT	GEOLOGIC PERIOD	APPROXIMATE AGE (10 ⁶ YR)	APPROXIMATE THICKNESS (FT)
ALLUVIUM (Qal)	QUATERNARY	0 TO 3	20 TO 240
VOLCANICS (Tv)	TERTIARY	17 TO 20	0 TO 137
TEMPE BEDS (Ttb)			0 TO 96
CAMELS HEAD FORMATION (Tcf)			0 TO 190
META RHYOLITE (Pcmr)	PRECAMBRIAN	1,600 TO 1,800	0 TO 437
GRANITE (Pcg)			0 TO 208

SOURCE: Dames & Moore Draft RI, 1987

- NOTE:
1. THICKNESSES BASED ON THICKNESSES IN BORINGS IN THE STUDY AREA. GREATER THICKNESSES OF ALL BEDROCK UNITS OCCUR IN OUTCROP.
 2. OUTCROP SCHEMATIC IS A COMPOSITE FROM PAPAGO BUTTES, TEMPE BUTTE, TWIN BUTTE AND CAMELBACK MOUNTAIN.
 3. NOT TO SCALE

Figure 3.2 - STRATIGRAPHIC COLUMN



SOURCE: Dames & Moore Draft RI, 1987.

Figure 3.3 - SALT RIVER VALLEY

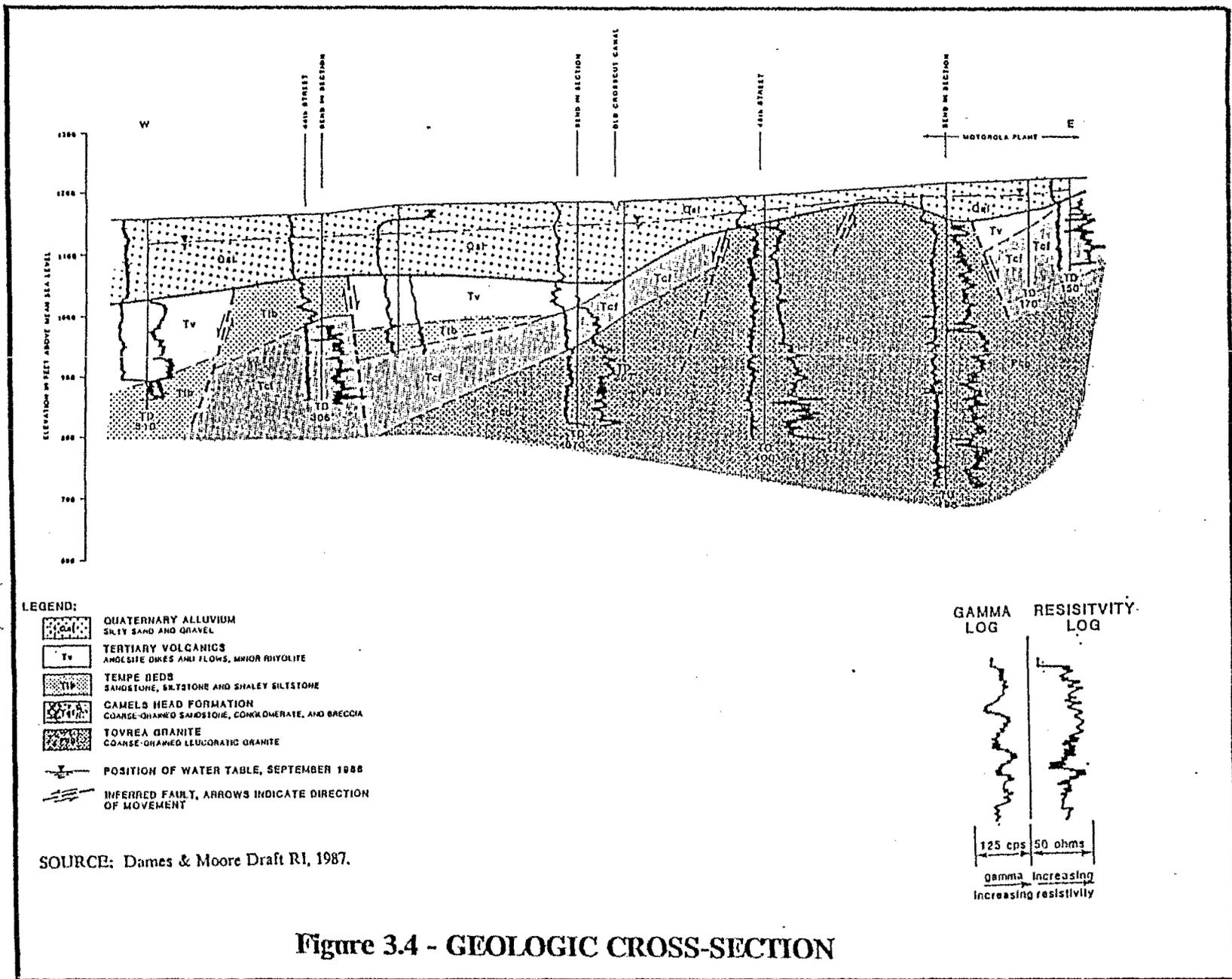


Figure 3.4 - GEOLOGIC CROSS-SECTION

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 (USDA, 1974). 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 caliche, but the cementation is variable and does not appear to form continuous layers.

Hydrology

Groundwater in the vicinity of the Motorola Inc., 52nd Street facility occurs in two distinct water bearing hydrogeologic formations, alluvium and bedrock, as part of a regional flow system. The water table, in most areas near the facility, is located in the alluvium. 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 saturated thickness of the alluvium varies from less than 10 feet to nearly 200 feet. The alluvium is thin near the eastern boundary of the site where the bedrock surface rises toward the Papago Buttes. The saturated thickness of the alluvium is also narrow along the top of the bedrock ridge which trends northwest from the southern part of the facility to a point near the intersection of 48th Street and McDowell Road. The permeability of the alluvium is about three orders of magnitude greater than the bedrock.

The bedrock underlying the study site is dominated by Tertiary sedimentary Camels Heads and Tempe formations. The water table intersects the bedrock surface near the small bedrock hill, south of the site, between Polk and Van Buren Streets. Groundwater flow in bedrock occurs primarily in open fractures, joints and bedding planes.

In the vicinity of the site, the predominant direction of regional groundwater flow is to the west/southwest. This flow is influenced by a bedrock ridge down-gradient from the facility courtyard. The bedrock ridge lies nearly perpendicular to the regional direction of groundwater flow, and impedes groundwater flow. As a result, the direction of flow in the alluvium out of the facility is initially directed west and northwest before the flow path turns southwest.

Surface water features near the site are the Salt River, two irrigation canals, and several small irrigation laterals. The Salt River channel flows about 1.5 miles south of the Motorola Inc., 52nd Street facility. 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 Old Crosscut Canal flows from the north to the

south approximately one-half mile west of the facility. It is used primarily to carry flood waters, although it has been used in the past to transfer irrigation water from the Arizona Canal to the Grand Canal. The Grand Canal flows from the southeast to the northwest about one mile southwest of the facility. 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

Relative Locations of Population With Respect to Site

Residential populations are concentrated to the north, west, and south of the site. The land to the east has no residential population in the area surrounding the site.

Current Land Use

The areas within a radius of one-half mile north, south and west of the Motorola Inc., 52nd Street facility are mixed residential single and multi-family neighborhoods. To the east are the Phoenix Military Reservation and Papago Park. Industrial use predominates the area between Washington Street and Phoenix Sky Harbor International Airport. The adjacent Van Buren Street and McDowell Road arterials are extensively used for commercial purposes. Major current land use within the study site (Figure 3.5) also includes two primary canals (Old Crosscut Canal and Grand Canal); a railway owned by Southern Pacific Railroad; and the Papago Freeway and Hohokam Parkway.

Future Land Use

Planned future land use in the area of the study site includes major redevelopment of much of the area for industrial and commercial purposes. Most of the rezoning is reflected in the immediate vicinity west/southwest of the facility to include the former East High School, the intersection of Van Buren and 44th Streets, and a large parcel of land between 36th and 40th Street (Figure 3.6). Another major planned change considers the use of the Old Crosscut Canal as part of a flood control system in conjunction with the U.S. Army Corps of Engineers' proposed Arizona Canal Diversion Channel.

Human Populations of Concern

At present there is no large population group exposed to groundwater contamination originating from the Motorola 52nd Street facility. No public supply wells have been or are located in the effected aquifer. Census Tract (CT) 1138 is identified as the most representative geographic subdivision of people

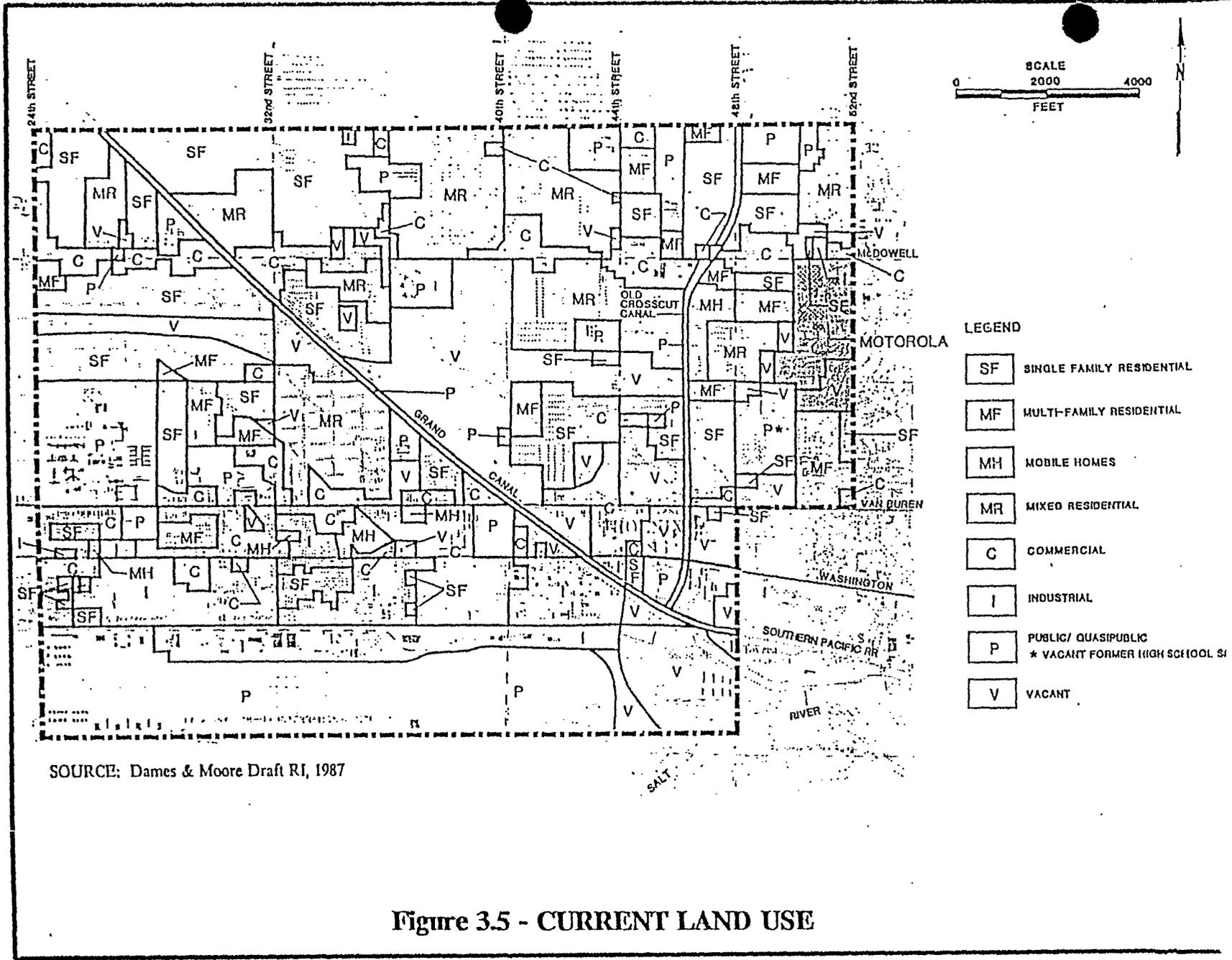


Figure 3.5 - CURRENT LAND USE

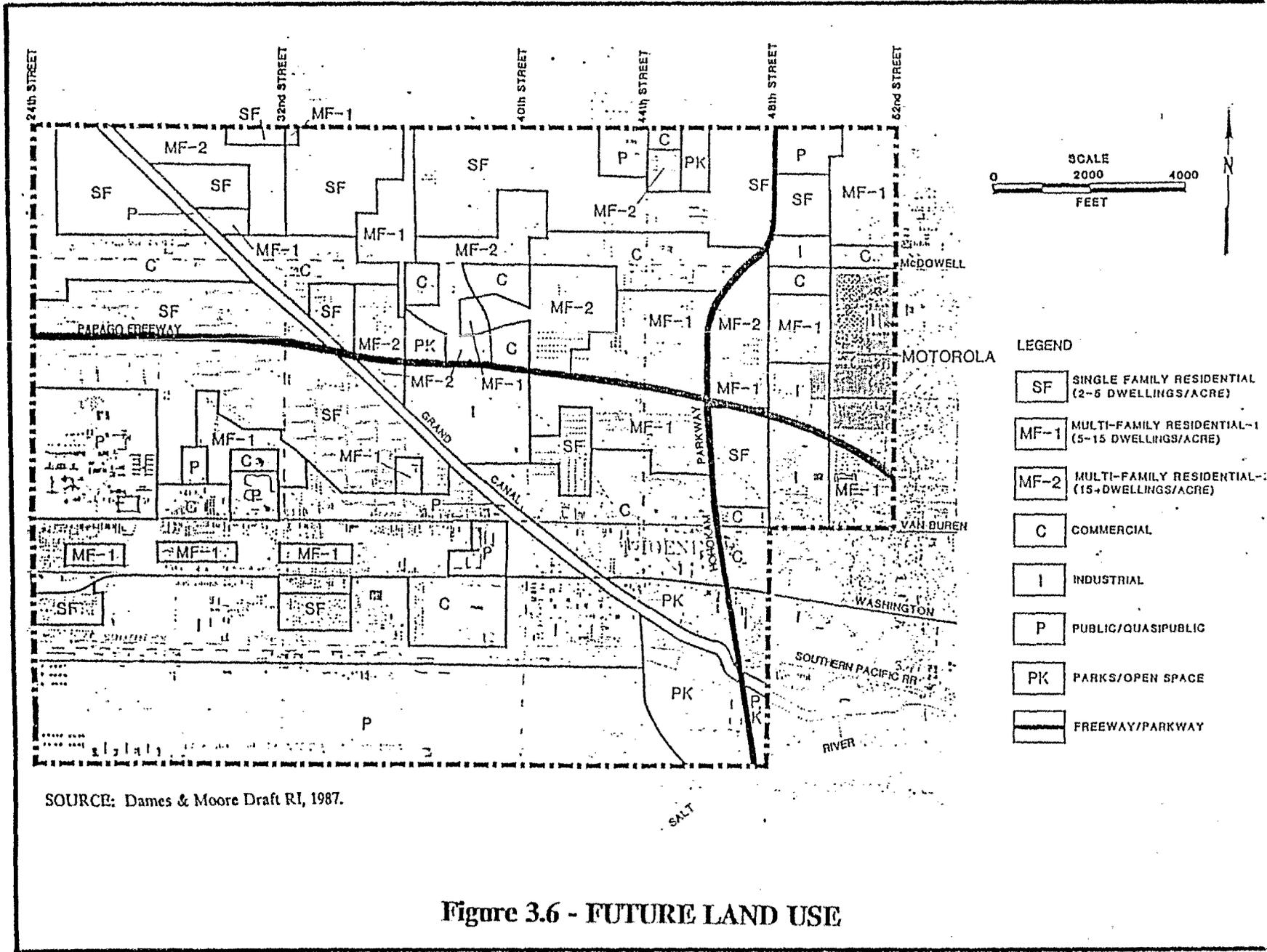


TABLE 3.1 -- Comparison of population changes.

Year	Phoenix	% Change	CT 1138	% Change
1970	581,562	—	3,736	—
1980	789,704	+ 35.8	2,497	-33.2
1990	983,403	+ 24.5	2,615	+ 4.7

SOURCE: 1990 Census of Population & Housing, P.L. 94-171 Data

potentially exposed to the effects of groundwater contamination from the Motorola Inc., 52nd Street Facility. The population immediately to the west of the plant may be exposed to soil gas releases due to contaminants in the groundwater. The census tract includes areas down gradient of the Motorola 52nd Street facility and overlays the contaminated groundwater plume. Population data for CT 1138 compared to City of Phoenix is summarized in Table 3.1. Twenty-five (25) percent of the population is under 18 years of age.

3.2 EXPOSURE PATHWAY IDENTIFICATION

A potentially complete human exposure pathway describes the route a chemical take from the source to a population or receptor. A complete exposure pathway includes the following components (USEPA, 1989a):

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

It is apparent that an exposure point may occur on-site or at a distance from the site, depending on transport mechanisms influencing the chemical(s). Exposure pathways for this risk assessment were identified based on a review of the Dames & Moore draft Remedial Investigation reports (1987a), draft Remedial Action Plan (1988), draft Final Remedy Remedial Investigation Report (1991a); communications from the USEPA, ADEQ, Salt River Project, Motorola Inc., concerned citizens, and an inspection of the Motorola 52nd Street facility and its environs.

3.2.1 Source and Receiving Media

The source of organic chemicals in the groundwater plume emanating from the Motorola Inc., 52nd Street facility is primarily attributed to waste handling procedures. An estimated total 200,000 gallons of chlorinated waste solvents were released at the site, since construction in 1957 (Dames and Moore, 1987a). During this time waste chemicals were disposed of or stored in drywells, leach fields, and underground storage tanks. In each instance the soil served as the receiving media, releasing the chemicals to groundwater. In the saturated zone, VOCs existed as undissolved free product in the soil pore spaces. These VOCs may slowly diffuse into the groundwater for an indefinite period of time. Concentrations in the unsaturated zone were discovered to increase with depth and are at a maximum near the water table.

Unlike most volatile organic compounds, inorganic constituents occur naturally in groundwater requiring the identification, evaluation, and estimation of inorganic chemicals in a relation to 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.

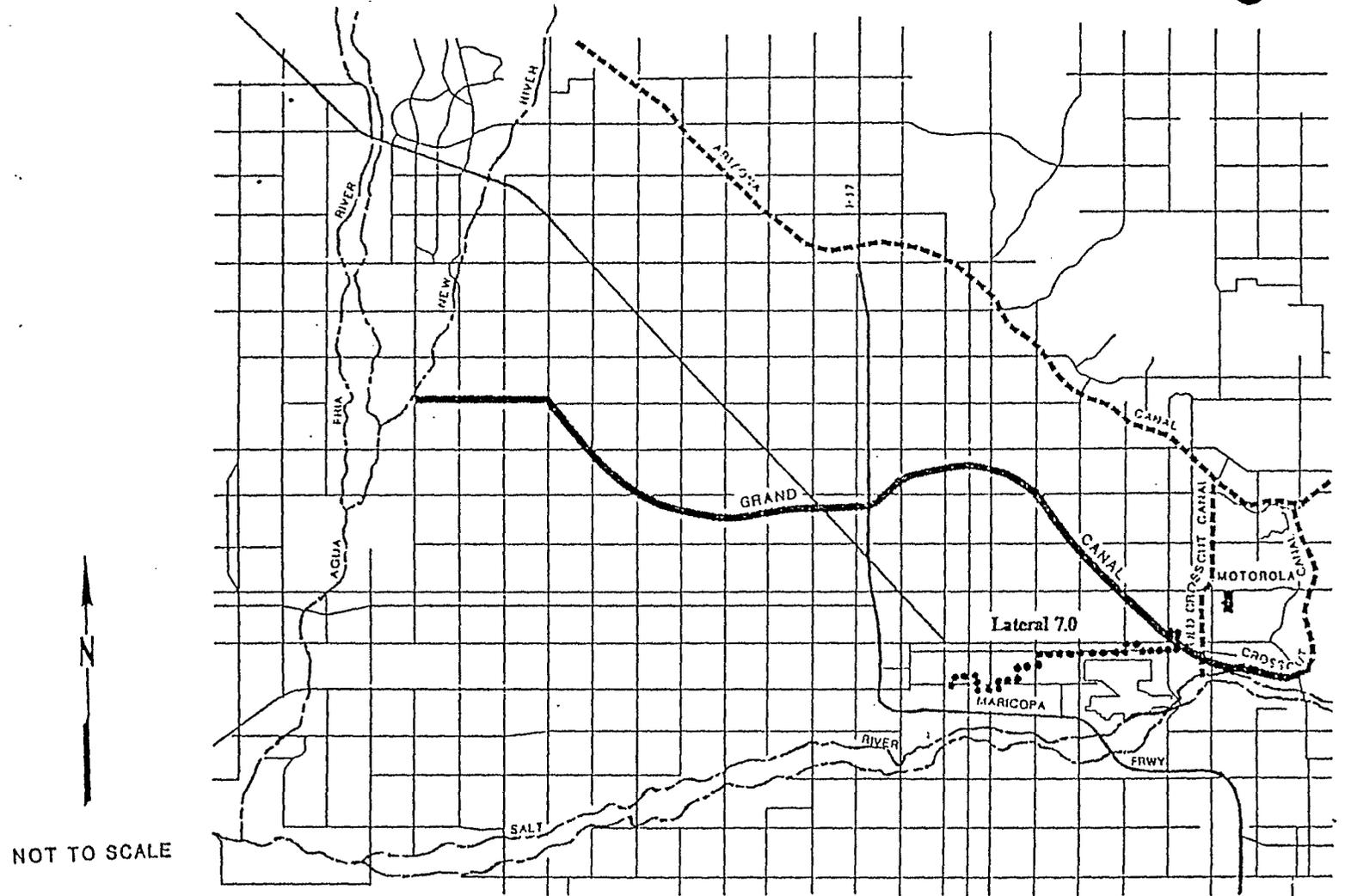
3.2.2 Fate and Transport in Release Media

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

Ground Water

Both organics, and, inorganics have been detected in a groundwater plume emanating from the site. Observed groundwater contaminants also include degradation products, particularly two isomers of 1,1-dichloroethylene, which were not used, stored, nor disposed at Motorola Inc., 52nd Street facility (Dames and Moore, 1987a).

Chemicals infiltrate and leach through the unsaturated and saturated zones of the soil to reach groundwater. VOC contamination has been detected in monitor wells located both on- and off-site. Organic and inorganic chemicals have been detected in one private well, 4626G, which the owner has reported to have been used for indoor domestic use, without benefit of dilution or treatment, for a period of about six months during 1989 to 1990. The well has also been used for filling a swimming pool and residential irrigation. SRP well, 18E-5N, located in the area of the groundwater plume, is used to augment water flows in the Grand Canal and Lateral 7.0 (Figure 3.7). The water is used for irrigation.



SOURCE: Dames & Moore Draft RI, 1987.

Figure 3.7 - CANAL DISTRIBUTION SYSTEM

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 in the vicinity of Motorola Inc., 52nd Street facility are intermittent and occur primarily as runoff from storms. The Old Crosscut Canal and the Grand Canal are the nearest permanent surface water features. Under current land use conditions for the Motorola site, artificial surface coverings (concrete, asphalt, etc.), buried receptors (insulated tanks, lines, etc.), and storm water runoff systems prevent most opportunities for contaminants in soil or groundwater to contact surface water. At present, only two complete exposure pathways are known to exist: SRP well 18E-5N and private well 4626G.

Air

All releases resulting in the listing of the site on the NPL were to soil. Some chemicals may be released into the air through volatilization from soil or, if the watertable is shallow enough, from the groundwater. Soil gas investigations have indicated that there is a potential for releases from these sources into the air on-site. At present, this is inhibited by the presence of paving or buildings covering much of the site. There is the potential for releases into buildings on-site through cracks in floors and foundations. Sampling of soil gas in the near-site area have indicated a rapid decline in VOC concentrations off-site. This trend was shown very clearly in the 1984 and 1985 soil gas data (Appendix Table 3).

The release of chemicals associated with fugitive dust generation occurs only when there is exposed soil. Under current land use most areas on-site are paved unless construction activities are in progress. There are no data indicating that off-site soils are contaminated due to the releases discussed in this report.

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 aliphatic (open chain), chlorinated hydrocarbons. The compounds which occur most frequently are chlorinated methanes, chlorinated ethanes, and chlorinated ethylenes. Compounds with the benzene ring structure have also been detected.

Transformations in subsurface areas are believed to be responsible for these detected VOCs: trans-1,2-dichloroethylene; 1,1-dichloroethylene; chloroform; 1,1-dichloroethane; and chlorobenzene. Vinyl chloride, which is an end-product of ethylene degradation under anaerobic conditions, has been observed in some monitor wells with corresponding lower ratios of TCE to total-DCE, a sign that degradation of TCE has taken place. These, and other chemical reactions, may explain why only approximately one-third of the many organic compounds detected are reported to have been used or released at the facility. Possible explanations for this discrepancy include:

- 1) Some of the chemicals used and released were mixtures of which not all constituents were accurately known.
- 2) Unknown compounds may have been present as impurities or contaminants in virgin solvents.
- 3) Records of use and disposal of some compounds may have been poorly kept or lost.
- 4) Subsurface transformations may have created compounds not originally present.
- 5) There may be unknown sources.

Most studies suggest that halogenated aliphatic organic compounds such as TCE, PCE, DCE, and TCA resist the degradation process under aerobic conditions. Under anaerobic (lack of oxygen) conditions reductive dehalogenation is known to occur and may account for the concentrations of DCE and vinyl chloride in the groundwater.

Table 3.2 – Exposure pathway summary.

Potential Exposed Population	Exposure Point	Exposure Route	Path Evaluated	Path Selected	Exposure Type	Rationale
GROUND WATER						
CURRENT LAND USE						
Residents	Private well (4262G), domestic use, swimming pool, and irrigation. No other wells known to be used.	Ingestion Inhalation Dermal	Yes Yes Yes	Yes Yes Yes	Actual	No restrictions on use of functioning well
Residential/ Commercial	SRP (18E-5N), irrigation	Ingestion Inhalation Dermal	Yes Yes Yes	Yes Yes Yes	Actual	Well used to supplement irrigation canal
Residents	Ground water, down gradient of site.	Ingestion Inhalation Dermal	Yes Yes Yes	Yes Yes Yes	Potential	No known exposures, no statutory restraints, risk management
SOIL GAS						
Workers	Soil vapor on-site, outdoors and indoors.	Inhalation	Yes	Yes	Actual	VOCs detected in shallow subsurface soil
Residents	Soil vapor, west of site, outdoors and indoors.	Inhalation	Yes	Yes	Actual	Possible volatilization of VOCs from watertable
SOIL						
Workers	Direct soil contact on-site.	Ingestion Inhalation Dermal	Yes Yes Yes	No No No	Intermittent	Insufficient data for quantitative assessment
Workers/ Residential	Fugitive dust	Ingestion Inhalation Dermal	Yes Yes Yes	No No No	Actual	Insufficient data for quantitative assessment
SOIL						
FUTURE LAND USE						
Residential	Direct soil contact on-site	Ingestion Inhalation Dermal	Yes Yes Yes	No No No	Potential	Residential use of site highly unlikely

3.2.3 Exposure Points and Routes

Potential or existing exposure points and routes are evaluated in this section for on and off-site areas and populations, as well as present and possible future land uses. Table 3.2 summarizes possible exposure pathways.

Ground Water Exposure

Ground water in this area has never been used for public water supply due to naturally high levels of total dissolved solids (TDS) and sulfate that make the water unpalatable for drinking. One private well (4626G), located on the northern perimeter of the groundwater plume, has been used, and is currently available for domestic use, irrigation, and for filling a swimming pool. Although there are no other known private domestic wells in use down-gradient of the facility, such wells may exist or could be drilled in the area. An irrigation supply well (SRP 18E-5N), used to augment flows in the Grand Canal and Lateral 7.0, is also within the area of the groundwater plume. The water is used for irrigation.

There are no other known exposures to the ground water in this area. In Chapter 2 thirty-four (34) COPC were selected for the alluvial groundwater and at the alluvium-bedrock interface. The only known exposure points are the two wells mentioned in the preceding paragraph. However, samples taken from well 4626G during the period from 1987 to 1992 indicated that boron, fluoride, and lead were above the HBGL or MCL and, for well 18E-5N, only boron, fluoride, nitrate and sulfate were above the criteria. The possibility exists that there are unknown exposure points (private wells) and there is no statutory restraint on the development of private wells in the area. Therefore, to better evaluate known and potential exposures to groundwater, potential risks will be evaluated for all sampled wells. There are three possible complete exposure pathways to contaminated groundwater: ingestion, inhalation, and dermal contact.

Surface Water Exposure

The only potential impact on surface water by the groundwater plume is at SRP well 18E-5N, where groundwater is mixed with canal water for irrigation purposes. No data have been provided that indicates other surface waters have been impacted by contaminant releases from the Motorola 52nd Street facility. There are drainage areas to the southwest of the facility that can not be entirely ruled out in the absence of confirming data. However, soil gas sampling has not indicated the presence of significant amounts of VOCs in the area.

Air Exposure

There are two possible sources of air exposures due to the uncontrolled releases addressed in this risk assessment; VOC vapors from the soil, and fugitive dust. The groundwater treatment plant that is being constructed on the site is designed to be a closed loop system that will not emit VOC vapors recovered from the groundwater. Under current land use, on-site exposure points include workers at the facility, both outdoors and indoors. Soil gas investigations have confirmed the presence of high levels of VOC vapors in soils of some areas on the Motorola site. Concentrations of VOCs in soil gas in residential neighborhoods surrounding the site were low or not detectable in results from samplings done in 1984 and 1985. Off-site sampling of soil gas completed in 1992 show that low concentrations of VOCs are currently present in soil gas at some locations immediately to the west of the facility. On-site samplings were performed in 1989 and 1991.

Complete exposure pathways off-site are possible at residences near the site and will be evaluated for indoor and outdoor exposures using available data. Occupational exposure pathways on-site are complete and will be evaluated for both indoor and outdoor exposures.

Exposure pathways for fugitive dust are complete during construction activities on-site and are possible off-site due to dust migration. There are also areas of bare soil on-site which could produce fugitive dust due to vehicular traffic or wind erosion. These exposure routes are intermittent. Current data are insufficient for a quantitative assessment of risk due to fugitive dust. No data have been provided that suggest that off-site surface soils are contaminated due to the releases discussed in this risk assessment. The exposure pathway due to fugitive dust originating off-site is considered incomplete.

Soil Exposure

Known soil contamination is limited to on-site areas. Under current land use, this exposure pathway can be complete only when the pavement covering most of the site is broken during construction activities or, for workers in unpaved areas. At present only soil gas data are available for volatiles in on-site soils. The greatest exposure to volatiles is through inhalation; this exposure is included under air exposures. Data provided characterizing the status of other potential contaminants, such as metals, in the soil on-site were insufficient for a quantitative assessment of risks to individuals coming in contact with soil on-site during construction activities.

3.2.4 Summary of Exposure Points to be Quantified

Exposure pathways evaluated, and those selected for quantified risk analysis, are summarized in Table 3.2. Six complete exposure pathways are known to exist. The data available are not sufficient for a quantitative assessment of risks associated with on-site soil exposure and off site fugitive dust exposures. Considering the recent history of the site and present zoning constraints it is not likely that the site will be put to residential use. This pathway is not considered complete. Soil gas concentrations on-site will be evaluated under the residential soil gas assessment in order to provide a comparison to off-site areas. At present there are only two known exposure points to chemicals in the groundwater plume; however, the *potential* risk associated with the entire plume will be evaluated. Routes of exposure to be evaluated are:

- 1) Residential use of groundwater over the area of the entire plume.
- 2) Residential use of private well 4626G.
- 3) Residential and agricultural use of irrigation water supplemented by SRP well 18E-5N.
- 4) Residential and occupational exposures to soil gas, both on- and off-site.

3.3 DATA MODELING

Modeling was used sparsely and, only when necessary. No modeling was done with groundwater concentrations. Figures used were those reported by laboratory analysis of the groundwater for the period 1988 to 1991. This is a conservative approach. It does not take into account the decrease in concentration of COPC during the 30 year assumed exposure to groundwater used in the risk calculations. Concentrations decrease due to migration with groundwater movement, transformations, and biodegradation. Exposures to soil gas were modeled using reported subsurface concentrations.

3.3.1 Soil Gas

Soil gas emissions were modeled using results obtained at each sampling point for soil vapor phase concentrations. The 1984 and 1985 samplings covered a very large area and concentrations were very dependent on location, therefore, it was not considered practical to average results over the area. Data used for the soil gas exposure assessment included all results reported for the 1984, 1985, 1989, 1991, and 1992 sampling events (Appendix Tables 3, through 7). The 1989 and 1991 data included only on-site locations (Appendix Figures 1, 2, 3, 4). The 1992 sampling locations were all off-site (Appendix Figure 5).

Assumptions for estimating exposure concentrations for three population groups in areas with soil gas sampling data from 1984 to 1991, including on-site sampling locations, are shown in Table 3.3. Assumptions used for the 1992 data are shown in Appendix Table 11. The three groups are: 1) on-site employees with outdoor exposures; 2) on-site employees with indoor exposures; and 3) area residents. On-site sampling locations were also included in assessment of residential exposures for comparison. Figures used in calculations of outdoor and indoor concentrations are shown in Appendix Tables 8 through 11.

Chemicals in the vapor phase will diffuse through the soil at a rate dependant on the concentration gradient in the soil, the soil porosity, and tortuosity. Millington and Quirk (1961) suggested an empirical model to calculate an effective diffusion coefficient:

$$D_s = \frac{D_o \times (P_a)^{10/3}}{(P_t)^2}$$

where: D_s = effective vapor phase diffusion coefficient (cm^2/sec);

D_o = vapor phase diffusion coefficient in air (cm^2/sec);

P_a = air filled porosity (unitless); and

P_t = total porosity (unitless).

The flux rate was determined by the following equation, as simplified by Karimi et al. (1987):

$$J = \frac{-D_s \times (C_s - C_g)}{L}$$

where: J = flux rate of the vapors through the soil ($\text{g}/\text{m}^2\text{-sec}$);

D_s = effective vapor phase diffusion coefficient (m^2/sec);

C_s = vapor phase concentration at the soil surface (g/m^3);

C_g = vapor phase concentration in the soil at depth L (g/m^3); and

L = thickness of the clean soil layer (m).

Karimi et al. (1987) suggested simplifying this equation by assuming C_g to be zero. C_g is very small compared to C_s and the assumption yields a liberal estimate of the flux rate, since values greater than zero will give a lower flux rate. This leads to a more conservative estimate of risk.

Table 3.3. - Equations and assumptions for calculation of soil gas exposure concentrations.

(1) KARIMI MODEL FOR ESTIMATING FLUX RATE FROM SOIL:

$$J = [(-D_s)(C_s - C_d)]/L$$

WHERE:

- $D_s = [(D_o)(P_a^{10/3})]/(P_t^2)$
- $P_a =$ AIR FILLED POROSITY = 0.25
- $D_o =$ DIFFUSION COEFFICIENT
- $P_t =$ TOTAL POROSITY = 0.45
- $C_s =$ VAPOR PHASE CONCENTRATION AT THE SOIL SURFACE (g/m^3) = 0
- $C_d =$ VAPOR PHASE CONCENTRATION AT DEPTH L (g/m^3) (AS MEASURED)
- $L =$ THICKNESS OF CLEAN SOIL LAYER (AS MEASURED)

(2) ESTIMATION OF OUTDOOR AIR CONCENTRATIONS (OAC):

$$OAC = E/[W(H)(U)]$$

- ASSUMPTIONS *:
- AREA OF CONCERN (A_c) = 2500 m^2
 - AREA OF EMISSION (A_e) = 12.5 m^2 (ASSUMES 0.5% OF PAVED SURFACE IS CRACKED)

WHERE:

- $E =$ EMISSION RATE INTO BOX ($E = (J)(A_e)$) g/day
- $W =$ SQUARE ROOT AREA OF BOX 50 m
- $H =$ HEIGHT OF BOX 1.5 m
- $U =$ WIND VELOCITY 2.8 m/sec

(3) ESTIMATION OF INDOOR AIR CONCENTRATIONS (IAC):

$$IAC = [(J)(A)(F)]/[(ACH/3600)(V)]$$

WHERE:

- $J =$ FLUX RATE OF VAPORS THROUGH SOIL
- $A =$ AREA OF INFILTRATION ^b 0.47 m^2
- $F =$ FRACTION OF GAS FLUX ENTERING BUILDING 1
- $ACH =$ AIR CHANGES PER HOUR 0.2
- $3600 =$ SECONDS PER HOUR 3600 sec/hr
- $V =$ VOLUME OF AIR IN BUILDING 350 m^3

a: VALUES SHOWN WERE USED FOR THE ON-SITE AREA. VALUES FOR OFF-SITE CALCULATIONS WERE; AREA OF CONCERN = AREA OF EMISSION = 100 m^2 AND $W = 10$ m. UNPAVED AREAS ARE ASSUMED.

b: ASSUMES A 1 cm WIDE CRACK AROUND PERIMETER OF BUILDING, AREA = 140 m^2 .

The total soil porosity (P^t) was assumed to be 0.45, based on information in the RI report (Dames and Moore, 1987a). This is a reasonable figure for soils with high percentages of silt and sand. Assuming a soil bulk density of about 1.7 Mg/m^3 and a moisture content of 12 g/100g , the air filled porosity (P_a) is about 0.25. The soil thickness used in the calculations is the sampling depth.

Outdoor Air Exposures

The Karimi model was used to estimate the flux rate, using the reported soil gas figures from the soil gas data. An emission can be calculated by introducing an area term. Outdoor exposure concentrations were estimated using a simple Box model:

$$\text{OAC} = (E) (W)^{-1} (H)^{-1} (U)^{-1}$$

where: OAC = outdoor air concentration (g/m^3);

E = emission rate ($E = J \times A_e$);

J = flux rate ($\text{g/m}^2\text{-sec}$);

A_e = area of emission (m^2);

W = square root of box area (m);

H = height of box (m); and

U = wind velocity (m/sec).

The on-site sampling data was predominantly from the court yard area and the southwest parking lot and surrounding area (SWPL). The highest levels of VOCs in soil gas have been reported in these areas. It was assumed that 0.5% of the total paved areas were occupied by cracks through which soil gas could diffuse. This is a liberal estimate based on observations of paved areas. The area occupied by cracks and/or bare ground is A_e . The height of the box is taken to be 1.5 m, approximately the average human nose height. Wind velocity was estimated at 2.8 m/sec , a value considered to be normal for the Phoenix area by the National Weather Service. A reasonable, but low value was chosen to maximize exposure concentrations.

Indoor Air Exposures

A one compartment indoor air model was used to estimate indoor air exposure concentrations. The model assumes that gas entering a structure is instantaneously mixed within the entire volume. VOC concentrations within a building are a function of the flux from the soil, the area and volume of the

building, and the rate of air exchange for the structure. The building was assumed to have a concrete slab foundation, for this assessment, as this is the most common mode of construction in the Phoenix area. Gas entry was assumed to be via a 1 cm crack around the perimeter of the foundation. This is a common assumption for assessments of this type and is intended as an upper bound estimate. Actual area of cracks should be lower. Indoor air concentrations were estimated using the following model:

$$IAC = \frac{J \times A_e \times F}{(ACH/3600) \times V}$$

where: IAC = indoor air concentration (g/m³);
J = chemical flux from soil gas (g/m²-sec);
A_e = area of emission (m²);
F = fraction of soil gas entering the building (Assumed = 1);
ACH = air changes per hour (hr⁻¹);
3600 = seconds/hour; and
V = volume of air in building (m³).

The calculations of exposure concentrations for indoor air assume a room area of 140 m². The volume is based on a ceiling height of 2.5 m. The fraction of the gas entering the building is assumed to be 100%; for other types of foundations it may be less. The air exchange rate may vary from 0.2 to 1.5 per hour depending on the age of the structure, its energy efficiency, and type of ventilation system. It is taken to be 0.2 per hour for this assessment in order not to underestimate calculated exposures. All sampling sites with a reported concentration greater than 1 µg/L (1 µg/L = 1 mg/m³) for at least one analyte were assessed. Appendix Tables 8 to 11 are worksheets for the calculations. Results of the soil gas modeling are summarized Tables 3.4 to 3.10.

Table 3.5 also shows the ambient air concentrations at the time of sampling in 1989. The ambient concentrations are several orders of magnitude higher than the modelled data due to the contribution of sources other than soil gas.

Table 3.4. -- Outdoor air concentrations of chemicals modeled from concentrations detected in soil gas samples collected in 1984 and 1985. (mg/m3)^a

	Location	PCE	TCA	TCE	F113
OFF-SITE	1030	2.9E-06	8.0E-09	1.0E-05	4.7E-05
	1031	3.8E-06	1.0E-08	6.2E-07	2.3E-07
	1040	3.8E-06	3.0E-06	3.1E-08	2.3E-05
	1094	6.7E-07	9.0E-07	4.2E-09	4.6E-06
	2043	1.4E-05	2.3E-08	2.3E-05	8.7E-06
	2045	4.0E-06	4.2E-09	6.2E-06	1.4E-05
	2054	1.1E-07	4.2E-09	1.9E-06	1.4E-08
	2055	2.3E-05	8.2E-09	6.0E-06	2.8E-07
	2056	3.4E-06	3.6E-07	1.2E-06	5.6E-06
	2057	2.6E-07	3.6E-08	3.7E-08	5.2E-06
	2069	1.4E-05	9.0E-08	3.7E-07	8.3E-07
	2072	5.7E-09	3.0E-09	5.6E-06	1.4E-09
	2087	2.3E-05	4.8E-06	NR	2.1E-05
	2088	3.4E-05	6.0E-06	1.2E-07	4.9E-05
	2089	5.1E-06	5.4E-07	3.7E-08	4.9E-06
	2090	2.3E-06	3.0E-08	3.1E-06	6.9E-07
	2114	3.4E-06	6.0E-07	1.2E-07	6.9E-06
	2120	2.9E-06	1.2E-09	1.2E-07	6.9E-07
	2130	3.4E-06	4.8E-08	4.4E-06	3.5E-06
	ON-SITE	1021	7.1E-08	7.5E-09	1.2E-08
1023		1.2E-08	3.8E-08	7.8E-09	1.4E-08
1024		1.2E-08	5.0E-08	2.6E-09	5.8E-08
1100		1.2E-07	2.5E-10	1.0E-09	2.9E-09
2122		9.2E-08	1.6E-10	1.7E-08	3.7E-09
2123		4.3E-06	3.0E-08	1.6E-08	3.5E-08
2127		8.6E-10	3.6E-09	9.4E-08	4.2E-09
2128		6.4E-10	2.0E-10	1.2E-06	7.8E-10
2131		2.9E-07	3.0E-07	6.2E-08	6.9E-08
2132		3.4E-04	3.2E-04	4.7E-06	1.0E-04
2133		8.6E-05	2.3E-04	1.2E-04	1.0E-03
2134		6.1E-09	6.4E-08	3.3E-09	3.7E-07
2135		4.8E-10	7.5E-09	1.0E-09	5.8E-08
2137		7.8E-07	4.1E-07	4.3E-08	2.4E-07
2138		1.1E-05	1.5E-06	1.2E-07	5.2E-06
2139		3.3E-06	5.2E-10	9.0E-08	6.0E-07
2140		1.4E-10	4.1E-09	4.3E-08	2.4E-09
2141		8.6E-08	2.3E-08	9.4E-08	5.2E-09
2143		8.2E-06	8.7E-09	3.6E-08	4.0E-07
2144		1.5E-08	4.5E-10	2.1E-09	5.2E-08

a. Refer to Table 3.3 for models and assumptions used for calculations.

Table 3.5 – Outdoor air concentrations of chemicals modeled from concentrations detected in soil gas samples collected on-site, 1989. (mg/m³)

LOCATION	PCE	TCA	TCE	F113
SOIL BORING				
	(modeled)			
18-89-1	7.14e-09	1.35e-09	5.87e-08	4.34e-10
18-89-2	1.78e-09	1.15e-09	7.83e-08	4.34e-10
22-89-01	3.57e-08	3.84e-09	3.92e-10	1.30e-07
22-89-02	2.32e-07	9.61e-10	7.83e-13	4.34e-08
22-89-03	1.07e-07	1.73e-09	5.87e-09	1.30e-08
CY-89-02	3.57e-08	5.76e-09	3.92e-09	1.30e-08
CY-89-05	3.57e-08	1.73e-08	3.52e-07	2.17e-07
CY-89-06	7.14e-09	1.15e-08	1.96e-08	4.34e-09
CY-89-07	3.96e-06	2.25e-06	1.14e-06	3.04e-07
CY-89-08	2.68e-07	3.27e-07	7.05e-07	3.91e-07
CY-89-09	8.03e-07	1.15e-07	3.92e-07	1.52e-07
CY-89-10	3.10e-06	3.84e-07	8.81e-07	2.69e-06
SV89-01A	7.14e-08	1.15e-09	3.92e-09	4.34e-10
SV89-02	8.92e-08	1.92e-09	3.92e-09	4.34e-10
SV89-03	3.57e-07	1.15e-09	5.87e-09	4.34e-10
SV89-04	5.35e-08	1.54e-09	1.17e-09	8.68e-10
AIR				
	(Measured) ¹			
SV89	2.0e-02	2.0e-02	<2.0e-04	NR
18-89	8.0e-03	4.0e-03	<2.0e-04	<2.0e-02
22-89	2.0e-03	2.0e-02	<1.0e-04	NR
-89	1.0e-02	NR	<4.0e-05	NR
-89	6.0e-03	2.0e-03	6.0e-05	<4.0e-05
CY-89	7.0e-04	8.0e-04	<4.0e-05	<4.0e-05

1. Ambient air concentrations were measured at the time soil gas was sampled. The ambient air concentrations are higher than the modelled concentrations due to contributions from other sources.
NR= Not Reported

Table 3.6. -- Outdoor air concentrations of chemicals modeled from concentrations detected in soil gas samples collected from on-site, 1991. (mg/m³).

LOCATION	1,1-DCE	PCE	TCA	TCE	F113	VC
SG-138-01	5.90e-09	1.40e-06	4.54e-09	2.31e-09	3.42e-09	2.03e-10
SG-138-02	6.53e-07	2.47e-06	3.04e-07	4.16e-09	2.31e-07	2.03e-10
SG-138-03	2.60e-06	8.72e-07	4.24e-07	4.93e-09	1.71e-10	2.03e-10
SG-138-04	1.86e-06	2.22e-06	4.17e-07	4.62e-10	1.71e-10	2.09e-08
SG-138-05	2.45e-05	8.69e-06	2.46e-06	2.39e-08	1.71e-10	2.03e-10
SG-138-06	6.58e-07	1.32e-07	2.82e-07	4.78e-09	7.25e-07	2.03e-10
SG-138-07	1.36e-08	6.68e-07	9.87e-08	1.54e-10	4.10e-09	2.03e-10
SG-138-08	NR	NR	1.51e-05	4.42e-08	NR	2.03e-10
SG-138-09	9.16e-05	1.59e-06	4.78e-05	1.54e-08	1.71e-08	2.03e-08
SG-138-09B	1.40e-05	6.78e-07	1.51e-05	NR	NR	1.04e-08
SG-138-10A	1.40e-05	4.41e-07	7.31e-07	9.30e-08	1.71e-10	1.50e-07
SG-138-10B	2.29e-05	6.27e-07	1.36e-06	8.60e-08	1.71e-10	2.06e-07
SG-138-11	1.40e-05	1.42e-06	9.52e-06	NR	NR	2.03e-10
SG-138-12	4.69e-06	2.03e-06	1.03e-06	1.11e-08	1.71e-10	2.50e-08
SG-138-13	7.15e-08	1.40e-10	3.14e-07	1.54e-10	5.89e-08	2.03e-10
SG-138-14	4.64e-09	7.53e-08	5.14e-09	1.54e-10	1.03e-08	2.03e-10
SG-138-15	3.14e-06	3.26e-07	3.51e-07	2.51e-08	1.71e-10	2.03e-10
SG-138-16	4.44e-06	1.06e-06	9.26e-07	1.46e-08	1.71e-10	2.03e-10
SG-138-17	1.61e-07	7.60e-07	1.61e-07	1.54e-10	3.06e-07	2.03e-10
SG-138-18A	1.40e-10	1.51e-07	2.27e-09	1.54e-10	6.32e-09	2.03e-10
SG-138-18B	1.40e-10	2.01e-07	1.26e-08	2.47e-09	5.13e-08	2.03e-10
SG-138-19A	1.40e-10	1.33e-08	1.51e-10	1.39e-09	1.71e-10	2.03e-10
SG-138-19B	1.40e-10	9.83e-10	1.51e-10	1.39e-09	1.71e-10	2.03e-10
SG-138-20	1.40e-10	4.21e-09	2.60e-08	1.54e-10	1.71e-10	2.03e-10
SG-138-21	1.48e-08	9.86e-08	8.17e-09	1.54e-10	2.97e-08	2.03e-10
SG-138-22	1.40e-10	1.40e-10	1.51e-10	1.04e-07	1.71e-10	2.03e-10
SG-138-23	1.40e-10	2.81e-09	1.51e-10	4.90e-08	1.71e-10	2.03e-10

NR= Not Reported

Table 3.7 – Indoor air concentrations of chemicals modeled from concentrations detected in soil gas samples collected in 1984 and 1985. (mg/m³)

LOCATION	PCE	TCA	TCE	F113
1021 ^s	2.85e-05	3.01e-06	4.68e-06	2.60e-05
1023 ^s	4.75e-06	1.50e-05	3.12e-06	5.78e-06
1024 ^s	4.75e-06	2.00e-05	1.04e-06	2.31e-05
1030	2.85e-05	8.02e-08	1.04e-04	4.74e-04
1031	3.80e-05	1.00e-07	6.24e-06	2.31e-06
1040	3.80e-05	3.01e-05	3.12e-07	2.31e-04
1094	6.66e-06	9.02e-06	4.16e-08	4.63e-05
1100 ^s	4.75e-05	1.00e-07	4.16e-07	1.16e-06
2043	1.50e-04	2.37e-07	2.47e-04	9.13e-05
2045	3.99e-05	4.21e-08	6.24e-05	1.39e-04
2054	1.14e-06	4.21e-08	1.87e-05	1.39e-07
2055	2.45e-04	8.59e-08	6.24e-05	2.97e-06
2056	3.42e-05	3.61e-06	1.25e-05	5.55e-05
2057	2.59e-06	3.64e-07	3.78e-07	5.26e-05
2069	1.34e-04	8.84e-07	3.67e-06	8.16e-06
2072	5.71e-08	3.01e-08	5.62e-05	1.39e-08
2087	2.28e-04	4.81e-05	NR	2.08e-04
2088	3.42e-04	6.01e-05	1.25e-06	4.86e-04
2089	5.13e-05	5.41e-06	3.75e-07	4.86e-05
2090	2.28e-05	3.01e-07	3.12e-05	6.94e-06
2114	3.42e-05	6.01e-06	1.25e-06	6.94e-05
2120	2.85e-05	1.20e-08	1.25e-06	6.94e-06
2122 ^s	3.72e-05	6.54e-08	6.79e-06	1.51e-06
2123 ^s	1.71e-03	1.20e-05	6.24e-06	1.39e-05
2127 ^s	3.57e-07	1.50e-06	3.90e-05	1.73e-06
2128 ^s	2.59e-07	8.20e-08	4.73e-04	3.15e-07
2130	3.42e-05	4.81e-07	4.37e-05	3.47e-05
2131 ^s	1.14e-04	1.20e-04	2.50e-05	2.78e-05
2132 ^s	1.27e-01	1.17e-01	1.73e-03	3.85e-02
2133 ^s	3.57e-02	9.40e-02	4.88e-02	4.34e-01
2134 ^s	2.59e-06	2.73e-05	1.42e-06	1.58e-04
2135 ^s	1.84e-07	2.91e-06	4.03e-07	2.24e-05
2137 ^s	3.08e-04	1.63e-04	1.69e-05	9.38e-05
2138 ^s	4.56e-03	6.01e-04	5.00e-05	2.08e-03
2139 ^s	1.30e-03	2.05e-07	3.55e-05	2.37e-04
2140 ^s	5.40e-08	1.63e-06	1.69e-05	9.38e-07
2141 ^s	3.17e-05	8.35e-06	3.47e-05	1.93e-06
2143 ^s	3.24e-03	3.42e-06	1.42e-05	1.58e-04
2144 ^s	6.05e-06	1.82e-07	8.52e-07	2.10e-05

S. Samples taken on the Motorola facility grounds.
NR= Not Reported

Table 3.8. — Indoor air concentrations of chemicals modeled from concentrations detected in soil gas samples collected on-site, 1989. (mg/m³)

LOCATION	PCE	TCA	TCE	F113
18-89-1	2.90e-06	5.46e-07	2.39e-05	1.76e-07
18-89-2	7.25e-07	4.68e-07	3.18e-05	1.76e-07
22-89-01	1.45e-05	1.56e-06	1.59e-07	5.29e-05
22-89-02	9.42e-05	3.90e-07	3.18e-10	1.76e-05
22-89-03	4.35e-05	7.02e-07	2.39e-06	5.29e-06
CY-89-02	1.45e-05	2.34e-06	1.59e-06	5.29e-06
CY-89-05	1.45e-05	7.02e-06	1.43e-04	8.81e-05
CY-89-06	2.90e-06	4.68e-06	7.95e-06	1.76e-06
CY-89-07	1.61e-03	9.13e-04	4.61e-04	1.23e-04
CY-89-08	1.09e-04	1.33e-04	2.86e-04	1.59e-04
CY-89-09	3.26e-04	4.68e-05	1.59e-04	6.17e-05
CY-89-10	1.26e-03	1.56e-04	3.58e-04	1.09e-03
SV89-01A	2.90e-05	4.68e-07	1.59e-06	1.76e-07
SV89-02	3.62e-05	7.80e-07	1.59e-06	1.76e-07
SV89-03	1.45e-04	4.68e-07	2.39e-06	1.76e-07
SV89-04	2.17e-05	6.24e-07	4.77e-07	3.52e-07

Table 3.9. -- Indoor air concentrations of chemicals modeled from concentrations detected in soil gas samples collected from on-site, 1991. (mg/m³)

LOCATION	1,1-DCE	PCE	TCA	TCE	F113	VC
SG-138-01	2.40e-06	5.67e-04	1.84e-06	9.39e-07	1.39e-06	8.25e-08
SG-138-02	2.65e-04	1.00e-03	1.23e-04	1.69e-06	9.39e-05	8.25e-08
SG-138-03	1.06e-03	3.54e-04	1.72e-04	2.00e-06	6.94e-08	8.25e-08
SG-138-04	7.54e-04	9.00e-04	1.69e-04	1.88e-07	6.94e-08	8.50e-06
SG-138-05	9.96e-03	3.53e-03	1.00e-03	9.70e-06	6.94e-08	8.25e-08
SG-138-06	2.67e-04	5.36e-05	1.15e-04	1.94e-06	2.94e-04	8.25e-08
SG-138-07	5.53e-06	2.71e-04	4.01e-05	6.26e-08	1.67e-06	8.25e-08
SG-138-08	NR	NR	6.14e-03	1.80e-05	NR	8.25e-08
SG-138-09	3.72e-02	6.45e-04	1.94e-02	6.26e-06	6.94e-06	8.25e-06
SG-138-09B	5.71e-03	2.75e-04	6.14e-03	NR	NR	4.21e-06
SG-138-10A	5.67e-03	1.79e-04	2.97e-04	3.77e-05	6.94e-08	6.07e-05
SG-138-10B	9.28e-03	2.55e-04	5.52e-04	3.49e-05	6.94e-08	8.37e-05
SG-138-11	5.71e-03	5.77e-04	3.87e-03	NR	NR	8.25e-08
SG-138-12	1.90e-03	8.24e-04	4.16e-04	4.51e-06	6.94e-08	1.01e-05
SG-138-13	2.90e-05	5.71e-08	1.27e-04	6.26e-08	2.39e-05	8.25e-08
SG-138-14	1.88e-06	3.06e-05	2.09e-06	6.26e-08	4.16e-06	8.25e-08
SG-138-15	1.27e-03	1.32e-04	1.42e-04	1.02e-05	6.94e-08	8.25e-08
SG-138-16	1.80e-03	4.31e-04	3.76e-04	5.95e-06	6.94e-08	8.25e-08
SG-138-17	6.56e-05	3.09e-04	6.53e-05	6.26e-08	1.24e-04	8.25e-08
SG-138-18A	5.71e-08	6.12e-05	9.22e-07	6.26e-08	2.57e-06	8.25e-08
SG-138-18B	5.71e-08	8.15e-05	5.10e-06	1.00e-06	2.08e-05	8.25e-08
SG-138-19A	5.71e-08	5.42e-06	6.14e-08	5.63e-07	6.94e-08	8.25e-08
SG-138-19B	5.71e-08	3.99e-07	6.14e-08	5.63e-07	6.94e-08	8.25e-08
SG-138-20	5.71e-08	1.71e-06	1.06e-05	6.26e-08	6.94e-08	8.25e-08
SG-138-21	5.99e-06	4.00e-05	3.32e-06	6.26e-08	1.21e-05	8.25e-08
SG-138-22	5.71e-08	5.71e-08	6.14e-08	4.22e-05	6.94e-08	8.25e-08
SG-138-23	5.71e-08	1.14e-06	6.14e-08	1.99e-05	6.94e-08	8.25e-08

NR= Not Reported

Table 3.10. -- Calculation of exposure concentrations due to soil gas release for March, 1992 sampling sites, using maximum detected concentrations.

	OUTSIDE AIR CONCEN. (mg/m ³)	INSIDE AIR CONCEN. (mg/m ³)
BENZENE	7.45E-07	7.56E-06
TOLUENE	1.85E-07	1.88E-06
ETHYLBENZENE	9.43E-08	9.58E-07
XYLENE	2.92E-07	2.97E-06
1,1-DCE	4.16E-05	4.22E-04
t-1,2-DCE	6.86E-07	6.96E-06
PCE	9.52E-06	9.66E-05
TCE	2.15E-07	2.18E-06
F-113	8.80E-05	8.93E-04

Table 3.11 -- Concentrations (mg/L) of chemicals of concern from SRP well 18E-5N before and after dilution mixing with Grand Canal water.^a

Chemical	Concentration from Well	Concentration after Dilution
Boron	1.9	0.006 - 0.03
Fluoride	4.5	0.02 - 0.08
Nitrate	42.8	0.15 - 0.73
Sulfate	354	1.2 - 6.0

3.3.2 Groundwater Exposures

Four potentially complete exposure routes for groundwater vapor releases have been identified; (1) private well 4626G: domestic water use (drinking, bathing, cooking, etc.); (2) private well 4626G: swimming pool; (3) private well 4626G: spray irrigation; and (4) SRP well 18E-5N.

Private Well 4626G

The limited sampling data collected for well 4626G over the last four years (Table 2.6), show VOC concentrations to be below USEPA drinking water MCLs and Arizona HBGLs. Inorganics identified using well specific data were boron, fluoride, and lead. Exposures from household use of private well 4626G have occurred in the past and could in the future. The well is not currently being used for household purposes and current data does not indicate potentially significant exposures to VOCs from this source (Table 2.6). Domestic use of the well as a drinking water source will be evaluated for chemicals of concern selected from the entire data set, using standard assumptions. Potential risks from swimming an irrigation uses will be assessed qualitatively, due to the lack of quantitative data.

SRP Well 18E-5N

No organic chemicals of concern were found using data reported for the SRP well (Table 2.7). Several inorganic constituents were present above HBGL or MCL levels and will be evaluated. The SRP well should be monitored when in use due to its location relative to the groundwater plume. Groundwater modeling has indicated that the well may be impacted by the plume in the future (Dames and Moore, 1992).

SRP well 18E-5N is located on the outer edge of the groundwater plume (Dames and More, 1991a and 1992). Groundwater from the well intermittently discharges to either the Grand Canal or Lateral 7, which may be used for commercial, industrial, agricultural, and residential flood irrigation. The area served by Lateral 7 is primarily industrial and includes a major portion of Sky Harbor International Airport, various commercial/industrial operations, and the Southern Pacific Railroad. The Grand Canal is in an open channel serving numerous discharge points until emptying into the Agua Fria River to the northwest (Figure 3.7). Water from SRP well 18E-5N is diluted when entering the canal. The pumping rate of the well is 1.7 cubic feet per second (CFS). The average flow rate of the Grand Canal is approximately 100 CFS in the winter and 500 CFS during the summer. Using these figures the amount by which the well water is diluted when it enters the canal (dilution factor) may be calculated. The dilution factor is about 59 in the winter and 294 in the summer. This reduces the concentrations of

inorganic chemicals of concern to very low levels (Table 3.11). No data on canal water composition were provided. The addition of water from well 18E-5N to the canal water will raise the levels of the four chemical in Table 3.11 by the amounts in the right column. No matter what the ambient concentrations for the canal water are, the impact of the added water is minimal and should not pose a risk to persons using canal water for irrigation purposes or consuming crops irrigated with the canal water. This exposure point will not be evaluated further. The well is located near the southern edge of the groundwater plume. Groundwater modeling has indicated that the well may be impacted by the contaminant plume in the future (Dames and Moore 1992). Therefore, it is recommended that well 18E-5N be monitored closely, when in use, particularly after it has been in service for a prolonged period of time (at least 24 to 72 hrs). Prolonged pumping of production wells has been shown to produce a cone of depression in the groundwater level which can influence movement of the contaminant plume toward the well.

3.3.3 Summary of Exposure Concentrations

Exposure concentrations were based on the 95% upper confidence limit (UCL) for the mean of the reported data when data were available. The maximum reported value was used when insufficient data existed for calculation of a UCL. If only one sampling result was available, the reported value was used.

The groundwater data were separated into two categories: data from wells sampling the alluvium and data from sampling ports at or below the alluvium-bedrock interface. Two private wells (4626G and 18E-5N) were assessed separately (Tables 2.6 and 2.7). Well 4626G was also included in the general groundwater assessment. Data collected from January of 1988 to August of 1991 were used for reasons discussed in Chapter 2.

Exposure concentrations used for calculating potential chronic daily intakes (CDI) from groundwater by ingestion are given in Appendix Table 1. The 95% UCL for each chemical from each well was used to estimate a potential CDI. These will be used in Chapter 5 to estimate a carcinogenic risk and noncarcinogenic hazard quotient for each chemical of concern in each well. The individual estimates will be summed, using standard USEPA procedures to characterize the total potential risk or hazard for each well for which sampling data were received. This procedure was followed in order to better define the potential risks associated with groundwater consumption over the entire area in which monitor wells were located.

Tables 3.4 through 3.9 summarize the soil gas exposure concentrations calculated by methods discussed in Section 3.3.2. The exposure concentrations are based on all sampling points with a concentration of 1 $\mu\text{g}/\text{L}$ for at least one analyte. For the 1992 data the maximum detected concentrations were used to estimate exposure concentrations.

3.4 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 relatively low levels of exposure via environmental media. Three receptor populations have been identified: 1) on-site workers; 2) off-site residents; and 3) users of private well 4626G. Potential exposure pathways for these groups were summarized in Table 3.2. Exposures will be quantified for the following:

1) On-site Workers

- ▶ Outdoor exposure to soil gas releases.
 - ◆ Inhalation
- ▶ Indoor Exposures to soil gas releases.
 - ◆ Inhalation

2) Off-site Residents

- ▶ Potential domestic exposures to groundwater.
 - ◆ Inhalation, Ingestion, Dermal
- ▶ Outdoor exposure to soil gas releases.
 - ◆ Inhalation
- ▶ Indoor exposure to soil gas releases.
 - ◆ Inhalation

3) Users of Private Well 4626G

- ▶ Domestic exposures to groundwater.
 - ◆ Ingestion, Inhalation, Dermal

3.4.1 Exposure Estimation Methods

Exposure concentrations have been calculated and summarized previously. They are estimates of concentrations that are or, potentially could be, contacted at an exposure point. Chemical intake or dose is expressed as mass per unit body weight and time ($\text{mg}/\text{kg}\text{-day}$) and is referred to as the chronic

daily intake (CDI). Most toxicity values are expressed on the basis of administered dose, not adsorbed dose, therefore, chemical intakes are expressed in the same manner.

Variable values incorporate standard assumptions adopted by the USEPA and other agencies for human health risk and exposure assessments (USEPA, 1990a and 1991a). An exposure frequency (EF) of 350 days per year was assumed; this allows for a family spending 15 days per year away from the residence. The standard exposure durations (ED) of 9 years for average exposure and 30 years for RME were used. These assumptions were developed by USEPA from national data indicating that the average American family lives 9 years in a residence and 30 years is the 95% upper bound for residing at a single residence. A mean body weight of 70 kg (154 lb) was used in the calculations; this is the standard assumption developed by USEPA. This is meant to be a representative weight that accounts for the fact that many men weigh more than 70 kg and that many women weigh less. Some assumptions are not standardized and may vary considerably. In such cases professional judgement was exercised.

Residential Drinking Water: Ingestion

The intake equation for calculation of CDI from ingestion exposures for domestic water use is presented in Table 3.12. Variable values for average and reasonable maximum (RME) exposures for adults are shown. Separate calculations were not done for children as results do not differ significantly from those for adults. The intake formulas follow USEPA guidance (USEPA, 1989a). The ingestion rate of 2 liters of water per day is the USEPA guideline and represents approximately the 90th percentile for drinking water consumption. It is also comparable to the 8 glasses of water historically recommended by health professionals. The value of 2 liters per day is also used in the calculation of drinking water standards and health-based guidance levels.

Residential Drinking Water: Inhalation and Dermal

Inhalation and dermal exposures to VOCs present in residential water supplies are known to be significant, although quantitative estimates of exposures vary greatly. Intake of VOCs by inhalation from domestic uses has been estimated to vary from one-quarter to five times that for ingestion (Jo et al. 1990a, Andelman 1985, Andelman et al. 1985). Estimates with a similar range have been made for residential dermal exposures (Brown et al. 1984, Jo et al. 1990b).

Volatile organic compounds transfer from water into air when the water is heated or aerated. Data suggest this process is continuous within the home and leads to an immediate enrichment of respirable air at the point of water use and to a diffusion throughout the home (McKone & Knezovich,

Table 3.12 - Residential ingestion intake from drinking water.

$$\text{CHRONIC DAILY INTAKE: } \frac{(CW)(IR)(EF)(ED)(CF)}{(BW)(AT)}$$

Where:

CW = Chemical Concentration in Water (micrograms/liter)
 IR = Drinking Water Ingestion Rate (liters/day)
 EF = Exposure Frequency (days/year)
 ED = Exposure Duration (years)
 BW = Body Weight (kilograms)
 AT = Averaging Time (days)
 CF = Conversion Factor (1E-3 mg/ug)

Variable Values:	Average	RME
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		

1991). Showers and baths taken within an enclosed bathroom result in the liberation of 43% to 67% of VOCs into the air (Andelman et al, 1985).

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 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 route mentioned above. 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.

Well 4626G: Residential

The exposure concentrations and CDI for COPC for well 4626G, chosen from data for all wells in the study area, are given in Table 3.13. These are the same figures that appear in Appendix Table 1. The 95% UCL is often the same as the mean because there were only one to five analyses available for each analyte. All chemicals of concern determined for the entire data set are included, although when assessed as a separate site the only chemicals with concentrations above the HBGLs or MCLs were boron, fluoride, and lead. Risk assessments were performed using the chemicals of concern developed for the entire data set and for chemicals independently selected for well 4626G.

Soil Gas: Residential

Soil gas exposures were evaluated using the modeled outdoor and indoor concentrations previously presented. CDIs for each residential exposure setting were calculated using the equation and assumptions shown in Table 3.14. The USEPA recommended upper bound for inhalation rates vary from 15 to 20 m³/day (USEPA 1991a). The more conservative, 20 m³/day was used for all scenarios in this assessment.

For outdoor exposures an exposure time (ET) of 2 hours per day for average exposure and 8 hours per day for the reasonable maximum exposure (RME). This is an annual exposure rate and was set to account for variable periods of outdoor activity. Indoor CDI vary from outdoor CDI in the use of modeled indoor air concentrations and the change of ET to 16 hours per day for the average and 24 hours per day for the RME.

Soil Gas: On-site Occupational

On-site occupational exposures assume an EF of 250 days per year; this is a 40 hour work week with 2 weeks of vacation. An ET of 4 hours for average exposure and 8 hours for the RME was used. A four hour period was chosen for the average ET because people often move around during the eight hour work day, this is particularly true for outdoor work. The 8 hour ET represents an upper bound exposure time and is included in the RME calculations. USEPA suggests a default value of 25 years for ED. In this case values of 9 and 30 years were used. This was for two reasons: first, single samples were used to derive risk estimates, so a 95% UCL could not be used for RME calculations, and the use

Table 3.13 - CDI by ingestion, for residential exposure to well 4626G.

Chemical of Concern	Average Exposure			Reasonable Maximum Exposure		
	Mean Concentration	Carcinogenic CDI	Noncarcinogenic CDI	95% UCL ^a Concentration	Carcinogenic CDI	Noncarcinogenic CDI
Arsenic ^a	0.01 mg/L	4.2E-08	3.3E-07	0.02 mg/L	9.5E-08	7.4E-07
Boron ^a	1.1 mg/L	3.9E-06	3.0E-05	1.1 mg/L	3.9E-06	3.0E-05
Bromodichloromethane ^b	0.2 ug/L	7.0E-07	5.5E-06	0.2 ug/L	7.0E-07	5.5E-06
Chlorobenzene ^b	0.4 ug/L	1.8E-06	1.4E-05	0.6 ug/L	1.8E-06	1.4E-05
Chloroform ^a	0.2 ug/L	5.6E-07	4.4E-06	0.4 ug/L	9.2E-07	7.1E-06
1,1-Dichloroethane ^b	0.2 ug/L	7.0E-07	5.5E-06	0.2 ug/L	7.0E-07	5.5E-06
1,1-Dichloroethylene ^b	0.2 ug/L	1.1E-06	8.2E-06	0.4 ug/L	1.8E-06	1.4E-05
1,2-Dichloroethylene ^b	0.2 ug/L	7.0E-07	5.5E-06	0.2 ug/L	7.0E-07	5.5E-06
Fluoride ^a	0.2 mg/L	7.0E-07	5.5E-06	0.2 mg/L	7.0E-07	5.5E-06
Lead ^b	0.01 mg/L	2.8E-08	2.2E-07	0.02 mg/L	9.9E-08	7.7E-07
Nitrate ^a	2.4 mg/L	8.5E-06	6.6E-05	2.4 mg/L	8.5E-06	6.6E-05
Sulfate ^a	180 mg/L	6.2E-04	4.8E-03	260 mg/L	9.1E-04	7.1E-03
Tetrachloroethylene ^b	0.1 ug/L	5.6E-07	4.4E-06	0.2 ug/L	9.2E-07	7.1E-06
Thallium ^b	0.005 mg/L	1.8E-08	1.4E-07	0.005 mg/L	1.8E-08	1.4E-07
1,1,1-Trichloroethane ^b	0.1 ug/L	5.6E-07	4.4E-06	0.2 ug/L	9.2E-07	7.1E-06
Trichloroethylene ^a	0.4 ug/L	1.1E-06	8.2E-06	0.6 ug/L	2.1E-06	1.6E-05
Vinyl Chloride ^b	0.2 ug/L	1.1E-06	8.2E-06	0.4 ug/L	1.8E-06	1.4E-05

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- a. There were positive detections of these chemicals in well 4626G during the period, 1987 to 1991 (refer to Table 2.6).
- b. There were no positive detections of these chemicals in well 4626G during the period 1987 to 1988 (refer to Table 2.6). The results reported represent detection limits or sample quantitation limits. These chemicals have been detected in monitor wells at other locations.
- c. If the 95% UCL is greater than the maximum reported value, then, the maximum reported value is used (refer to Table 2.6).

Table 3.14 - Calculation of inhalation intakes.

$$\text{CHRONIC DAILY INTAKE: } \frac{(CA)(IR)(ET)(CF)(EF)(ED)}{(BW)(AT)}$$

Where:

- CA = Chemical Concentration in Air (milligrams/meter³)
- IR = Inhalation Rate (meters³/day)
- ET = Exposure Time (hours/day)
- CF = Conversion Factor (1 day/24 hours)
- EF = Exposure Frequency (days/year)
- ED = Exposure Duration (years)
- BW = Body Weight (kilograms)
- AT = Averaging Time (days)

Variable Values:

OUTDOOR

	Residential		Occupational	
	Average	RME	Average	RME
IR: (m ³ /day)	20	20	20	20
ET: (hr/day)	2	8	4	8
EF: (days/year)	350	350	250	250
ED: (years)	9	30	9	30
BW: (kg)	70	70	70	70
AT:	For carcinogenic effects = 70 years x 365 days/year			
	For noncarcinogenic effects = ED x 365 days/year			

INDOOR

	Residential		Occupational	
	Average	RME	Average	RME
IR: (m ³ /day)	20	20	20	20
ET: (hr/day)	16	24	4	8
EF: (days/year)	350	350	250	250
ED: (years)	9	30	9	30
BW: (kg)	70	70	70	70
AT:	For carcinogenic effects = 70 years x 365 days/year			
	For noncarcinogenic effects = ED x 365 days/year			

of two EDs allowed the calculation of reasonable average and RME estimates; second, the values used allow for a comparison to residential intakes.

3.4.2 Summary of Exposure Doses

Potential CDI for groundwater used in calculation of estimated carcinogenic risk and noncarcinogenic hazard quotients are shown in Appendix Table 27. Those for private well 4626G are also shown in Table 3.13. Exposure doses (CDI) for soil gas emissions are shown in Appendix Tables 12 to 26.

3.5 UNCERTAINTIES IN THE EXPOSURE ASSESSMENT

Uncertainties enter into the calculations at all levels, for all populations, and land uses.

3.5.1 Exposure Pathways

Exposures calculated from ground water monitoring data are *potential* exposures which *may never be complete*. The exception is the calculated exposures for private well 4626G. Sampling data for this well are minimal. It is recommended that in the future this well be put on a regular sampling schedule of two to four samplings per year. Exposures resulting from use of the swimming pool and irrigation system do not appear to present an unacceptable level of risk based on the data available. This conclusion will be addressed further in Chapter 5.

Other potentially complete exposure pathways include: discharges from SRP well 18E-5N into the Grand Canal and on-site soil contact, including fugitive dust emissions. These pathways are not quantitatively assessed, as previously discussed. All major exposure pathways resulting from uncontrolled releases at the site, for which data was available, have been evaluated.

Quantitative data was not available for assessment of exposures to on-site soils.

3.5.2 Modeling

The major modeling efforts in this assessment are related to the releases of VOCs to the atmosphere from the soil. The assumptions used are designed to produce conservative estimates of risk. The model used has been approved for this use by the USEPA. It should be recognized that anytime a model is used the uncertainty of the estimated quantities is greater than if an accurate measurement were taken. When this is not possible, the use of models greatly increases the range of exposures that can be examined.

3.5.3 Exposure Parameters

All exposure parameters were chosen to produce conservative estimates of total risk from exposures to contaminants for both on- and off-site locations. Exposure concentrations used in the calculation of intakes were mean concentrations for average exposures and 95% upper bounds of the sampling means for RME exposures. For soil gas the reported data was used. When SQLs were reported in the data, the one-half the reported values were used if the chemical had been detected in other samplings. This is a conservative and health protective interpretation of the data.

There is uncertainty attached to each parameter. Slope factors and reference doses are also upper bound estimates. The accumulative effect should be to err on the side of over-estimation of risk.

3.6 EXPOSURE ASSESSMENT SUMMARY

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 in conjunction 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

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) utilized in the risk assessment analysis. Each chemical is summarized with regard to use, interactions with other chemicals, exposure routes, toxicokinetics, toxic (health) effects, and carcinogenicity. The toxicity assessment section is divided into the following four parts:

- * Section 4.1: Dose-Response Variable for Non-Carcinogenic Effects of Chemicals
- * Section 4.2: Dose-Response Variable for Carcinogenic Effects of Chemicals
- * Section 4.3: Toxicity Summaries for the Chemicals of Concern
- * Section 4.4: Summary

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 utilized 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" (USEPA, p. 8-2). 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 indicates further uncertainty as judged by the professional.

$$\text{RfD} = \text{NOAEL} / \text{UF} \times \text{MF} \quad (1)$$

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 utilized 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. Table 4-1 displays RfDs for chemicals of potential concern in this study.

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" (USEPA 1989a, p. 8-2). The SF is an estimate of the quantitative relationship between dose and carcinogenic response.

The SF is measured in units of $(\text{mg}/\text{kg}\cdot\text{day})^{-1}$ 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 utilized 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 utilization 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 found to be 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 Integrated Risk Information System (IRIS) database (USEPA, 1991b), and the USEPA Health Effects Assessment Summary Tables (HEAST), FY-1991 (USEPA, 1991c). Slope factors and weight of evidence ratings for carcinogens are listed in Table 4-2.

4.3 TOXICITY SUMMARIES

The chemicals of concern are discussed with regard to use, chemical interactions, exposure routes, toxicokinetics, toxic (health) effects, and carcinogenicity. These summaries do not represent a comprehensive discussion of these substances, but offer highlights about their toxicity. Reference sources, from which this information was obtained, include the Toxicological Profiles from the Agency for Toxic Substances and Disease Registry for specific chemicals, National Primary Drinking Water Regulations (USEPA, 1987), Draft Health Assessment Guidance Manual (ATSDR, 1990), and Handbook of Toxic and Hazardous Chemicals and Carcinogens (Sitig, 1985).

4.3.1 Arsenic (As)

Arsenic (CAS No. 7440-38-2) is an element which occurs naturally in rocks and soils and is a constituent of various organic and inorganic compounds. Synonyms include arsenic-75, metallic arsenic, arsenic black, and colloidal arsenic. Arsenic is found throughout the environment in soil, air, food, and water and is a component of arsenical pesticides and emissions from metal smelters. Arsenic concentrations are high in certain industries, chemical waste sites, areas where arsenical pesticides have been used, and geographic areas with natural arsenic deposits.

Arsenic has been found to interact with a number of other substances. The mechanism is unknown; however, when arsenic and selenium are administered together, each chemical tends to diminish the effect caused by the other in a mutually antagonistic manner. For instance, high doses of selenium are toxic to livestock. When arsenic is given in the diet or water of livestock, the adverse effect of high doses of selenium (also added to the diet) is diminished. An anticarcinogenic effect in animals and humans has also been observed with low doses of selenium, but arsenic exposure diminishes selenium's anticarcinogenic tendency with a subsequent rise in tumor formation. Selenium administration has produced a protective effect against arsenic-induced chromosome aberrations in human lymphocytes

Table 4.1 - Reference dose (RfD) for ingestion and inhalation for chemicals of concern.

Chemical	Inhalation RfD ¹ (mg/kg-d)	Ingestion RfD ¹ (mg/kg-d)	Confidence In Data ² (Oral)	Sensitive Organs and Systems Affected ³	RfC/RfD ⁴ Source	UF/MF ⁴
Arsenic	— ⁵	3E-4	Medium	Blood, CNS, GI System, Heart, Kidney, Liver, Skin	— /IRIS	3/1
Benzene	—	—	—	Blood; CNS; Developmental; GI, Immune, Reproductive Systems; Skin	—	—
Boron	—	9E-2	Medium	Brain, CNS, GI System, Kidney, Liver, Lung, Skin	— /IRIS	100/1
Bromodichloromethane	—	2E-2	Medium	Adrenal, Blood, Brain, CNS, Developmental & Genotoxicity, Immune System, Kidney, Liver, Lungs ⁶	— /IRIS	1,000/1
Cadmium	—	5E-4	High	GI System, Kidney, Liver, Respiratory System, Skin	— /IRIS	100/10
Carbon Tetrachloride	—	7E-4	Medium	Blood, CNS, Kidney, Liver	— /IRIS	1,000/1
Chlorobenzene	5E-3	2E-2	Medium	CNS, Kidney, Liver	HEAST/IRIS	10,000/- Inh. 1,000/1 Ing.
Chloroform	—	1E-2	Medium	CNS, Kidney, Liver	— /IRIS	1,000/1
Chloromethane	—	—	—	CNS, Kidney, Liver	—	—
Chromium (III)	—	1E+0	Low	Kidney, Liver, Respiratory System, Skin	— /IRIS	100/10
Chromium (VI)	—	5E-3	Low	Kidney, Liver, Respiratory System, Skin	— /IRIS	500/1
Cyanide	—	2E-2	Medium	CNS, Cellular Respiration, Respiratory System, Thyroid	— /IRIS	100/5
Dibromochloromethane	—	2E-2	Medium	CNS, Kidney, Liver, Skin	— /IRIS	1,000/1
1,2-Dichlorobenzene	—	9E-2	Low	CNS, Kidney, Liver, Respiratory System, Skin	— /IRIS	1,000/1
1,4-Dichlorobenzene	—	—	—	Blood, CNS, Kidney, Liver, Respiratory Systems	—	—

Table 4.1 - Continued.

Chemical	Inhalation RfD ¹ (mg/kg-d)	Ingestion RfD ¹ (mg/kg-d)	Confidence In Data ² (Oral)	Sensitive Organs and Systems Affected ³	RfC/RfD ⁴ Source	UF/MF ⁵
1,1-Dichloroethane	1E-1	1E-1	—	CNS, Heart	HEAST/HEAST	1,000/- Inh. 1,000/- Ing.
1,2-Dichloroethane	—	—	—	GI System, Kidney, Liver, Respiratory System, Skin	—	—
1,1-Dichloroethylene	—	9E-3	Medium	Developmental, GI, Respiratory Systems; Liver ⁶	— /IRIS	1,000/1
1,2-Dichloroethylene ⁷	—	2E-2	Low	Blood; CNS; GI, Immune, Respiratory Systems; Kidney; Liver	— /IRIS	1,000/1
Dichloromethane	—	6E-2	Medium	CNS, Kidney, Liver	— /IRIS	100/1
1,3-Dichloropropene	2E-2 mg/m ³	3E-4	Low	CNS, Skin	IRIS/IRIS	10,000/1
Fluoride	—	6E-2	High	Bones and Teeth, CNS, GI System, Heart, Kidney, Lung,	— /IRIS	1/1
Lead	—	—	—	Blood; CNS; Developmental; GI, Immune, and Reproductive Systems; Heart; Kidneys	— / —	— / —
Manganese	4E-4 mg/m ³	1E-1	Medium	CNS, Respiratory System	IRIS/IRIS	300/3-Inh. 1/1-Ing.
Nickel	—	2E-2	Medium	GI System, Respiratory System, Skin	— /IRIS	300/1
Nitrate	—	1.6E+0	High	Blood	— /IRIS	1/1
Silver	—	5E-3	Low	Skin (Argyria)	— /IRIS	3/1
Sulfate	—	—	—	GI Tract	— / —	— / —
Tetrachloroethylene	—	1E-2	Medium	CNS, Kidney, Liver	— / IRIS	1,000/1
Thallium (in soluble salts)	—	7E-5	—	CNS, GI System, Heart, Kidneys, Liver, Lungs, Muscle, Skin	— /HEAST	3,000/ —
1,1,1-Trichloroethane	3E-1	9E-2	—	CNS, GI and Reproductive Systems, Heart, Liver, Lung, Skin	HEAST/HEAST	1,000/-Inh 1,000/-Inh

Table 4.1 - Continued.

Chemical	Inhalation RID (ug/kg-d)	Ingestion RID (ug/kg-d)	Confidence In Data ² (Oral)	Sensitive Organs and Systems Affected ³	RIC/RID ⁴ Source	UF/MF ⁵
1,1,2-Trichloroethane	—	4E-3	Medium	CNS, Skin	—/IRIS	1,000/1
Trichloroethylene	—	6E-3	Low	CNS, Eye, GI System, Heart, Kidney, Liver, Lung	—/ECAO ⁶	—/3,000
Vinyl Chloride	—	—	—	Blood, CNS, Connective Tissue, Genotoxicity, Heart-Circulation, Liver, Lung, Reproductive System, Skin	—/—	—/—
Zinc	—	2E-1	—	GI System, Blood (Anemia)	—/HEAST	10/—

¹ RID, UF, and MF: See text for definition.

RIC: RIC applies to the airborne concentration levels of a substance which results in intakes equal to the RID.

² Confidence in Data: Adequacy of the ingestion data from which RID is derived.

³ Information on Sensitive Organs and Systems derived from the ATSDR Toxicological Profile for the specific substance, Handbook of Toxic and Hazardous Chemicals and Carcinogens (1985) for Nitrates, and the Federal Register (Vol. 52, 130, 8 July 1987) for Sulfates, and are based on human study results.

⁴ When no data were found in IRIS, information was obtained from HEAST.

⁵ All blanks indicate no information was available in IRIS or HEAST.

⁶ Information derived from animal studies.

⁷ RID available only for Trans-1,2-Dichloroethylene, so this value was used.

⁸ RID developed by Environmental Criteria and Assessment Office (ECAO) specifically for this risk assessment.

Table 4.2 - Slope Factor (SF) for carcinogenic chemicals of concern.

Chemical	WoE ¹	Slope Factor ¹		Type of Cancer ³ Inhalation/Ingestion	Study Source of SF	Reference for SF
		Inhalation (ug/m ³) ⁻¹	Ingestion (ug/L) ⁻¹ [(ug/kg-day) ¹]			
Arsenic ¹	A	—	[1.8E+0]	Lung Cancer / Skin Cancer	— / Human	IRIS
Benzene	A	—	[2.9E-2] ¹	Leukemia / Lymphomas ³	Human / Human	IRIS
Bromodichloromethane	B2	—	[1.3E-1]	— / Tumors of Large Intestines, Kidney, Liver ⁴	— / Mouse	IRIS
Carbon Tetrachloride	B2	1.5 E-5	3.7E-6 [1.3E-1]	— / Hepatocellular Carcinoma	Mouse, Rat / Mouse, Rat, Hamster	IRIS
Chloroform	B2	—	[6.1E-3]	— / Kidney and Liver Cancers ⁴	Mouse / Rat	IRIS
Chloromethane	C	1.8 E-2	3.7 E-7 [1.3 E-2]	Kidney / —	Mouse / —	HEAST
Chromium (VI)	A-(Inh.)	1.2 E-2	—	Lung Cancer / —	Human / —	IRIS
Dibromochloromethane	C	—	2.4 E-6 [8.4 E-2]	— / Hepatocellular Adenoma	— / Mouse	IRIS
1,4-Dichlorobenzene	C	—	2.4 E-2	— / Liver Cancer	— / Mouse	HEAST
1,1-Dichloroethane	C	—	—	— / Hemangiosarcoma, Mammary and Liver Cancers, Uterine Polyps ⁴	—	—
1,2-Dichloroethane	B2	2.6 E-5	2.6 E-6 [9.1 E-2]	— / Hemangiosarcoma, Hepatocellular Carcinoma	Mouse, Rat / Mouse Rat	IRIS
1,1-Dichloroethylene	C	—	[6.0E-1]	Kidney and Mammary Cancers, Leukemia, Lung Tumor ⁴ / Liver Tumor ⁴	Mouse / Rat	IRIS
Dichloromethane	B2	4.7 E-7	2.1 E-7 [7.5 E-3]	Liver, Lung / Hepatocellular Neoplasms, Lung, Leukemia, Mammary	Mouse / Mouse Rat	IRIS
1,3-Dichloropropene	B2	3.7 E-5	5.1 E-6 [1.8 E-1]	Lung / Liver Neoplasms, Squamous Cell Carcinoma	Mouse / Rat	HEAST
Lead	B2	—	—	— / Kidney Tumor ⁴	—	—

Table 4.2 - Continued.

Chemical	WoE ¹	Slope Factor		Type of Cancer ²	Study Source of SF	Reference for SF
		Inhalation (ug/m ³) ⁻¹	Ingestion (ug/L) ⁻¹ [(mg/kg-day) ⁻¹]			
Tetrachloroethylene ⁶	B2	5.2 E-7	1.5E-6 [5.1E-2]	Bladder, Cervix, Kidney, Lung and Skin Cancers / Liver Cancer ⁴	Rat, Mouse / Mouse	HEAST
1,1,2-Trichloroethane	C	1.6 E-5	1.6 E-6 [5.7 E-2]	———— / Hepatocellular Carcinoma	———— / Mouse	IRIS
Trichloroethylene ⁶	B2	1.7E-6	3.2E-7 [1.1E-2]	Testicular Tumor, Lymphomas, Cancers of Kidney, Liver, Lungs ⁴ / Leukemia, Cancer of Liver and Kidney ⁴	Mouse / Mouse	HEAST
Vinyl Chloride	A	8.4E-5	5.4E-5 [1.9E+0]	Liver Cancer / Liver Cancer, Lung Tumor ⁴	Human / Rat	HEAST

1. SF and WoE: See text for definition.

2. Type of Cancer: Information derived from the Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for specific chemicals. Unless otherwise stated, Type of Cancer refers to human cancers.

3. Oral Slope Factor was based upon human inhalation exposure data.

4. Refers to animal cancers.

5. The slope factor for arsenic is derived from the unit dose published in IRIS. It is considered to have a high degree of uncertainty.

6. Slope factors for tetrachloroethylene and trichloroethylene have been withdrawn from IRIS for review.

and teratogenesis in hamsters, and possibly against lung cancer in smelter workers exposed to carcinogens which include arsenic. Other interactive effects include a multiplicative effect on lung cancer death with smoking and arsenic inhalation, decrease in the weight gain of rats when cadmium and arsenic were administered simultaneously, decrease in tissue concentrations of arsenic with cadmium exposure, and the interaction of arsenic and aluminum to produce an increase in the aggressive behavior of children.

The routes of exposure include inhalation, ingestion, or dermal contact. Greater absorption occurs with readily soluble compounds. Arsenic (III) is assumed to react with the sulfhydryl group of cellular protein causing toxic effects. Soluble inorganic compounds of Arsenic (V) are also toxic but not to the extent of Arsenic (III). The main toxic agents are the soluble inorganic Arsenic (III) compounds which are absorbed well by the gastrointestinal (GI) tract and lungs and then circulated throughout the body. Over 90% of the trivalent and pentavalent arsenic which is ingested by humans is absorbed by the body. Smaller, airborne arsenic particles are inhaled, absorbed, and eventually excreted in the urine. Inhaled inorganic arsenic has been found distributed in the brain, bones and teeth, hair, nails, heart, kidney, liver, and lungs of human tissues. In one autopsy study of retired refinery and smelter workers, the concentration of inorganic arsenic was six times greater in the lungs of workers when compared to controls. A Scottish study revealed the highest concentration of arsenic was in the lungs when compared to the deposition in kidneys and the liver. Trivalent arsenic is metabolized in the liver and eliminated in the urine. Inorganic arsenic is quickly removed from the body of animals and humans. Almost all blood arsenic was cleared within 24 hours in human subjects injected subcutaneously with an arsenic salt. Toxicity from ingestion of inorganic arsenic include blood (anemia), cardiovascular (myocardial infarction and arterial thickening), CNS (peripheral neuropathy), dermal, GI (nausea, vomiting, diarrhea, and thirst), hepatic (cirrhosis of the liver), and renal (blood in the urine) effects. With chronic ingestion exposure, skin and possibly internal cancer may develop. With inhalation exposure, lung cancer is the principal effect. Skin problems without systemic effects are seen with dermal contact.

The two most toxic organic arsenic compounds are the methanearsonates (methyl derivatives of arsenic acid) and the phenylarsonates (phenyl derivatives of arsenic acid). Following oral administration in rodents, high acute concentrations of methanearsonates were detected in the GI tract, kidney, lung, and testes. Methanearsonates are excreted mainly in the urine. The phenylarsonates are not absorbed well from the GI tract of humans and animals after ingestion. Phenyl derivatives have been detected in the feces of humans and in the urine of animals. With ingestion, methanearsonates have been found to cause GI or skin disorder in animals, and phenylarsonates have proven to be neurotoxic in animals.

Epidemiologic studies have shown a link between inhalation exposure to inorganic arsenic compounds and an increase in the risk of lung cancer, especially in smelter workers and in those individuals residing near industries with arsenic emissions. Skin cancer and other indications of inorganic arsenic intoxication were observed with ingestion of water containing an average arsenic level of 0.4-0.6 mg/L in a large Taiwanese population. Arsenic has an USEPA WoE classification of A (human carcinogen).

4.3.2 Benzene (BZ)

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 benzole, 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.

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.

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 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 route of exposure influenced benzene metabolism. In humans and animals, benzene is

metabolized mainly by the liver's cytochrome P-450 system with toxicity believed due to the covalent binding of benzene metabolites (e.g. hydroquinone, phenol, and muconic dialdehyde) to cellular macromolecules. 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.

Toxic effects in humans from inhalation and ingestion exposures to benzene have resulted in death from respiratory arrest, CNS depression, and cardiac collapse. Inhalation exposures to humans have also resulted in hematological (deficit in the circulating blood cells, aplastic anemia, leukemia), immunological (changes in the blood levels of antibodies and circulating leukocytes), neurological (dizziness, tremor, delirium, unconsciousness), developmental (chromatid breaks, sister chromatid exchange in children of exposed females), and reproductive (impaired fertility, menstrual disorder, spontaneous abortion) effects, particularly in studies of occupationally-exposed groups. With human ingestion exposures, GI (gastritis, pyloric stenosis), hematological (decrease in erythrocytes and leukocytes), dermal (swelling and edema of skin), and neurological (vertigo, muscular incoordination, unconsciousness) effects have also been reported. Dermal exposures have resulted in skin irritation. In addition, hepatic (alteration of hepatic drug metabolism), immunological (decrease in peripheral blood leukocytes), and developmental (reduction in the weight of rodent pups) effects have also been noted in animals with ingestion exposure.

Epidemiological studies have shown an association between inhalation exposure to benzene and the development of leukemia (particular the acute myeloid form) and lymphopietic cancer in humans. Animal studies have supported the finding of leukemia with inhalation exposure and have also shown lymphomas with ingestion. Skin tumors have been demonstrated with dermal exposures. Genotoxic effects (chromosomal aberrations) in occupational groups have also been documented with inhalation and dermal exposures. Benzene has an USEPA WoE classification of A (human carcinogen).

4.3.3 Boron (B)

Boron (CAS No. 7440-42-8) is an element found naturally in sediment and sedimentary rock. The environmental discharge of boron occurs mainly from the natural weathering process. In addition, air, water, or soil may be contaminated with boron following discharge from coal-burning plants, copper smelters, and pesticides. Typical boron compounds are boric acid, borax, borate, and boron oxide. Boron's main use is in the manufacture of glass with other applications in fire retardant and in leather tanning and finishing industries. High exposure levels are found with workers employed in industries

utilizing boron-containing products, with persons residing near waste sites or areas with natural boron deposits, and with consumers utilizing cosmetics, medicines, or pesticides containing boron.

No studies were found on the interaction of boron with other substances.

The routes of exposure for boron include inhalation, ingestion, or dermal contact. No human or animal studies were found which dealt with absorption, distribution, metabolism, or excretion of boron by the three routes of exposure.

Toxic effects following inhalation of boron include irritation to the upper respiratory tract (cough; dry mouth, nose, throat; sore throat) and chronic eye irritation in occupational groups exposed to boron oxide and boric acid dust. No human studies were found for other systems of the body. With ingestion, a variety of toxic effects have been documented for infants who have received an accidental ingestion dose of boron. Infant deaths have been observed due to respiratory failure. Prior to death, manifestations of lethargy, vomiting, and diarrhea have been observed. Degenerative changes have been observed in the brain, kidney, and liver. Respiratory (congestion and hemorrhage of the lungs), gastrointestinal (nausea and vomiting, diarrhea, colic, abdominal pain), hepatic (jaundice, fatty changes in the liver), renal (degenerative changes in the cells), dermal (dermatitis), and neurological (headache, tremor, convulsion, coma) effects have also been observed in infants. In two adults, symptoms of vomiting occurred following ingestion of boric acid-containing fungicide and insecticide. No human studies were available which dealt with the effect of dermal exposure. In rabbits, conjunctivitis and dermatitis were seen with dermal and ocular exposures. No studies were found dealing with the development of cancer in animals or humans following boron exposure by inhalation, ingestion, or dermal contact. Boron has an USEPA WoE classification of D (not classifiable as to human carcinogenicity).

4.3.4 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. Synonyms include dichlorobromomethane, monobromodichloromethane, and methane, bromodichloro-. 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 levels of exposure to BDCM is 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 (diabetic) or who are exposed by inhalation and dermal contact in swimming pools will have potentially higher exposures to BDCM than others.

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.

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 process for BDCM in humans has not been established. 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.

No humans studies were available which examined the toxic health effects of BDCM with inhalation, ingestion, or dermal exposures. Toxic health effects were, however, observed following ingestion exposure in animals. Oral administration of BDCM ranging from 400-1000 mg/kg proved fatal to rodents with pathological effects to the adrenals, brain, kidney, liver, and lungs. In male rats, a decrease in the hemoglobin and hematocrit levels was seen following a single oral dose of BDCM which was less than 400 mg/kg of body weight. In animals, hepatic (degeneration of the liver, increase in liver enzymes), renal (focal necrosis or cell death), immunological (reduction in antibody forming cells), neurological (signs of CNS depression such as lethargy), and developmental (sternal anomalies in the fetus) effects were documented with oral exposures.

No human studies were available which documented the effect of BDCM exposure and the development of cancer with inhalation, ingestion, and dermal contact. 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. In oral studies of rodents, however, tumors of the large intestines, kidney, and liver have been observed. Genotoxic effects (sister chromatid exchange) have also been seen. The carcinogenic and genotoxic effects in animals suggests that BDCM exposure in chlorinated water may give rise to cancer in humans. BDCM has an USEPA WoE classification of B2 (probable human carcinogen).

4.3.5 Cadmium (Cd)

Cadmium (CAS# 7440-43-9) is a naturally occurring element found in the earth in concentrations of about 1-2 ppm. Cadmium is primarily used in the production of nickel-cadmium batteries and for

metal plating. It may be present in the air as a suspended solid, as a solid in soil, or may be dissolved in water if it is present as a chloride or sulfate.

The routes of exposure to cadmium include inhalation, ingestion and to limited extent dermal contact. Breathing air containing small cadmium particles may result in deposition of cadmium particles in the lung. Smoking cigarettes may also expose you to cadmium. Exposure to cadmium may also result from ingestion of food or water containing cadmium. Very little is absorbed through the skin unless the skin is scraped or cut.

Cadmium is readily absorbed by the lung. Up to 50% of inhaled cadmium particles less than .1 micron in size will be deposited in the lungs. Between 50% and 100% of the cadmium will ultimately be absorbed into the body. Cadmium inhaled through cigarette smoking is very efficient at being deposited into the lung and absorbed into the blood. Most ingested cadmium passes through the gastrointestinal track without being absorbed. Most cadmium that is inhaled or ingested is eventually excreted in the feces. Most of this excreted material represents cadmium that was not absorbed by the gastrointestinal tract. Cadmium that is absorbed is excreted very slowly, with excretion in the urine and feces being nearly equal.

Health effects from short term inhalation of large quantities of cadmium include irritation of the nose and throat, chest pain, headache, chills, muscle aches, nausea, vomiting and diarrhea. Inhalation of 5 mg/m³ for 8 hours may result in death. Longer term inhalation may result irreversible lung injury, open sores in the nose and loss of sense of smell. Both inhalation and ingestion of cadmium over a long period of time may result in liver and kidney damage.

There is some evidence that cadmium is a carcinogen in humans when inhaled, however, only one study has shown an increase in lung cancer associated with cumulative exposure. There is some evidence that inhalation of cadmium may result in prostate cancer. There is no evidence that cadmium is carcinogenic when ingested. Cadmium has a USEPA WoE classification for inhalation exposure of B1, (probable human carcinogen).

4.3.6 Carbon Tetrachloride (CCl₄)

Carbon tetrachloride (CAS# 56-23-5, CCl₄) is a man made chemical which is used primarily in the production of chlorofluorocarbons. In the past, carbon tetrachloride was widely used in industry, medicine and in the home. Synonyms of carbon tetrachloride include tetrachloroethane and perchloromethane. Carbon tetrachloride is very stable once released into the environment and is relatively non reactive.

The routes of exposure to carbon tetrachloride include inhalation of vapor, oral ingestion, and dermal and ocular contact. Most overexposures result from use of carbon tetrachloride as a cleaning fluid.

Carbon tetrachloride is well absorbed by both inhalation and ingestion, with about 60% of the dose absorbed when inhaled and 80% absorbed when ingested. It is also absorbed through the skin, through less readily than from the lung. Once absorbed, it is rapidly distributed by the blood where it concentrates in fat and organs. Carbon tetrachloride is metabolized in the liver by the P-450 cytochrome system. Between 40% and 70% is excreted unchanged in expired air. The remainder is excreted in the urine and feces or is metabolized and excreted as CO₂ or other metabolites.

The primary non carcinogenic health effects from exposure to carbon tetrachloride are central nervous system depression and liver and kidney damage. Evidence of liver damage includes jaundice, swollen liver, and biochemical alterations of the blood. Damage to the kidney and liver is often delayed after exposure. Ingestion or inhalation of carbon tetrachloride may result in death as a result of liver or kidney damage. Fatal doses are in the range of 40-320 mg/kg. If death can be averted, liver and kidney function usually recover within 1 to 2 weeks, and recovery generally appears to be complete.

There is some evidence that indicates carbon tetrachloride as a carcinogen in humans, however, the evidence is not conclusive. Animal studies indicate that it causes liver cancer in laboratory animals. Carbon tetrachloride has a USEPA WoE classification of B2 (probable human carcinogen).

4.3.7 Chlorobenzene (MCB)

Chlorobenzene (CAS No. 108-90-7, C₆H₅Cl) is a chlorinated benzene. Synonyms include monochlorobenzene, benzene chloride, phenylchloride, and chlorobenzol. MCB is used as a solvent, chemical intermediate, and degreaser. Chlorobenzene concentrations are high in certain occupational groups and in industrial areas with improper control of emissions.

The interactive effect of cyclohexane oxide and BDCM has been documented with reports of a reduction in the metabolism of chlorobenzene and thus, its liver toxicity.

Routes of exposure include inhalation, ingestion, or dermal contact. With inhalation exposure, two workers were found to have absorbed 38% and 45% of the chlorobenzene dose which was administered. With ingestion, 31% of an oral dose was absorbed from the GI tract in a single human subject while 18% of the ingested dose was absorbed in an animal study. No research was found on absorption with dermal exposure. No human or animal studies were found dealing with the distribution of chlorobenzene following ingestion and dermal contact. Although human studies were unavailable,

animal studies demonstrated that adipose tissue was the most likely site for distribution of chlorobenzene with inhalation exposure. By oral or inhalation exposure, chlorobenzene was metabolized into 4-chlorocatechol and p-chlorophenylmercapturic acid and excreted in the urine in three human subjects. Animal studies have also revealed excretion through the kidneys.

Human and animal inhalation studies have shown toxic effects to the CNS, liver, and kidneys. No human studies were found examining the development of cancer with exposure to chlorobenzene. Although an increased incidence of neoplastic nodules of the liver with ingestion exposure was seen in animals, no clear evidence exists presently to show that MCB causes cancer. Chlorobenzene has an USEPA WoE classification of D (not classifiable as to human carcinogenicity).

4.3.8 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 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 CO₂. 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.

Toxic health effects have been documented for human inhalation and ingestion exposures. Toxic levels of chloroform have proven fatal in inhalation and ingestion exposures with death resulting from damage to the liver. With inhalation exposures, cardiovascular (bradycardia, arrhythmia, heart block), gastrointestinal (nausea and vomiting), hepatic (necrosis, jaundice), neurological (dizziness, headache, convulsions, hallucinations and delusions), renal (fatty degeneration), and respiratory (depression) effects have been observed. With ingestion exposure, cardiovascular (EKG changes), gastrointestinal (gastric distress, vomiting), hematological (decrease in erythrocytes and hemoglobin), hepatic (liver enlargement, fatty degeneration, necrosis), musculoskeletal (muscular relaxation), neurological (coma), respiratory (congestion of lungs), and renal (fatty degeneration) effects have been documented. Dermal exposures have resulted in the destruction of the stratum corneum, one of the layers of the skin.

A number of epidemiologic studies have examined the association between cancer and the consumption of chlorinated water. Cancer of the large intestines, rectum, and/or bladder have been observed in these studies. Since many potential carcinogens have been identified in chlorinated water, difficulties have arisen in identifying the cancer-causing agent. Kidney and liver cancers and lymphosarcoma have been detected in rodents with ingestion exposure. Genotoxic effects have also been reported with inhalation and ingestion exposures in mice. Chloroform has an EPA Weight-of-Evidence Classification of B2 (probable human carcinogen).

4.3.9 Chloromethane (CM)

Chloromethane (CAS #74-87-3, CH₃Cl) is a colorless gas which is produced in large amounts in the ocean and during microbial decomposition of plants and wood. It is also produced industrially and

has been used as a refrigerant. When present in water, chloromethane evaporates rapidly. Chloromethane may also be referred to as methyl chloride.

The routes of exposure to chloromethane include inhalation and ingestion. Breathing air containing chloromethane is the most common exposure route. Ingesting chloromethane is possible if it is present in drinking water.

Chloromethane is rapidly and efficiently absorbed following inhalation or ingestion. Following absorption, chloromethane is distributed rapidly by the blood and deposited in various tissues. Chloromethane is then metabolized and excreted primarily as metabolites. Very little unmetabolized chloromethane is excreted in the urine or feces.

The central nervous system is the major site of toxicity from exposure to chloromethane. Typical CNS depression symptoms such as dizziness, blurred vision, muscle incoordination and coma result from high exposures. The liver and kidneys may be damaged following exposure. Death has resulted from overexposure, however, concentrations necessary to cause death would usually occur only in industrial settings with little ventilation.

The evidence that chloromethane is a carcinogen is limited to one animal study in which only one sex of one species developed a statistically significant increase in tumors. There is no evidence to suggest that chloromethane is a carcinogen in humans. Chloromethane has a USEPA WoE classification of C (possible human carcinogen).

4.3.10 Chromium III, IV, Total (CrIII, CrIV, Tot)

Chromium is a naturally occurring element found in rocks, soil, animals and plants and is found in different forms or ions. Chromium (0) (CAS# 7440-47-3) is a steel gray solid used in making steel and other alloys and does not occur naturally. Chromium (III) and chromium (VI) (CAS nos. 16065-83-1 and 18540-29-9 respectively) are ions used for chrome plating and in the manufacture of dyes and pigments.

Chromium (III) may be oxidized to chromium (VI) in the presence of oxidizable organic substances, oxygen, manganese dioxide and moisture. Under anaerobic conditions, chromium (VI) is reduced to chromium (III) in the presence of S^{2-} and Fe^{+2} .

The routes of exposure to chromium include inhalation, ingestion and dermal contact. Breathing air containing chromium can result in deposition of chromium in the lungs or ingestion of the chromium as the body removes it from the lungs. Exposure to chromium may also result from incidental ingestion of dirt containing chromium or from eating foods or drinking water containing chromium. Exposure may

also occur as a result of dermal contact with chromium, although little will be absorbed into the body unless the skin is scraped or cut.

Chromium (VI) is more readily absorbed by the body than chromium (III). Studies indicate that between 53% and 85% of inhaled chromium (VI) is absorbed by the lungs into the bloodstream or cleared by the pharynx and ingested. The remainder of the chromate remains in the lungs. Approximately .5% to 2% of ingested chromium is absorbed by the gastrointestinal tract. When ingested, chromium (VI) compounds are converted to chromium (III) in the stomach. Both chromium (III) and (VI) can penetrate the skin to some extent if the chromium is in an acidic solution or if applied as a salve. Chromium compounds may also penetrate the skin if the skin is scraped or cut.

In general, chromium (VI) compounds are more toxic than chromium (III) compounds. Health effects due to inhalation are the most significant of the exposure routes. Noncancer health effects from inhalation include nasal septum damage, irritating respiratory effects, liver and kidney effects and increased risk of death from noncancer respiratory effects. Dermal exposure to chromium compounds may result in allergic dermatitis and formation of skin ulcers known as chrome holes.

Human epidemiological studies clearly indicate increased risk of lung cancer in chromate (chromium VI) production workers and in some pigment and chrome plating workers. Based upon epidemiological evidence, chromium (VI) is considered carcinogenic in humans when inhaled. Chromium (III) and (0) are not considered to be carcinogenic and have USEPA WoE classifications of D (not classifiable as to carcinogenicity). Chromium (VI) has a USEPA WoE classification of A for inhalation exposure (human carcinogen). Chromium (VI) has a WoE classification of D for ingestion exposure.

4.3.11 Cyanide, free (CN)

Cyanide (CAS# 57-12-5, CN⁻) is the ionic form of a group of compounds known as cyanides. Cyanide is often found combined with hydrogen, potassium or sodium to form compounds with differing properties and toxicities. Hydrogen cyanide is used in the production of nylon, as an insecticide, and in the production of chemicals and pharmaceuticals. Cyanide salts are used in electroplating and metal treatment.

The routes of exposure to cyanides include inhalation, ingestion and to a lesser extent, dermal contact. Since hydrogen cyanide is a gas, its most important exposure route is inhalation. Oral exposure to cyanide results from ingestion of cyanide salts such as sodium and potassium cyanide. Exposure to cyanide by dermal contact usually occurs only in an industrial setting.

Hydrogen cyanide is rapidly absorbed into the body following inhalation. Absorption of cyanide salts following ingestion varies but is generally about 50%. After cyanides are absorbed, they are rapidly distributed by the blood throughout the body. Hydrogen cyanide is acutely toxic, acting as a cellular inhibitor of respiration. Cyanide is metabolized in the liver into a number of less harmful products and excreted in the urine, usually within 24 hours of exposure.

Health effects from lower exposures to hydrogen cyanide include headaches, confusion, nausea, vomiting and slow gasping respiration. Hydrogen cyanide may rapidly cause death if present in air in excess of 200 ppm. Oral exposure to cyanide salts may also result in death if consumed in excess of .56 mg/kg. Health effects from oral exposure include symptoms similar to those described for inhalation.

No evidence is available indicating that cyanide compounds are carcinogenic. Cyanide has a USEPA WoE classification of D (not classifiable as to human carcinogenicity).

4.3.12 Dibromochloromethane (DBCM)

Dibromochloromethane (CAS# 124-48-1, CHBr_2Cl) is a liquid halogenated hydrocarbon (trihalomethane) which is used as a chemical intermediate in the manufacture of refrigerants, pesticides, aerosol propellants and fire extinguishing agents. It is sometimes present in drinking water, usually resulting from reactions that occur during water chlorination. Dibromochloromethane is quite stable in the environment due to its resistance to degradation. DBCM may also be known as chlorodibromomethane.

Routes of exposure include inhalation of vapors, ingestion and dermal contact. Very little is known about the absorption and toxicokinetics of dibromochloromethane, but by comparison with other similar chemicals, it is likely that it is well absorbed both through inhalation and ingestion. About 50% of absorbed DBCM is likely to be metabolized, with the remainder being excreted unchanged in expired breath.

Very little is known about its toxic health effects, however, DBCM is an irritant and narcotic. Effects of the central nervous system include dizziness, and headache. In doses of from 25 to 100 mg/kg/day, DBCM may cause liver and kidney damage. When DBCM comes into direct contact with the skin, it can cause severe irritation and burning.

It is unknown whether or not DBCM causes cancer in humans, however, there is evidence that it causes cancer in laboratory animals. DBCM has a USEPA WoE classification of C (possible human carcinogen).

4.3.13 1,2-Dichlorobenzene (DCB2)

1,2-Dichlorobenzene (CAS No. 95-50-1, $C_6H_4Cl_2$) is a chlorinated benzene which exists in the liquid form. Synonyms include benzene, 1,2-dichloro-; o-dichlorobenzene; dichlorobenzene, ortho; dichlorobenzene, ortho; and orthodichlorobenzene. DCB is used as a solvent in the production of toluene diisocyanate and as a chemical intermediate in the manufacture of dyestuffs, herbicides, and degreasers. DCB is a by-product in the manufacture of monochlorobenzene. High risk groups are individuals with preexisting liver, kidney, and CNS illness; those on drugs (hormones); occupations with DCB exposure; or domestic users of the product.

DCB is incompatible with strong oxidizers, hot aluminum, or aluminum alloys.

The routes of exposure for DCB include inhalation, ingestion, or dermal contact. Human or mammal metabolism of DCB results in the formation of dichlorophenols, some of which are considered toxic.

DCB causes eye and nose irritation, damage to the kidney and liver, and skin blistering. High doses cause CNS depression. Animal studies have shown renal tubular changes and liver necrosis with ingestion exposure, and decreased weight of the body and spleen and a gain in the weight of the liver with inhalation exposure. Other studies of animals have demonstrated an increased incidence of malignant lymphomas and respiratory cancers with ingestion exposure. An increased incidence of chromosomal alterations in the peripheral blood cells has been observed in workers with DCB inhalation exposure. DCB has an EPA Weight-of-Evidence Classification of D (not classifiable as to human carcinogenicity).

4.3.14 1,4-Dichlorobenzene (DCB4)

1,4-Dichlorobenzene (CAS No. 106-46-7, $C_6H_4Cl_2$) is a chlorinated benzene which is a by-product in the production of monochlorobenzene. Synonyms include benzene, 1,4-dichloro; benzene, p-dichloro; p-dichlorobenzene; p-dichlorobenzol; paradichlorobenzene; and paradichlorobenzol. The primary uses include its application as a room deodorizer, moth repellent, and intermediate in the manufacture of polyphenylene sulfide resins. Although 1,4-dichlorobenzene is a solid at room temperature, it vaporizes in air and this vapor acts as a room deodorizer or insect repellent. Populations at risk include certain occupational groups employed in the industrial setting, individuals residing near industrial sites emitting 1,4-dichlorobenzene, and consumers using products which give off these vapors.

No data was available regarding the interaction of 1,4-dichlorobenzene with other substances.

Exposure occurs by inhalation, ingestion of contaminated food or water, or dermal contact. Inhalation is the most likely form of exposure. Absorption of 1,4-dichlorobenzene is presumed to occur

by inhalation, ingestion and dermal contact. No quantitative studies were found; however, the ingestion absorption is considered 100% and by inhalation 30%. Following inhalation exposure, 1,4-dichlorobenzene has been found in human blood, fatty tissue, and breast milk with animal distribution in fatty tissue, kidneys, and liver. No ingestion data was found for humans, but animal data have demonstrated high concentrations of 1,4-dichlorobenzene in the fatty tissue, kidney, and liver following ingestion. The main metabolites of 1,4-dichlorobenzene with inhalation and ingestion exposures are the dichlorophenols which are excreted in the urine of humans. Animal experiments indicate excretion by exhalation and in the urine following inhalation, and in the urine and feces with ingestion exposure.

The organ systems which are affected by 1,4-dichlorobenzene exposure are the CNS and liver in humans on inhalation and the liver and kidneys in animals on ingestion. Human case studies have demonstrated CNS (dizziness, weakness, slurred speech) and hepatic (atrophy and cirrhosis) effects upon inhalation exposure. In animal studies, hepatic (degeneration) effects have been observed with inhalation and ingestion exposures and renal effects seen with ingestion exposure. Some evidence in animals links developmental toxicity with inhalation and ingestion exposures.

No studies were available examining the carcinogenicity of 1,4-dichlorobenzene in humans. Animal studies have shown an increased incidence of adrenal, kidney, and liver cancers in animals with ingestion exposure. 1,4-Dichlorobenzene has an EPA Weight-of-Evidence Classification of C (possible human carcinogen).

4.3.15 1,1-Dichloroethane(DCA)

1,1-Dichloroethane (CAS No. 75-34-3, $C_2H_4Cl_2$) is a halogenated hydrocarbon made by man. Synonyms include alpha alpha-dichloroethane; asymmetrical dichloroethane; chlorinated hydrochloric ether; ethane, 1,1-dichloro- (9CI); and ethylidene chloride. 1,1-Dichloroethane is used as a chemical intermediate (for producing 1,1-trichloroethane and vinyl chloride), solvent, finish remover, and degreaser. Environmental emissions arise from the industrial production and use of 1,1-dichloroethane and are mainly discharged to the atmosphere. People employed in the chemical and allied products industry or individuals residing near industrial or waste sites have the highest potential exposure to 1,1-dichloroethane. In the past, 1,1-dichloroethane was used as an anesthetic.

No data was available dealing with toxic interactions between 1,1-dichloroethane and other chemicals. However, data suggest that 1,1-dichloroethane detoxification is carried out by glutathione. Since chlorinated hydrocarbons, acetaminophen, and bromobenzene reduce the body's glutathione, an

increase in the toxicity of 1,1-dichloroethane would be expected in the presence of these substances. In addition, in-vitro metabolism of 1,1-dichloroethane is enhanced by ethanol.

The routes of exposure include inhalation, ingestion, and less likely dermal contact. No human inhalation, ingestion, or dermal exposure studies were available which dealt with absorption of 1,1-dichloroethane. However, investigations following inhalation exposure to 1,1-dichloroethane as an anesthetic would indicate that absorption does occur. Animal evidence exists which is supportive of absorption by ingestion and dermal contact. No studies were found dealing with distribution in the body following inhalation, ingestion, or dermal exposure in humans or animals. It is assumed that distribution occurs to the CNS from reports of patients anesthetized with 1,1-dichloroethane. Metabolism, though not studied extensively, occurs in the liver with the cytochrome P-450 system as documented in mice and rats following ingestion exposure. One human study examined excretion following inhalation exposure to 1,1-dichloroethane and demonstrated that 59% of the 1,1-dichloroethane was metabolized and eliminated in the urine while the remaining 41% was eliminated by exhalation.

Cardiac stimulation and arrhythmias were observed in humans when 1,1-dichloroethane was given by inhalation as an anesthetic. This effect prompted its discontinuance as an anesthetic. CNS depression has also been reported in humans anesthetized with 1,1-dichloroethane. Although not detected in humans, animal studies demonstrated renal injury and retarded fetal development with inhalation exposure. Two animal studies were found dealing with toxic health effects to 1,1-dichloroethane with ingestion. Although body weight depression was observed, the data were inconclusive. No studies were found for health effects from dermal exposure.

In addition, no human studies were available which examined the carcinogenic effect of 1,1-dichloroethane following inhalation or dermal contact. Animal ingestion studies have given evidence that 1,1-dichloroethane was carcinogenic. Hemangiosarcomas, mammary and liver cancers, and uterine polyps have been reported in rodents with ingestion exposure. The toxic and carcinogenic effects are considered to be caused by free radicals formed under hypoxic conditions from 1,1-dichloroethane. 1,1-Dichloroethane has an EPA Weight-of-Evidence Classification of C (possible human carcinogen).

4.3.16 1,2-Dichloroethane (DCA2)

1,2-Dichloroethane (CAS No. 107-06-2, $C_2H_4Cl_2$) is a halogenated hydrocarbon which is man made and an intermediate chemical utilized in the manufacture of vinyl chloride, 1,1,1-trichloroethane, trichloroethylene, and tetrachloroethylene. It is also used as an additive for leaded gasoline. Synonyms include 1,2-dichloroethane; 1,2-ethylene dichloride; alpha, beta-dichloroethane; dichloro-1,2-ethane;

dichloroethylene; ethane 1,2-dichloride; and ethylene chloride. Environmental emissions occur primarily into the atmosphere from industry. 1,2-Dichloroethane evaporates rapidly from surface water and spills to soil surfaces. Dependent on the organic content of the soil, 1,2-dichloroethane may be transported into the groundwater. Human exposure occurs in certain occupations and from residing in industrial areas or close to chemical waste sites with high emissions of 1,2-dichloroethane.

A number of interactions between 1,2-dichloroethane and other chemicals have been documented in animal studies. Administration of phenobarbital, 3-methylcholanthrene, and/or ethanol (low concentrations) resulted in increased liver enzymatic action (cytochrome P-450) which hastened the formation of toxic metabolites of 1,2-dichloroethane. Toxicity to the liver was enhanced when 1,2-dichloroethane was given by inhalation with oral sulfiram. Other studies have demonstrated that the administration of glutathione, precursors of glutathione, or amino acids reduced the toxic effects and mortality from oral exposure to 1,2-dichloroethane. Disulfiram by ingestion and 1,2-dichloroethane administered by inhalation enhanced liver toxicity beyond the level of exposure with 1,2-dichloroethane alone. High concentrations of ethanol reduced toxicity of 1,2-dichloroethane.

Although exposure may result from ingestion of contaminated food and water or by dermal contact, the most common mode of exposure is the inhalation of contaminated air. Animal studies have demonstrated that absorption occurs following inhalation, ingestion, or dermal exposure. No human studies involving metabolism of 1,2-dichloroethane were found; however, animal studies indicate that 1,2-dichloroethane is easily metabolized producing urinary metabolites resulting from inhalation and ingestion exposure. With inhalation exposure, 1,2-dichloroethane has been distributed in human breath and breast milk while ingestion exposure has resulted in the distribution of 1,2-dichloroethane in the blood, liver, and lungs. In addition, 1,2-dichloroethane has been detected in human breast milk with dermal exposure. Animal studies have reported removal of 1,2-dichloroethane from the body through exhalation and by urinary output following inhalation or oral exposure. In women who had inhaled 1,2-dichloroethane in the workplace, the substance was exhaled in the unchanged form.

A number of toxic effects has been observed in humans with inhalation and ingestion exposures to 1,2-dichloroethane. With acute inhalation and ingestion exposures, CNS (depression), GI (nausea, vomiting), hepatic (necrosis), renal (necrosis), and respiratory tract (pulmonary edema) effects have been observed with deaths attributed to cardiac arrest and arrhythmia (irregular heart rate). Following death in animals and humans, pathological changes on autopsy have been observed in the brain, heart, kidneys, liver, and lungs. Ocular effects have been seen in humans with inhalation exposures, and a decrease in blood clotting was observed with ingestion exposure.

Specific epidemiologic studies of exposure to 1,2-dichloroethane and incidence of cancer have not been carried out. Human studies have shown an increased rate of cancers with inhalation and ingestion exposures, but the presence of multiple chemicals has prevented isolation of a single causative agent as 1,2-dichloroethane. In animal studies, 1,2-dichloroethane has been reported to be carcinogenic with oral exposure but not with inhalation and dermal exposures; however, nonmalignant tumors were seen in animals with dermal exposures. A statistically significant rise in multiple tumor types was seen with exposure by ingestion. The tumor types included circulatory system, endometrial, liver, mammary and stomach cancers; fibromas of the subcutaneous tissue; and lung adenomas in rodents. 1,2-Dichloroethane has an EPA Weight-of-Evidence Classification of B2 (probable human carcinogen).

4.3.17 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, 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 CO₂ (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).

4.3.18 1,2-Dichloroethylene (DCE2)

1,2-Dichloroethylene (CAS No. 540-59-0, C₂H₂Cl₂) is a halogenated hydrocarbon which is an intermediate chemical in the manufacture of chlorinated solvents and compounds. Synonyms include 1,2-dichloroethene, acetylene dichloride, and sym-1,2-dichloroethylene. The total 1,2-dichloroethylene consists of the two isomers: trans-1,2-dichloroethylene and cis-1,2-dichloroethylene. High levels of exposure occur in certain occupations and from residing near chemical waste sites with emissions of 1,2-dichloroethylene. Environmental exposure occurs as a result of industrial emissions from the production and use of 1,2-dichloroethylene; wastewater, landfill, and solvent vaporization; breakdown of polyvinyl chloride and vinyl copolymers; and leaching from chemical landfills.

No studies were found which dealt with the interaction of 1,2-dichloroethylene with other chemicals.

Since 1,2-dichloroethylene is found in air, soil, and water, exposure occurs through inhalation, ingestion, and dermal contact. The most likely form of exposure is by inhalation. Absorption of both isomers occurs through the lungs following inhalation exposure. Rat studies have demonstrated gastrointestinal absorption after oral administration. No research was available on dermal absorption, or on the distribution of 1,2-dichloroethylene. Metabolism of 1,2-dichloroethylene commences with the

liver's cytochrome P-450 system, but little work has been done in the metabolic process outside of the liver. No studies were found dealing with the excretion following inhalation, ingestion, or dermal exposure.

Human toxicity data were also sparse. Some information was available on acute exposures, but the effects were not well documented. One man died following inhalation exposure but the conditions producing this effect were not reported. High oral doses of the two isomers have produced death in rats and mice. In humans, inhalation of trans-1,2-dichloroethylene can cause neurological effects (nausea, lethargy, fatigue) and burning of the eyes. In animals, respiratory (pulmonary edema), cardiovascular (swelling of myocardium), hematological (decrease in circulating RBC and WBC), hepatic (degeneration), and suggestive immunological effects have been reported with trans-1,2-dichloroethylene exposure by inhalation. Exposure to the two isomers have resulted in neurological effects (behavioral changes) by inhalation in rodents. No human ingestion studies were available. In animal studies, GI (hyperemia of stomach and small intestines), hepatic (fatty degeneration), immune (suppression of humoral immune system), and respiratory (pulmonary capillary hyperemia) effects were observed with ingestion exposure with trans-1,2-dichloroethylene; hematological (decrease RBC and hematocrit) and renal (increase in kidney weight with decrease in blood urea nitrogen) effects with cis-1,2-dichloroethylene; and CNS depression with exposure to the two isomers. The long-term effects, including cancer, have not been documented. No epidemiologic studies dealing with 1,2-dichloroethylene were found. 1,2-dichloroethylene has an EPA Weight-of-Evidence Classification of D (not classifiable as to human carcinogenicity).

4.3.19 Dichloromethane (DCM)

Dichloromethane (CAS# 75-09-2, CH_2Cl_2) is a man made chemical that is widely used in industry as a paint stripper, as a propellant in aerosol sprays, and in the photographic and electronics industry. Dichloromethane is commonly referred to as methylene chloride.

The routes of exposure to dichloromethane include inhalation, ingestion and dermal contact. Since dichloromethane evaporates readily, the most important exposure route is by inhalation of vapors. The highest human exposures to dichloromethane usually occur in the industrial workplace.

Since dichloromethane is usually present as a vapor, the primary route of exposure is by inhalation. Approximately 70% - 75% of inhaled and ingested dichloromethane is absorbed. After absorption it is rapidly distributed by the blood to adipose tissue and body organs. Dichloromethane is then metabolized along two pathways which produce either CO or CO_2 , with CO being the major

product. The CO produced during metabolism then forms carboxyhemoglobin to produce symptoms of carbon monoxide poisoning.

The primary non-cancer health effects involve the central nervous system, but also involve the kidney and liver following long term exposure. Acute CNS effects include loss of muscle control, stupor, dizziness, chest pain, unconsciousness and death. Acute symptoms occur following inhalation exposure of 300 - 700 ppm for 3 to 5 hours.

Human epidemiological studies have not shown a causal relationship between occupational exposure to dichloromethane and cancer. Animal studies have demonstrated that it is carcinogenic in laboratory animals. Dichloromethane has a USEPA WoE classification of B2 (probable human carcinogen).

4.3.20 t-1,3-Dichloropropene (tDCP3)

T-1,3-Dichloropropene (CAS#10061-02-6, $C_3H_4Cl_2$) is an isomer of a man made liquid used in agriculture as a soil fumigant for parasitic nematodes. All commercial DCP3 consists of both the cis and the trans isomers. The production and use of this chemical has increased recently due to a ban on the production and use of ethylene dibromide. Synonyms for DCP3 include alpha-chlorallyl chloride and Telone.

Exposure to DCP3 usually occurs to those manufacturing the chemical or those using it in agricultural applications. Routes of exposure include inhalation, ingestion and dermal contact. DCP3 is well absorbed by the body following inhalation and ingestion and is believed to be well absorbed dermally. Once absorbed, DCP3 is rapidly distributed by the blood where it or its metabolites concentrate in the body organs. DCP3 appears to be converted primarily to the glutathione conjugate in the liver. Almost all of the absorbed DCP3 is eventually eliminated in the urine as metabolites.

Non carcinogenic health effects from exposure to large amounts of DCP3 include headache, mucous membrane irritation, dizziness, nausea, and vomiting. No deaths have been reported following exposure to DCP3. Direct contact with skin or eyes causes severe burning, resulting in permanent damage.

Evidence that DCP3 is carcinogenic in humans is limited. A few clinical reports have indicated that it may cause cancer in humans. Several animal studies have indicated that DCP3 caused various tumors in laboratory animals. 1,3 dichloropropene has an USEPA WoE classification of B2 (probable human carcinogen).

4.3.21 Fluoride (F)

Fluorine (CAS No. 7782-41-4, F_2) is an element in the gaseous state which combines with other substances to form fluoride salts (e.g. sodium fluoride). Synonyms include fluoride ion; fluorine ion; hydrofluoric acid, ion (1-); and perfluoride. Fluoride compounds are used mainly in the steel industry and secondarily in the chemical and glass industries. The element, fluorine, has been employed as an oxidizer in rocket fuels and in the manufacture of metallics, fluorides, glass, enamel, and brick. Sodium fluoride has been added to drinking water as a preventive for tooth decay with other applications in insecticides and as a disinfectant in breweries. Calcium fluoride has been utilized in the manufacture of steel, frosting glass and enamels and as a coating on welding rods. Hydrogen fluoride has been used in the manufacture of aluminum and chlorofluorocarbons. Environmental discharge of fluorides occurs near industrial sites and naturally through the erosion of rocks and minerals. Individuals working in or residing near processing plants experience high exposure levels to fluorides. The general public receives exposure in the drinking water, foods, and products for the teeth.

Fluoride interacts with a number of substances which influence its absorption from the gastrointestinal tract. When calcium and/or phosphorus in the form of bone meal, cryolite, or calcium fluoride were given orally to humans, a reduction in the absorption of fluoride was seen. In the treatment of osteoporosis, fluoride's unfavorable effects seemed to be diminished in the presence of magnesium administered orally. By forming fluoride complexes, aluminum hydroxide used as an antacid also decreased the gastrointestinal absorption of fluoride.

The routes of exposure for fluoride include ingestion, inhalation, or dermal contact. Human fluoride absorption occurs following inhalation and ingestion exposures and probably by dermal contact. Whatever the route of exposure in humans, fluoride has been found in the plasma and distributed in bone and teeth. As an anion, fluoride is not metabolized as other compounds. Although the mechanism is not fully understood, fluoride does interact with other elements and with enzymes within the human body. With inhalation and ingestion exposures, fluoride is excreted in the urine of humans. No information was available for excretion of fluoride with dermal exposure.

Toxic effects from fluoride have been examined for various systems of the human body. With inhalation exposure, a number of outcomes have been observed. The lethal effect of hydrofluoric acid has been documented in the occupational setting. Death has generally resulted from pulmonary edema and cardiac arrhythmia. Respiratory effects such as the reduction in pulmonary function of workers have also been observed. Other consequences of human inhalation exposure include gastrointestinal (nausea), musculoskeletal (mottled teeth, hard and brittle bones, increased bone opacity, skeletal fluorosis) and

cular (conjunctival irritation) effects. Following accidental or intentional ingestion of sodium fluoride in humans, death has resulted in findings of pulmonary edema and cerebral edema on autopsy. Other consequences of human ingestion exposure include cardiovascular (cardiac arrhythmia), gastrointestinal (nausea and vomiting, gastric pain, diarrhea), musculoskeletal (mottled teeth, skeletal fluorosis), renal (renal insufficiency), and neurological (paresthesia, paresis, convulsions) effects. With human dermal exposure, lethal outcomes have been observed in the occupational setting with pulmonary edema and cardiac arrhythmia documented as the cause of death. In addition, respiratory events such as pulmonary hemorrhagic edema and tracheobronchitis have also been seen as well as skin burns, opacity of the corneal epithelium, and conjunctival thrombosis.

Epidemiologic studies have examined the effect of fluoride exposure on the development of cancer. The majority of occupational exposures to fluoride occur from inhalation of hydrofluoric acid fumes or of dust from cryolite or fluorospar. In one cohort mortality study of an occupational group, an increased rate of prostatic cancer was seen in workers who had probable inhalation exposure to fluorides and insecticides. An increased mortality was also observed for respiratory cancer in cryolite workers. Since workers generally have simultaneous exposure to more than one chemical substance, identification of the causative agent in these studies becomes a major problem. Fluoride has an USEPA WoE classification of D (not classifiable as to human carcinogenicity).

4.3.22 Lead (Pb)

Lead (CAS No. 7439-92-1) is an element found throughout the environment in the earth's crust and from processes initiated by man. Synonyms include lead metal, plumbum, and pigment metal. Lead is found in air, food, water, and dust. Its primary use is in the production of storage batteries with additional applications which include the manufacture of paint, gasoline additives, metal products (sheet lead, solder), and ammunition. The highest airborne concentrations of lead have been from vehicle emissions during the period when gasoline with lead additive was widely used. Other airborne sources include industrial emissions (smelting operations and the production of lead batteries), natural emissions (active volcano), and cigarette smoking. The primary source of lead in water is from plumbing and solder and lead-containing dust, soil, and wastewater. Food and beverages may also contain lead if crops or the food operations are contaminated with lead-filled dust. Workers are mainly exposed through inhalation in jobs involving smelting, production of steel and batteries, gasoline stations, and auto repair.

Lead interacts with a number of substances as demonstrated in human and animal studies. For example, absorption of lead in the body was lower in subjects given oral calcium and phosphorus

supplements. An inverse relationship was also seen between dietary iron, vitamin D, and zinc and lead. With high lead levels in the body, the concentrations of these three substances were low. In fact, iron deficiency resulted in a two to threefold greater absorption rate of lead in study subjects when compared to those individuals who were not deficient. In animals, similar conditions were observed. For example, the administration of iron orally or by injection seemed to lessen the effect of lead on body enzyme activity in one animal study. When lead was administered to rats, mercury deposition increased in the rat kidneys. Animal studies have shown that the combined activity of cadmium and lead manifested itself in rats with weight loss and an increase in the weight of body organs (brain, liver, and adrenal glands). Rats exposed to lead and ethanol demonstrated a greater inclination toward the neurological and hepatic effects of lead. Phenylhydrazine and lead combined intensified the effect on the different phases of anemia in a rat experiment.

The routes of exposure for lead include inhalation, ingestion, or dermal contact. If deposition of lead particles occurs in the lower respiratory tract, the particle absorption is almost total. Fifty percent of the lead which is ingested by children is absorbed by the body with an 8% and 15% rate of absorption in two separate studies examining ingestion exposure in adults. Fasting has been shown to enhance ingestion absorption to 45% in adults. In animals, the absorption of alkyl lead (tetraethyl lead) occurred more rapidly by dermal application in rabbits than by ingestion. Since man's dermal absorption rate is lower, absorption in humans by dermal contact is less than by inhalation or ingestion. Inorganic lead is not metabolized or biotransformed; however, metabolism does occur in the liver with organic (alkyl) lead. Regardless of the route of absorption, lead is distributed in the blood, soft tissue, and bone with the majority of the total body burden in the bone. For the lead which is not absorbed, excretion in humans occurs through the urine and feces. Transplacental transfer has also been observed in humans.

A variety of toxic effects have been documented in humans from inhalation and ingestion exposures to lead. Severity of symptoms is dose dependent with higher doses of lead producing more severe symptoms. Impairment of heme (iron) synthesis with resultant anemia has been seen. Neurobehavioral toxicity has been documented in occupational groups mainly from inhalation but also from ingestion. Lead encephalopathy is the most serious neurobehavioral effect with symptoms of dullness, irritability, poor attention span, headache, muscular tremor, memory loss, and hallucinations. If the exposure concentration is high enough, the condition becomes quite severe with coma and death resulting. Acute encephalopathy and death have been documented in children with mainly ingestion and secondarily inhalation exposure. At lower lead concentration levels, children have manifested neurological impairment (hyperactivity, peripheral neuropathy) and cognitive deficits (lower IQ). With

inhalation and ingestion, some of the other consequences of lead exposure include cardiovascular toxicity (abnormal EKGs, high blood pressure), nephropathy, interference with Vitamin D metabolism, gastrointestinal symptoms (colic), developmental toxicity (low birthweight), compromise of the immune system, and reproductive toxicity (miscarriage). Studies in these areas for dermal exposures were not found. With ingestion exposures to lead, growth retardation has also been observed in children.

Data in epidemiologic studies were not adequate to establish an association between lead exposure and the development of cancer. Failure to document the specific lead compound, its dose, and the compound's exposure routes were all weaknesses of these studies. An examination of lead production and battery workers who had inhaled lead in the workplace has demonstrated higher rates of total malignancies and mortality from total malignancies than would otherwise have been expected. An increased number of renal cancers were also observed in lead smelter workers. In a number of animal studies, kidney tumors have consistently been reported with lead ingestion exposure. Lead has an USEPA WoE classification of B2 (probable human carcinogen).

4.3.23 Manganese (Mn)

Manganese (CAS# 7439-96-5) is a naturally occurring substance found in various types of rock, and is a trace nutrient in food. In the environment, manganese is combined with oxygen, sulfur or chlorine to form a variety of compounds. Rocks containing manganese are mined for use in the production of steel. Manganese is also used in the production of batteries, pesticides, and fertilizers.

Human exposure occurs through inhalation of fumes or dust (usually in an industrial setting) and by ingestion. Very little manganese is absorbed through the skin. Low concentrations of Mn containing compounds are often present in water. The average concentration of Mn in water is about .004 mg/l (milligrams of manganese per liter). The average human intake is about 10 mg/day. About 3 to 5% of ingested manganese is absorbed by the body. When blood levels of iron are low, a greater percentage of the manganese is absorbed. The manganese that is not absorbed by the gastrointestinal tract is eliminated in the feces. When it is inhaled as a fume or dust, much of the Mn is transported to the gastrointestinal tract and ingested. Excess Mn in the blood is removed in the liver and excreted in bile.

While manganese may be beneficial in low doses, exposure in high doses has been shown to cause adverse health effects. Inhalation of large quantities of manganese dust or fumes causes serious and disabling neurological effects. The symptoms of this disease, called manganism, are speech disturbances, mask-like facial appearance, tremors and psychosis.

While inhalation of manganese clearly causes neurological disturbances, there is little evidence that ingestion of food or water containing Manganese causes these problems. A study conducted in an area with high concentrations of manganese in water (14 mg/l) found some limited evidence that neurological effects may result from oral exposure to manganese. The similarity in symptoms between ingestion and inhalation suggests that excess oral exposure may lead to neurological injury.

There is no evidence to suggest that manganese causes cancer in humans or laboratory animals. Manganese has a USEPA WoE classification of D (not classifiable as to human carcinogenicity).

4.3.24 Nickel (Ni)

Nickel (CAS# 7440-02-0) is a hard metal found in a number of ores, often combined with sulfur, antimony and arsenic and is often used in metal alloys such as stainless steel. Nickel is also used in a number of industrial processes like electroplating, anodizing and casting.

Humans are exposed to nickel by inhaling nickel dust or fumes (usually in an occupational setting), by ingesting nickel in food and water, and from skin contact. For most people, ingestion of food containing nickel is the main source of exposure. The average person takes in .3 mg of nickel per day from food. Typical drinking water contains about .005 mg/l nickel. About 10% of the nickel ingested is absorbed by the intestinal tract. When nickel fumes or dust are inhaled, a larger percentage of the nickel is absorbed. The percentage absorbed depends upon the size and type of particle inhaled. When nickel comes into contact with the skin, it may or may not be absorbed, depending upon what chemicals are combined with the nickel.

The primary health effect from ingestion of excess nickel is gastrointestinal distress including diarrhea, vomiting, abdominal cramps and nausea. When skin contact with nickel is made, a skin allergy often develops resulting in itching, redness and a rash. The most serious effects from exposure to nickel result from inhalation of nickel fumes or dust. Inhalation of nickel in the short run can result in cough, shortness of breath, and fluid in the lungs. Inhalation of nickel is known to cause damage to a developing fetus and can result in cancer.

Nickel is known to be a human carcinogen when inhaled, however, there is no evidence that it causes cancer as a result of ingestion or dermal contact. Nickel has a USEPA WoE classification for inhalation of A (human carcinogen).

4.3.25 Nitrate (NO₃)

Nitrate (CAS No. 14797-55-8) and ammonia are forms of nitrogen which are commonly found in the environment. Ammonia is a component of human and animal waste and penetrates the soil from improperly functioning septic systems, animal feedlots, or manure which has been applied as fertilizer or placed in storage. Microorganisms then transform the ammonia to nitrate. Nitrogen, a component of fertilizer, is also transformed into nitrate. As the nitrate level in the soil exceeds what is needed by plants, water from rainfall or irrigation transports the nitrate (leaching) through the soil into groundwater (which may be used for drinking). This problem is more likely to occur in the rural water supplies.

When ingested, nitrate may be converted by the bacteria in the stomach to nitrite. Because of the low acidity level in the stomachs of infants, bacterial growth is encouraged which enhances the conversion of nitrate to nitrite. The nitrite then reacts with the hemoglobin in blood to form methemoglobin which is unable to carry oxygen. This results in oxygen starvation with death from suffocation in extreme cases. This condition is most often seen in infants. Other nitrogen compounds in the body may react with nitrites to form substances known as N-nitrosamines which have been determined to be carcinogenic in animals. Although inconclusive, epidemiologic studies have shown a possible association between high level exposures to nitrate and nitrites and the development of stomach and esophageal cancer. Nitrate is presently being assessed by USEPA for human carcinogenic potential.

4.3.26 Silver (Ag)

Silver (CAS# 7440-22-4) is a rare element often found in mineral ore in association with other elements. Silver is used in industry for photographic materials, electrical products such as electrical contacts and in batteries. Silver may occur alone or as an oxide, nitrate, or chloride.

The routes of exposure to silver include inhalation of fumes or dust, ingestion of solutions or dust containing silver, and skin or eye contact. Most people are exposed to silver at low levels in food and drinking water.

There is limited information about the absorption of silver following inhalation of silver fumes or dust, however, animal studies indicate that about 90% of silver deposited in the lungs is absorbed into the blood. Many silver compounds including the silver salts are absorbed in varying quantities, but may be as high as 21%. Silver is absorbed through the skin, although the degree of absorption appears to be around 1%. The absorbed silver is then transported by the blood and eventually either deposited in tissue or eliminated in the feces.

The one clinical condition caused by silver in humans is a blue-gray discoloring of the skin called argyria. Argyria usually occurs in an area of repeated or abrasive dermal contact, or over large areas of skin following inhalation exposure. Occupational studies with humans indicate that inhalation of silver compounds may irritate the respiratory tract.

There is no evidence which indicates that silver is a carcinogen. Silver has a USEPA has a WoE classification of D (not classifiable as to human carcinogenicity).

4.3.27 Sulfate (SO₄)

Sulfates are divalent anions (negatively charged radicals) which are found naturally in almost all waters. Human exposure occurs primarily from ingestion of drinking water containing sulfates with toxic effects at high doses (diarrhea and dehydration), especially in infants. No chronic or adverse effects have been documented from long-term exposure to sulfates. No other information, including USEPA carcinogenicity classification, was found for sulfates.

4.3.28 Tetrachloroethylene (PCE)

Tetrachloroethylene (CAS No. 127-18-4, C₂Cl₄) is a halogenated hydrocarbon which is man made. Synonyms include carbon bichloride; 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 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.

The routes of exposure 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.

Toxic effects from PCE exposure have been documented, for the most part, from acute or accidental exposures to humans. Acute inhalation and ingestion exposures in humans have resulted in death. CNS (impaired coordination, anesthesia, unconsciousness, coma), cardiovascular (arrhythmia), hepatic (damage), ocular (irritation), reproductive (spontaneous abortion), renal (dysfunction), and respiratory (irritation) effects were reported with inhalation exposure in humans. CNS (drowsiness, vertigo, coma) and hepatic (jaundice, hepatomegaly) effects were seen in human ingestion exposures. Chemical burns were seen with dermal exposure in dry cleaning workers. Epidemiologic research has shown a potential association between chronic PCE exposure and an increased cancer risk. The findings were inconclusive due to limitations such as a simultaneous exposure to a number of chemicals. In a cohort study which included dry-cleaning workers, a statistically significant excess in mortality from bladder, cervical, and kidney cancers was found with inhalation exposure to PCE. However, the subcohort of dry-cleaning worker, which had the actual exposure to PCE, did not have an excess risk of cancer. No research was found documenting carcinogenicity from human ingestion. In animal inhalation studies, liver and renal cancers and leukemia were associated with inhalation exposure to PCE with a significant rise in liver cancers with ingestion exposure. Genotoxic (sister chromatid exchange) effects have also been reported with inhalation exposure in humans. 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.

4.3.29 Thallium (Tl)

Thallium (CAS No. 7440-28-0) is an element which is found in the earth's crust. It may be detected alone in nature or in combination with other elements such as oxygen, sulfur, and halogens.

Thallium is utilized primarily in the production of electronic devices, switches, and closures. The majority of environmental emissions arise from coal-burning and smelting processes which utilize substances contaminated with thallium, rather than from facilities using thallium compounds. People residing near coal-burning power plants, metal smelters, or cement plants or workers in industries producing or using thallium compounds have the highest exposure levels from thallium. Foods containing thallium are a source of exposure for the public. Since thallium has been detected in cigar stubs and cigarette smoke, smokers may also have high exposure levels of thallium from this source.

Trace metals can affect the toxicity of thallium as demonstrated in animal studies. Potassium has been shown to increase urinary excretion of thallium and reduce its degenerative changes on epiphyseal cartilage and its placental transfer, but, has also been observed to intensify thallium's fatal effect. Potent diuretics (furosemide), activated charcoal, and Prussian blue have been reported to speed up the elimination of thallium.

The routes of exposure include inhalation, ingestion, or dermal contact. Human absorption data were not available for inhalation or dermal exposures to thallium. In animal studies, however, absorption appeared to be complete when thallium was administered intratracheally. Thallium also appears to be absorbed completely when orally administered. A case study suggested complete absorption of thallium following a single oral dose given to a patient with osteogenic sarcoma. No human data were found on the distribution of thallium following inhalation or dermal exposures. Thallium has been observed in various organs of the human body following ingestion exposure with highest levels seen in the human bone, hair, heart, kidney, scalp, and spleen. No data were available on metabolism nor on excretion of thallium following inhalation or dermal exposures. Thallium was, however, found in the urine of a patient 21 days following initial ingestion.

Thallium toxicity has been documented with inhalation and ingestion exposures. In one occupational study, neurological effects of paresthesia, numbness of toes and fingers, burning feet phenomenon, and muscle cramping followed inhalation exposure to thallium. With ingestion exposure, a number of adverse outcomes have been documented. Deaths have been reported due to cardiac or respiratory failure with post-mortem examination revealing axon (nerve) degeneration. Respiratory (alveolar damage, bronchopneumonia), cardiovascular (damage to the heart muscle, cardiac arrhythmia), gastrointestinal (diarrhea, constipation, vomiting, abdominal pain), musculoskeletal (muscle pathology), hepatic (liver damage), renal (kidney damage), and dermal (hair loss) effects have been documented following ingestion exposures in humans. No studies were found which examined the association of

inhalation, ingestion, or dermal exposure to thallium and the development of cancer. Thallium has a WoE classification of D (not classifiable as to human carcinogen).

4.3.30 1,1,1-Trichloroethane (TCA)

1,1,1-Trichloroethane (CAS No. 71-55-6, $\text{CCl}_3\text{-CH}_3$) is a halogenated hydrocarbon which is man made. Synonyms include methylchloroform, methytrichloromethane, trichloromethylmethane, and α -trichloromethane. 1,1,1-Trichloroethane was developed as a safe solvent substitute for carbon tetrachloride and is used for cold cleaning, degreasing, adhesives, aerosols, electronics, and coating in industry and also as a chemical intermediate. In the household, 1,1,1-trichloroethane may be found in liquid detergent, wallpaper glue, insecticides, carpet glue, chlorine bleach scouring powder, and rodenticide. High exposure levels may be found in workers with jobs utilizing 1,1,1-trichloroethane and the general public from ingesting contaminated food or water. 1,1,1-Trichloroethane is released into the environment as a result of its industrial applications and its use by consumers.

No reports were available for the chemical interaction of 1,1,1-trichloroethane with other chemicals in humans. In animals, however, a low dose of ethanol enhances the lethal and behavioral effects of 1,1,1-trichloroethane. The fatal effect of 1,1,1-trichloroethane by intraperitoneal injection in mice is enhanced by the simultaneous injection of nicotine. Phenobarbital promoted liver toxicity in rats. When rabbits were simultaneously exposed to 1,1,1-trichloroethane and ozone, respiratory depression was also heightened.

The routes of exposure for 1,1,1-trichloroethane include inhalation, ingestion, or dermal contact with human absorption of 1,1,1-trichloroethane occurring following these three routes of exposure. In addition, 1,1,1-trichloroethane is distributed in the blood following inhalation and dermal contact and in the fat and liver with ingestion exposure in humans. A small percent of the 1,1,1-trichloroethane is metabolized following inhalation and ingestion. Excretion following exposure by the three routes is believed to occur in the exhaled breathe and urine.

A number of toxic effects in humans have been observed with inhalation and ingestion exposures. With inhalation exposure, 1,1,1-trichloroethane caused death due to CNS depression and cardiac arrhythmia. Cardiovascular (decreased blood pressure), CNS (mild motor impairment to death), GI (nausea and vomiting, diarrhea), hepatic (possible liver damage), ocular (eye irritation), and respiratory (depression) effects have also been seen with inhalation exposure. Gastrointestinal (vomiting, diarrhea) and hepatic (liver damage) effects were documented with ingestion exposure. Dermal exposures resulted in skin irritation and burns in humans.

No human studies were available examining the association of 1,1,1-trichloroethane exposure and the development of cancer with inhalation and dermal contact. However, an increase in lymphosarcomas was found in one animal inhalation study. Research was conducted examining the effect of human ingestion of water with detectable levels of 1,1,1-trichloroethane and the development of cancer. No differences were documented between residents of communities which had detectable levels of 1,1,1-trichloroethane and those which did not. The researchers concluded that the data's insensitivity prevented the detection of differences. An increased incidence of leukemia was observed in one animal ingestion study; however, limitations in the study's experimental design prevented a conclusive statement regarding this finding. 1,1,1-Trichloroethane has an EPA Weight-of-Evidence Classification of D (not classifiable as to human carcinogen).

4.3.31 1,1,2-Trichloroethane (TCA2)

1,1,2-Trichloroethane (CAS# 79-00-5, $\text{CHCl}_2\text{-CH}_2\text{Cl}$) is a halogenated hydrocarbon which does not occur naturally in the environment. Synonyms include vinyl trichloride and B trichloroethane. 1,1,2-Trichloroethane is primarily used as an intermediate in the production of 1,1-dichloroethane. It may occasionally be used as a solvent for fats, waxes and resins. 1,1,2-Trichloroethane is fairly stable when present in the soil or groundwater and may persist for years.

The routes of exposure to 1,1,2-trichloroethane include inhalation, ingestion and dermal contact. Little is known about the absorption efficiency of 1,1,2-trichloroethane, however studies indicate that about 80% is rapidly absorbed. Animal studies suggest that it is also well absorbed by the skin. Following absorption, it is rapidly distributed by the blood and deposited in fat and body organs. A large percentage of the chemical is then metabolized in the liver and converted to various other compounds. Most of the unmetabolized 1,1,2-trichloroethane is exhaled.

The only documented human health effect from exposure to 1,1,2-trichloroethane is skin irritation and burning following direct dermal contact. Studies in laboratory animals show that in sufficient quantities it may cause kidney damage. Other effects include central nervous system depression typical of many chlorinated hydrocarbons.

There is no evidence to indicate that 1,1,2-trichloroethane is carcinogenic in humans, however, it has been shown to be carcinogenic in a strain of mice. 1,1,2-Trichloroethane has a USEPA WoE classification of C (possible human carcinogen).

4.3.32 Trichloroethylene (TCE)

Trichloroethylene (CAS No. 79-01-6, C_2HCl_3) is a halogenated hydrocarbon. Synonyms include 1,1,1-trichloro-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.

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 doses 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.

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. Toxic effects from TCE exposure have been

observed in humans, particularly in the occupational setting. A number of deaths have been documented in workers exposed by inhalation and by accidental ingestion. With acute inhalation exposure, cardiac arrhythmia was found to be the cause of death. With ingestion exposure, death was due to hepatorenal failure. Cardiovascular (arrhythmia, abnormal EKG), CNS (dizziness, sleepiness, unconsciousness), GI (nausea and vomiting), hepatic (necrosis, degeneration), ocular (eye irritation), and renal (dysfunction) effects have been documented in humans with inhalation exposure. Hematological (depressed δ -aminolevulinate dehydrase activity in liver, bone, erythrocytes) and reproductive (increase sperm morphology abnormality) effects have been observed in animals with inhalation exposure. Cardiovascular (myocardial infarction), neurological (muscle weakness, unconsciousness), and hepatic (hepatorenal failure) effects have been observed in acute human ingestion exposures. Dermal, developmental, GI, immunological, renal, and respiratory effects have also been documented in humans who had ingested water contaminated with TCE; however, other contaminants were in the water making it impossible to determine if TCE alone caused the adverse health effects. Dermal exposures in workers have also resulted in skin problems.

Due to study limitations (e.g. absence of exposure data, small sample size), epidemiologic research has proven to be inconclusive with regard to the cancer causing potential of TCE. With inhalation exposure, cancer of various sites (lymphomas; bladder, respiratory, cervical, and skin cancers) have been observed in epidemiological studies of workers. Other studies have not detected an increased incidence of cancers by inhalation. Conflicting reports have also been observed with ingestion exposures. The rate of childhood leukemia was elevated in Woburn, Massachusetts, where drinking water was contaminated with TCE and other contaminants. An association was seen between drinking contaminated well water and increased risk of childhood leukemia. The data were subsequently reevaluated, and the association was not sustained. The original results of the study have been questioned due to these inconsistent findings, the use of relatives of leukemia victims acting as interviewers, presence of multiple contaminants in water, and other questionable factors. In animals, however, significant increases have been seen in the rate of lung cancer and hepatic and testicular tumors, and a slight increase in renal cancers with inhalation. The rate of liver and kidney cancers were increased with ingestion exposures. Genotoxic (suggestive of sister chromatid exchange) effects have been observed with human inhalation exposures. 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.

1.3.33 Vinyl Chloride (VC)

Vinyl chloride (CAS No. 75-01-4, C_2H_3Cl) is a halogenated hydrocarbon which occurs in the gaseous state. Synonyms include chloroethene, chloroethylene, ethylene monochloride, and monochloroethylene. Vinyl chloride is commonly used in the manufacture of polyvinyl chloride (PVC), a component of plastic and vinyl products. Vinyl chloride is produced from the anaerobic degradation of trichloroethylene and is found in the air and in wastewater discharges from the plastics industries. The highest potential exposure levels are seen in workers employed in the production of vinyl chloride and in populations residing near these industrial facilities or near landfills and waste disposal sites.

Vinyl chloride interacts with other substances to produce acute toxic effects to the liver as demonstrated by animal studies. Phenobarbital, Arochlor 1254 (a polychlorinated biphenyl), and trichloropropene oxide have been shown to promote the liver toxicity in animals from vinyl chloride exposure. Ingested ethanol given simultaneously with inhaled vinyl chloride has also been observed to influence the adverse fetal and maternal effects from vinyl chloride exposure. Contrary to these other substances, cysteine reduced the liver toxicity in animals from vinyl chloride exposure.

The routes of exposure for vinyl chloride include inhalation and ingestion and less likely by dermal exposure. Human inhalation data have demonstrated that 42% of an inhaled dose of vinyl chloride is absorbed. No human studies were available examining absorption following oral and dermal exposure to vinyl chloride. However, a number of rat studies have shown complete absorption of vinyl chloride from the gastrointestinal tract following ingestion exposure. Absorption was estimated to be 0.03% or less in monkeys exposed for approximately two hours dermally. No human data were available on the distribution of vinyl chloride following inhalation, ingestion, or dermal contact. Following inhalation, animal studies have demonstrated distribution of vinyl chloride to the bile duct, blood, digestive tract, fat, kidney, liver, lung, muscle, salivary and lacrimal glands, skin, spleen, thymus, and urinary tract with highest levels of vinyl chloride metabolites found in the kidney and liver. With oral exposure, vinyl chloride was distributed to the fat, liver, lung, muscle, plasma, and skin with highest levels in the liver. The metabolic pathway has been documented for animals with inhalation and ingestion exposures. One of the pathways results in the formation of a highly reactive epoxide known as 2-chloroethylene oxide. Human data indicate that vinyl chloride metabolites are excreted in the urine and much less by exhalation following an inhalation exposure. Excretion data were unavailable for oral and dermal exposures in humans. With animals, oral exposure to vinyl chloride has resulted in excretion by exhalation and in the urine and feces.

Toxicity from vinyl chloride exposure has been primarily observed with inhalation exposure. Cases of death from narcosis have been documented in the occupational setting. Hepatic (liver damage), neurological (dizziness, ataxia, headache, narcosis, peripheral neuropathy), and reproductive (ovarian dysfunction, uterine growth) effects have also been noted with inhalation exposures. A systemic effect known as vinyl chloride disease has been seen with occupational inhalation exposure to vinyl chloride. The clinical condition is manifested by dissolution of the distal phalanges (fingers), circulatory disturbance, Raynaud's syndrome, scleroderma and effects to the blood, liver, and lung. This condition is also seen in animals with ingestion exposures.

A number of epidemiologic studies have demonstrated an association between inhalation exposure to vinyl chloride and the development of angiosarcoma, a rare liver cancer. This finding was substantiated in animal studies. More recent studies have also shown an association between inhalation exposure to vinyl chloride and the development of brain cancer. In animal studies, liver angiosarcoma and lung tumors were reported with inhalation and ingestion exposures, and brain tumors with inhalation exposure. Genotoxicity (chromosomal aberrations in lymphocytes of exposed workers) has also been reported. Children appear to be a sensitive subpopulation. Vinyl chloride has an USEPA WoE classification of A (human carcinogen).

4.3.34 Zinc (Zn)

Zinc (CAS# 7740-66-6) and compounds containing zinc are found naturally in the air, water, soil and foods. Zinc has many industrial uses and is a component in several metal alloys including brass. Zinc is also an important food element needed by the body in low doses, but can be harmful if too much is taken in.

The primary exposure routes for zinc are ingestion and inhalation. About 20-30% of ingested zinc is absorbed by the body when ingested. Most zinc is unabsorbed and passes in the feces. The greater the quantity of zinc present in the blood and tissues, the less it will be absorbed. There is some evidence to indicate that high calcium intake may also decrease the amount of zinc absorbed by the body. Zinc may also be absorbed through inhalation of zinc containing fumes, usually in an industrial setting. Very little zinc is absorbed through the skin.

The major health effects of drinking water with too much zinc are digestive problems. These problems include intestinal cramps and diarrhea. Higher doses of zinc that may occur from taking too many dietary supplements may result in more acute symptoms including nausea, vomiting, pancreas problems and intestinal bleeding.

There is no evidence that zinc causes cancer or birth defects in humans. Zinc has a USEPA WoE classification of D (not classifiable as to human carcinogenicity).

4.4 SUMMARY

Eight of the study's chemicals of concern are of particular interest because of their USEPA WoE classifications of A and B. Known human carcinogens (WoE group A) among the COPC include arsenic, chromium (VI) (when inhaled), benzene, and vinyl chloride. Nine (9) other COPC are probable human carcinogens in group B2. These include lead, bromodichloromethane, carbon tetrachloride, chloroform, 1,2-dichloroethane, dichloromethane, and trans-1,3-dichloropropene. PCE and TCE are also classified in group B2; their status is currently being reviewed by USEPA. The analysis of risk from exposure to these substances is an important preventive step in averting health problems because of their carcinogenic potential. The results of the risk assessment and the implications will be discussed in Chapter 5.

5.0 RISK CHARACTERIZATION

Risks, both current and potential, are characterized and evaluated in this chapter utilizing exposure and toxicology information previously developed and discussed. Risk characterization is presented in both quantitative and/or qualitative format. When data are available, quantitative risk characterizations are performed and evaluated qualitatively. If data are unavailable, possible risks are discussed in a qualitative manner. The specifics of these risk characterizations are reported in the following sections:

- * Section 5.1 - Risk Estimation Methods
- * Section 5.2 - Current Conditions
- * Section 5.3 - Future Land Use
- * Section 5.4 - Uncertainties in the Risk Characterization Process
- * Section 5.5 - Summary

5.1 RISK ESTIMATION METHODS

Risk estimation methods used in this report were based on USEPA guidelines (USEPA, 1989b, 1991a). Risk calculations proceed from estimation for a single compound and exposure route, to a summation of risk for all chemicals of concern for a given route (USEPA, 1188b), and culminating with a summation of risk across exposure routes.

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 at about 3×10^{-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. If there were no positive detections for a chemical of concern at a particular well, then UCL of one-half the SQLs was used in calculation of the risk or hazard. 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.

Chemicals with a class "C" WoE are evaluated on a chemical by chemical basis to determine whether a quantitative approach, using a SF, or a modified RfD approach is most appropriate. This methodology is considered proper for the assessment of carcinogenic exposures to class "C" compounds in the USEPA Region IX Superfund program.¹

This risk assessment uses the modified RfD approach to assess cancer risk potential for the following class "C" chemicals: chloromethane, dibromochloromethane, 1,4-dichlorobenzene, 1,1-dichloroethane, 1,1-dichloroethylene, and 1,1,2-trichloroethane. These chemicals were detected at very low frequencies with the exception of 1,1-DCA and 1,1-DCE. DBCM was detected in less than three (3) per cent of samples and CM, 1,4-DCB, and 1,1,2-TCA in less than one (1) per cent of the samples (Table 2.3). In addition the slope factors developed for these chemicals are based upon limited evidence of carcinogenicity. The modified RfD approach was used to evaluate 1,1-DCE due to uncertainty in the slope factor. The SF is based on data from one study which showed no significant increase in rat tumor incidence. There is no SF for 1,1-DCA and therefore the modified RfD approach must be used.

To assess cancer risk potential using a modified-RfD approach the estimated CDI (dose) is compared to the oral RfD, divided by an additional safety factor of ten:

¹ Personal communication from Gerald Hiatt, Senior Risk Assessment Policy Advisor, EPA, Region IX.

$$\text{Cancer Hazard Quotient} = \text{CDI}/(\text{RfD}/10)$$

The result, the CHQ, is interpreted in the same manner as a hazard quotient; values less than unity are indicative of no significant carcinogenic risk. This approach was also extended to substances with WoE ratings of "C" or above if an RfD but no slope factor was available (1,1-DCA). Noncancer toxicity potential is evaluated using the usual RfD approach (Section 5.1.2). Slope factors for TCE and PCE are presently under review by USEPA. Due to significant detections of these chemicals in the groundwater, and the lack of new guidance, the previously developed slope factors shall be used for this risk assessment.

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:

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

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

RfD = reference dose in mg/kg-day.

The HQ is *not a probability*. If the HQ exceeds 1.0 there is concern that the exposed population may experience adverse health effects. 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.

5.1.3 Health Risks for Multiple Substances

Exposures to more than one chemical may often occur at sites under consideration. Very little data are available on the combined action of chemical mixtures. It is possible that the presence of two or more chemicals may have an antagonistic, synergistic, or additive effect on health. Unless data are

available supporting another interpretation, carcinogenic risk, carcinogenic and non-carcinogenic hazard quotients are assumed to be additive (USEPA, 1988b). In both cases, values for individual chemical specific values are summed to obtain an estimate of total ELCR, the carcinogenic hazard index (CHI), or the systemic hazard index (HI).

5.1.4 Health Risks Across Pathways

If the same population is exposed via more than one pathway, results from individual pathways may be summed for both carcinogenic and noncarcinogenic effects. Care must be taken to assure that appropriate pathways are summed for a population. A typical exposure across all pathways is usually calculated by summing average exposures. The reasonable maximum exposure (RME) across pathways may use a combination of average and RME exposures if this provides the best maximum estimate for a population. When pathways are summed, it must be done for the same receptor population.

5.2 CURRENT CONDITIONS

There is currently only one potentially complete exposure pathway for ingestion of groundwater. This is the discharge of groundwater from the well 4626G for private domestic uses. However, no statutory prohibitions exist for the drilling of other private wells in the study area. Therefore, the potential exists for exposures, if a well were to be drilled. This is very unlikely for three reasons: 1) water is supplied to the area by the City of Phoenix at a low cost, and it would not be cost effective to drill a private well; 2) Drilling permits must be obtained from the Arizona Department of Water Resources; the state and the City of Phoenix would strongly discourage the granting of such a permit; 3) Phoenix must approve zoning permits for the drilling, and are not considered likely to do so.

5.2.1 Alluvial Groundwater

Potential receptors are residents living in the investigation area underlain by the contaminant plume (Figure 1.1 and 1.2). Table 5.1 refers to the list of contaminants detected in groundwater or soil gas. An assessment was performed on each monitor well, using 1988-1991 data. A separate set of potential risk estimates was calculated for samples taken from the alluvium and for those taken at or below the interface of the alluvium and the bedrock. The former samples have been referred to as

Table 5.1 - Summary of Chemicals of Potential Concern.

Chemical	WoE	Slope Factors ^d		RfD ^e		Water	
		Oral	Inhalation	Oral	Inhalation	HBGL ($\mu\text{g/L}$)	MCL ($\mu\text{g/L}$)
Organic							
Benzene	A	2.9E-02	2.9E-02	--	--	1E+00	5E+00
Bromodichloromethane	B2	1.3E-01	--	2E-02	--	3E-01	1E+02
Carbon tetrachloride	B2	1.3E-01	1.5E-05	7E-04	--	3E-02	5E+00
Chlorobenzene	D	--	--	2E-02	5E-03	1E+02	--
Chloroform	B2	6.1E-03	8.1E-02	1E-02	--	6E+02	1E+02
Chloromethane	C	1.3E-02	1.8E-02	--	--	3E+00	--
Dibromochloromethane	C	8.4E-02	--	2E-02	--	1E+01	1E+02
1,2-Dichlorobenzene	D	--	--	9E-02	--	6E+02	--
1,4-Dichlorobenzene	C	2.4E-02	--	--	--	7E+01	8E+01
1,1-Dichloroethane	C	--	--	1E-01	1E-01	7E+01	--
1,2-Dichloroethane	B2	9.1E-02	2.6E-05	--	--	4E-01	5E+00
1,1-Dichloroethylene ^a	C	6.0E-01	1.2E+00	9E-03	--	7E+01	7E+01
1,2-Dichloroethylene	D	--	--	2E-02	--	7E+01	7E+01
Dichloromethane	B2	7.5E-03	4.7E-07	6E-02	--	5E+00	--
1,3-Dichloropropene	B2	1.8E-01	3.7E-05	3E-04	6E-03	--	--
Tetrachloroethylene ^a	B2	5.1E-02	1.1E-03	1E-02	--	7E-01	5E+00
1,1,1-Trichloroethane	D	--	--	9E-02	3E-01	2E+02	2E+02
1,1,2-Trichloroethane	C	5.7E-02	1.6E-05	4E-03	--	3E+00	--
Trichloroethylene ^a	B2	1.1E-02	1.7E-02	--	--	3E+00	5E+00
Vinyl chloride	A	1.9E+00	2.9E-01	--	--	2E-02	2E+00

Table 5.1 - Continued.

Chemical	WoE	Slope Factors ^d		RfD ^e		----- Water -----	
		Oral	Inhalation	Oral	Inhalation	HBGL ($\mu\text{g/L}$)	MCL ($\mu\text{g/L}$)
Inorganic						(mg/L)	(mg/L)
Arsenic (As)	A	1.8E+00 ^f	--	3E-04	--	5E-02	5E-02
Boron (B)	D	--	--	9E-02	--	6E-01	--
Cadmium (Cd)	D	--	--	5E-04	--	4E-03	5E-03
Chromium (III)	D	--	--	1E+00	--	1E-01	1E-01
Chromium (VI)	A	--	1.2E-02	5E-03	--	4E-02	--
Cyanide	D	--	--	2E-02	--	2E-01	--
Fluoride (F)	D	--	--	6E-02	--	4E-01	4E+01
Lead (Pb) ^b	B2	--	--	--	--	5E-03	--
Manganese (Mn)	--	--	--	1E-01	1E-04	--	--
Nickel (Ni)	D	--	--	2E-02	--	1E-01	--
Nitrate (NO ₃) ^b	--	--	--	1.6E+00	--	1E+01	1E+01
Silver (Ag)	D	--	--	5E-03	--	5E-02	5E-02
Sulfate (SO ₄) ^c	--	--	--	--	--	4E+02	--
Thallium (Tl)	D	--	--	7E-05	--	5E-04	--
Zinc (Zn)	--	--	--	2E-01	--	1E+00	--

a: Currently under review by USEPA, former classification is used.

b: Currently being evaluated by USEPA.

c: No USEPA classification.

d: Slope factors are in units of $(\text{mg}\backslash\text{kg}\text{-day})^{-1}$.

e: RfD are in units of $\text{mg}\backslash\text{kg}\text{-d}$

f: Calculated from unit risk. (IRIS, 7/92)

alluvial, and the latter as bedrock. Risk was assessed for domestic use of well 4626G, using data collected from 1987-92, inclusive (Chapter 3).

The well by well approach was taken due to the large area covered by the monitor wells and the large differences in concentrations of chemicals over that area. It is hoped that risk management decisions would be facilitated through the use of this method. The potential ingestion risk, CHQ, and HQ were calculated for each chemical of concern, for each well. A well total for ELCR and the CHI and HI were determined by summing the entries for each well. Appendix Table 27 is the worksheet showing the procedure used, and the results for potential ingestion risks and hazards. A summary of the estimated potential risk and hazard from ingestion is summarized by well in Table 5.2. The formulae used in the calculations are included on the following page. Estimated potential risks and hazards for combined ingestion, inhalation, and dermal domestic exposures is shown in Table 5.3.

The distribution of potential ELCR due to domestic ground water use has been mapped for the study area. The potential ELCR estimates are presented by two dimensional contour maps and also three dimensionally. Figure 5.1 shows the locations of the sampling sites and Figures 5.2 through 5.11 are representations of the potential ELCR for groundwater. The images were produced using the software package Surfer. An inverse distance method was used to estimate the contours shown in the figures.

Average Potential Excess Lifetime Cancer Risk

Average, potential ELCR for domestic use of water drawn from alluvium, varied from a maximum of $1E-02$ (one-in-one-hundred) at off-site wells DM117 and MP11, to a minimum of less than $1E-06$ (one-in-one-million) at well DM123 (Table 5.3). Wells DM 125 and DM126 were slightly higher at $2E-06$.

Seventy-three percent (73%) of the potential ELCR for DM117 is due to the presence of high concentrations of VC ($1700 \mu\text{g/L}$). The remaining 27% of the ELCR is almost entirely due to arsenic (Table 5.4). The distribution of potential ELCR for average domestic exposures to alluvial groundwater is represented graphically in Figure 5.2 and 5.3. The wells with the highest potential risk were east of the Old Crosscut Canal, immediately to the west of the facility and on-site. Another area of elevated potential ELCR was in the southwest corner of the site. There were four regions of elevated potential ELCR to the southwest of the facility; the first, east of the Grand Canal, and three further to the southwest. These did not follow the general pattern of the risk contours, and may be due to other sources.

Table 5.2. — Groundwater ingestion potential excess lifetime cancer risk, cancer hazard index, and non-cancer hazard index for each groundwater sampling site.^a

ALLUVIAL WELL	AVERAGE EXPOSURE (MEAN)			REASONABLE MAXIMUM EXPOSURE		
	CARCINOGENIC		NON-CARC	CARCINOGENIC		NON-CARC
	RISK	HZD INDX	HZD INDX	RISK	HZD INDX	HZD INDX
4626G	5E-05	1.1E-03	2.0E+00	2E-04	5.2E-03	2.2E+00
AZSLD	5E-05	1.2E-03	1.6E+00	2E-04	3.9E-03	2.1E+00
DM103	3E-03	1.5E-01	2.2E+00	2E-02	8.8E-01	3.6E+00
DM104	1E-03	4.1E-02	2.7E+01	5E-03	3.1E-01	2.8E+01
DM106	1E-05	6.6E-03	8.1E-01	1E-04	6.8E-02	2.6E+00
DM107	1E-03	1.5E-01	2.3E+01	4E-03	1.5E+00	3.3E+01
DM111	2E-04	3.5E-01	8.4E+00	8E-04	3.2E+00	1.2E+01
DM112	2E-04	1.6E+00	8.0E+00	1E-03	1.5E+01	1.3E+01
DM113	3E-04	2.1E-01	1.0E+01	1E-03	1.6E+00	2.1E+01
DM114	3E-04	1.1E-02	4.8E+00	2E-03	3.2E-01	9.2E+00
DM115	2E-04	2.2E-02	1.3E+01	9E-04	1.6E-01	1.8E+01
DM117	7E-03	4.2E-01	3.5E+01	3E-02	2.2E+00	4.3E+01
DM118	6E-05	1.2E-03	5.7E+00	3E-04	3.9E-03	5.9E+00
DM119	8E-07	1.3E-03	1.9E-03	4E-06	5.4E-03	2.8E-03
DM120	1E-04	9.3E-03	6.5E+00	5E-04	4.6E-02	8.6E+00
DM121	1E-05	1.7E-02	7.9E-01	1E-04	9.5E-02	1.9E+00
DM122	2E-03	3.1E-03	3.2E+01	2E-02	2.0E-02	9.0E+01
DM123	4E-07	1.2E-03	1.4E-03	1E-06	3.9E-03	1.4E-03
DM124	3E-04	1.9E-03	6.6E+00	1E-03	9.6E-03	9.3E+00
DM125	1E-06	3.0E-03	5.8E-03	7E-06	2.0E-02	1.1E-02
DM126	8E-07	1.3E-03	6.4E-03	4E-06	5.3E-03	1.2E-02
DM201	6E-04	4.3E+01	4.4E+01	5E-03	2.5E+02	8.1E+01
DM202	1E-04	2.3E-01	8.8E+00	7E-04	1.4E+00	1.3E+01
DM303	4E-04	2.2E+00	1.3E+01	2E-03	1.6E+01	1.7E+01
DM304	5E-04	2.2E+00	1.4E+01	2E-03	1.8E+01	2.7E+01
DM501	4E-06	2.8E-03	1.8E-01	2E-05	1.4E-02	2.7E-01
DM502	7E-06	2.1E-01	1.1E+00	3E-05	1.1E+00	1.7E+00
DM503	2E-04	1.2E-03	7.6E+00	9E-04	3.9E-03	1.1E+01
DM504	1E-04	8.6E-02	1.1E+01	8E-04	4.6E-01	1.4E+01
DM505	1E-05	1.2E-03	2.0E+00	3E-05	3.9E-03	2.2E+00
DM506	4E-06	2.2E-02	2.9E-01	2E-05	8.8E-02	4.9E-01
DM507	4E-05	5.9E-02	3.0E+00	3E-04	5.1E-01	5.9E+00
DM508	1E-04	1.8E-03	3.9E+00	3E-04	8.9E-03	3.9E+00
DM509	8E-05	4.2E+03	3.7E+00	7E-04	3.9E-02	6.5E+00
MP03	3E-03	5.8E+01	1.8E+02	2E-02	3.9E+02	4.1E+02
MP09	2E-03	5.6E+00	9.3E+01	1E-02	3.4E+01	1.8E+02
MP11	6E-03	8.6E-01	9.6E+01	4E-02	5.8E+00	1.8E+02
MP13	3E-04	4.3E-02	1.4E+01	1E-03	2.4E-01	2.0E+01
MP16	2E-04	5.3E-02	1.2E+01	1E-03	4.6E-01	1.7E+01
MP20	2E-04	1.2E-03	2.9E+00	2E-03	3.9E-03	7.8E+00
MP28	4E-05	4.3E-03	1.4E+00	9E-04	4.9E-02	5.0E+00
MP30	3E-04	1.2E-03	7.8E+00	3E-03	3.9E-03	1.7E+01
MP36	8E-04	3.0E+00	3.3E+01	3E-03	1.6E+01	4.6E+01
MP49	3E-04	7.4E-01	2.0E+01	1E-03	4.3E+00	3.1E+01
MP50	1E-04	3.0E-01	6.5E+00	6E-04	1.8E+00	1.2E+01
MP51	2E-04	3.9E-02	1.1E+01	1E-03	3.2E-01	1.6E+01
MP52	6E-05	8.2E-04	3.9E+00	2E-04	5.0E-03	4.5E+00
MP53	6E-04	8.2E-04	1.2E+01	2E-03	5.0E-03	1.2E+01
WILLIS	9E-05	1.5E-01	6.9E+00	3E-04	1.9E+00	9.1E+00

Table 5.2. -- Continued

BEDROCK WELL	AVERAGE EXPOSURE (MEAN)			REASONABLE MAXIMUM EXPOSURE		
	CARCINOGENIC		NON-CARC	CARCINOGENIC		NON-CARC
	RISK	HZD INDX	HZD INDX	RISK	HZD INDX	HZD INDX
DM101	8E-06	2.4E-02	3.9E-02	4E-05	1.2E-01	6.1E-02
DM103	4E-03	1.1E-01	8.6E+00	2E-02	4.8E-01	1.4E+01
DM104	3E-05	4.5E-03	5.5E+00	1E-04	2.1E-02	5.5E+00
DM106	3E-06	3.6E-03	4.5E-02	1E-05	2.2E-02	7.9E-02
DM119	8E-07	1.3E-03	2.4E-03	4E-06	5.7E-03	4.4E-03
DM121	8E-06	5.4E-03	2.1E-01	5E-05	3.1E-02	4.9E-01
DM123	4E-07	1.2E-03	1.6E-03	1E-06	3.9E-03	1.8E-03
DM125	3E-06	6.6E-03	2.2E-02	1E-05	3.1E-02	3.5E-02
DM501	6E-07	2.3E-03	1.0E-02	2E-06	9.8E-03	1.8E-02
DM502	1E-06	6.1E-02	2.3E-01	7E-06	3.5E-01	3.9E-01
DM506	3E-06	1.2E-02	2.5E-01	2E-05	5.9E-02	4.2E-01
DM507	1E-05	5.6E-02	1.3E+00	7E-05	5.0E-01	1.7E+00
MP03	7E-02	1.1E+01	8.3E+03	5E-01	6.4E+01	1.5E+04
MP09	5E-02	2.3E+00	2.9E+01	3E-01	2.1E+01	8.6E+01
MP11	2E-04	4.6E-02	6.4E+00	7E-04	3.5E-01	7.8E+00
MP13	7E-05	3.1E-03	1.8E+00	4E-04	2.2E-02	2.6E+00
MP16	2E-04	9.4E-04	3.2E+00	1E-03	4.3E-03	6.6E+00
MP20	3E-04	1.2E-03	5.1E+00	3E-03	3.9E-03	1.2E+01
MP25	1E-04	1.2E-03	2.6E+00	4E-04	3.9E-03	2.7E+00
MP28	7E-05	1.2E-03	1.6E+00	5E-04	3.9E-03	2.0E+00
MP30	1E-04	1.2E-03	2.7E+00	1E-03	3.9E-03	6.8E+00
MP36	7E-03	7.9E+00	7.0E+02	4E-02	4.5E+01	1.2E+03
MP49	3E-04	6.3E-02	1.7E+01	6E-03	4.0E-01	5.6E+01
MP50	1E-04	1.2E-01	8.8E+00	6E-04	3.9E-01	1.7E+01
MP51	2E-05	3.1E-02	5.4E-02	3E-04	4.1E-01	2.2E-01
MP53	4E-07	9.0E-04	4.3E-03	2E-06	3.9E-03	7.3E-03

a. Variable and formula definitions. If there were no positive detections for a chemical of concern at a particular well, then one-half of the mean or UCL of the SQL was used in calculation of the risk or hazard.

MEAN - Arithmetic average of samples including one-half times the sample quantitation limits (SQLs)

CARCINOGENIC	$CDI = \frac{(\text{MEAN Concentration} \times \text{ingestion rate}(2 \text{ L/d}) \times \text{frequency}(350 \text{ d/yr}) \times \text{duration}(9 \text{ yrs}))}{(\text{body weight}(70 \text{ kg}) \times \text{averaging time}(70 \text{ yrs} \times 365 \text{ d/yr}))}$
--------------	--

CARCINOGENIC	RISK = CARCINOGENIC EXPOSURE X slope factor(chemical specific)
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CARCINOGENIC	HAZARD QUOTIENT = (CARCINOGENIC EXPOSURE) + (reference dose(Rfd) + safety factor(10))
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NON-CARCINOGENIC	$CDI = \frac{(\text{MEAN Concentration} \times \text{ingestion rate}(2 \text{ L/d}) \times \text{frequency}(350 \text{ d/yr}) \times \text{duration}(9 \text{ yrs}))}{(\text{body weight}(70 \text{ kg}) \times \text{averaging time}(9 \text{ yrs} \times 365 \text{ d/yr}))}$
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NON-CARCINOGENIC	HAZARD QUOTIENT = NON-CARCINOGENIC EXPOSURE + reference dose(Rfd)
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RME - Reasonable Maximum Exposure using 95% upper confidence limit (UCL) of mean concentration

CARCINOGENIC	$CDI = \frac{(\text{UCL Concentration} \times \text{ingestion rate}(2 \text{ L/d}) \times \text{frequency}(350 \text{ d/yr}) \times \text{duration}(30 \text{ yrs}))}{(\text{body weight}(70 \text{ kg}) \times \text{averaging time}(70 \text{ yrs} \times 365 \text{ d/yr}))}$
--------------	--

CARCINOGENIC	RISK = same as above
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CARCINOGENIC	HAZARD QUOTIENT = same as above
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NON-CARCINOGENIC	$CDI = \frac{(\text{UCL Concentration} \times \text{ingestion rate}(2 \text{ L/d}) \times \text{frequency}(350 \text{ d/yr}) \times \text{duration}(30 \text{ yrs}))}{(\text{body weight}(70 \text{ kg}) \times \text{averaging time}(30 \text{ yrs} \times 365 \text{ d/yr}))}$
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NON-CARCINOGENIC	HAZARD QUOTIENT = same as above
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Table 5.3 -- Total groundwater ingestion, inhalation, and dermal potential excess lifetime carcinogenic risk, cancer hazard index and non-cancer hazard index for each groundwater sampling site.^a

ALLUVIAL WELL	AVERAGE EXPOSURE (MEAN)			REASONABLE MAXIMUM EXPOSURE		
	CARCINOGENIC		NON-CARC	CARCINOGENIC		NON-CARC
	RISK	HZD INDX	HZD INDX	RISK	HZD INDX	HZD INDX
4626G	1E-04	2.3E-03	3.9E+00	4E-04	1.0E-02	4.3E+00
AZSLD	1E-04	2.4E-03	3.3E+00	3E-04	7.9E-03	4.1E+00
DM103	5E-03	3.0E-01	4.5E+00	3E-02	1.8E+00	7.3E+00
DM104	3E-03	8.3E-02	5.4E+01	1E-02	6.1E-01	5.5E+01
DM106	2E-05	1.3E-02	1.6E+00	2E-04	1.4E-01	5.2E+00
DM107	2E-03	3.1E-01	4.5E+01	9E-03	3.0E+00	6.7E+01
DM111	4E-04	7.1E-01	1.7E+01	2E-03	6.4E+00	2.4E+01
DM112	5E-04	3.1E+00	1.6E+01	3E-03	3.1E+01	2.5E+01
DM113	6E-04	4.3E-01	2.0E+01	3E-03	3.3E+00	4.2E+01
DM114	6E-04	2.3E-02	9.5E+00	4E-03	6.4E-01	1.8E+01
DM115	4E-04	4.4E-02	2.6E+01	2E-03	3.2E-01	3.6E+01
DM117	1E-02	8.4E-01	7.0E+01	6E-02	4.4E+00	8.7E+01
DM118	1E-04	2.4E-03	1.1E+01	5E-04	7.9E-03	1.2E+01
DM119	2E-06	2.6E-03	3.9E-03	8E-06	1.1E-02	5.5E-03
DM120	2E-04	1.9E-02	1.3E+01	1E-03	9.2E-02	1.7E+01
DM121	3E-05	3.3E-02	1.6E+00	2E-04	1.9E-01	3.9E+00
DM122	4E-03	6.2E-03	6.4E+01	4E-02	3.9E-02	1.8E+02
DM123	8E-07	2.4E-03	2.9E-03	3E-06	7.9E-03	2.9E-03
DM124	5E-04	3.7E-03	1.3E+01	3E-03	1.9E-02	1.9E+01
DM125	2E-06	6.0E-03	1.2E-02	1E-05	4.0E-02	2.3E-02
DM126	2E-06	2.5E-03	1.3E-02	9E-06	1.1E-02	2.3E-02
DM201	1E-03	8.7E+01	8.8E+01	1E-02	5.0E+02	1.6E+02
DM202	3E-04	4.7E-01	1.8E+01	1E-03	2.9E+00	2.7E+01
DM303	9E-04	4.4E+00	2.5E+01	4E-03	3.2E+01	3.5E+01
DM04	1E-03	4.3E+00	2.9E+01	5E-03	3.5E+01	5.5E+01
DM501	8E-06	5.7E-03	3.6E-01	4E-05	2.8E-02	5.5E-01
DM502	1E-05	4.1E-01	2.2E+00	7E-05	2.1E+00	3.4E+00
DM503	3E-04	2.4E-03	1.5E+01	2E-03	7.9E-03	2.1E+01
DM504	3E-04	1.7E-01	2.3E+01	2E-03	9.2E-01	2.8E+01
DM505	2E-05	2.4E-03	4.0E+00	7E-05	7.9E-03	4.4E+00
DM506	8E-06	4.4E-02	5.7E-01	5E-05	1.8E-01	9.7E-01
DM507	8E-05	1.2E-01	5.9E+00	6E-04	1.0E+00	1.2E+01
DM508	2E-04	3.6E-03	7.8E+00	6E-04	1.8E-02	7.8E+00
DM509	2E-04	8.3E-03	7.5E+00	1E-03	7.7E-02	1.3E+01
MP03	5E-03	1.2E+02	3.7E+02	4E-02	7.9E+02	8.2E+02
MP09	3E-03	1.1E+01	1.9E+02	2E-02	6.9E+01	3.5E+02
MP11	1E-02	1.7E+00	1.9E+02	7E-02	1.2E+01	3.6E+02
MP13	7E-04	8.7E-02	2.8E+01	3E-03	4.8E-01	4.1E+01
MP16	5E-04	1.1E-01	2.4E+01	2E-03	9.1E-01	3.5E+01
MP20	3E-04	2.4E-03	5.8E+00	3E-03	7.9E-03	1.6E+01
MP28	8E-05	8.6E-03	2.8E+00	2E-03	9.8E-02	9.9E+00
MP30	7E-04	2.4E-03	1.6E+01	6E-03	7.9E-03	3.3E+01
MP36	2E-03	6.0E+00	6.6E+01	6E-03	3.1E+01	9.1E+01
MP49	6E-04	1.5E+00	4.0E+01	3E-03	8.6E+00	6.1E+01
MP50	2E-04	5.9E-01	1.3E+01	1E-03	3.6E+00	2.5E+01
MP51	4E-04	7.9E-02	2.3E+01	2E-03	6.5E-01	3.2E+01
MP52	1E-04	1.6E-03	7.8E+00	5E-04	1.0E-02	8.9E+00
MP53	1E-03	1.6E-03	2.5E+01	4E-03	1.0E-02	2.5E+01
WILLIS	2E-04	3.1E-01	1.4E+01	7E-04	3.9E+00	1.8E+01

Table 5.3 -- Continued

BEDROCK WELL	AVERAGE EXPOSURE (MEAN)			REASONABLE MAXIMUM EXPOSURE		
	CARCINOGENIC		NON-CARC	CARCINOGENIC		NON-CARC
	RISK	HZD INDX	HZD INDX	RISK	HZD INDX	HZD INDX
DM101	2E-05	4.9E-02	7.7E-02	8E-05	2.4E-01	1.2E-01
DM103	9E-03	2.1E-01	1.7E+01	5E-02	9.7E-01	2.9E+01
DM104	7E-05	9.0E-03	1.1E+01	2E-04	4.2E-02	1.1E+01
DM106	5E-06	7.2E-03	9.0E-02	3E-05	4.5E-02	1.6E-01
DM119	2E-06	2.6E-03	4.8E-03	8E-06	1.1E-02	8.8E-03
DM121	2E-05	1.1E-02	4.2E-01	1E-04	6.1E-02	9.8E-01
DM123	8E-07	2.4E-03	3.3E-03	3E-06	7.9E-03	3.6E-03
DM125	5E-06	1.3E-02	4.4E-02	2E-05	6.1E-02	7.0E-02
DM501	1E-06	4.6E-03	2.0E-02	4E-06	2.0E-02	3.6E-02
DM502	3E-06	1.2E-01	4.7E-01	1E-05	7.0E-01	7.8E-01
DM506	7E-06	2.5E-02	5.0E-01	4E-05	1.2E-01	8.4E-01
DM507	3E-05	1.1E-01	2.6E+00	1E-04	9.9E-01	3.4E+00
MP03	1E-01	2.3E+01	1.7E+04	9E-01	1.3E+02	3.0E+04
MP09	1E-01	4.6E+00	5.7E+01	6E-01	4.1E+01	1.7E+02
MP11	4E-04	9.1E-02	1.3E+01	1E-03	7.0E-01	1.6E+01
MP13	1E-04	6.2E-03	3.5E+00	9E-04	4.4E-02	5.3E+00
MP16	4E-04	1.9E-03	6.5E+00	3E-03	8.6E-03	1.3E+01
MP20	6E-04	2.4E-03	1.0E+01	5E-03	7.9E-03	2.3E+01
MP25	2E-04	2.4E-03	5.2E+00	8E-04	7.9E-03	5.4E+00
MP28	1E-04	2.4E-03	3.1E+00	9E-04	7.9E-03	4.1E+00
MP30	2E-04	2.4E-03	5.4E+00	2E-03	7.9E-03	1.4E+01
MP36	1E-02	1.6E+01	1.4E+03	8E-02	9.0E+01	2.4E+03
MP49	6E-04	1.3E-01	3.5E+01	1E-02	8.1E-01	1.1E+02
MP50	2E-04	2.4E-01	1.8E+01	1E-03	7.9E-01	3.4E+01
MP51	4E-05	6.2E-02	1.1E-01	7E-04	8.2E-01	4.3E-01
MP53	9E-07	1.8E-03	8.6E-03	3E-06	7.7E-03	1.5E-02

a. Calculations for total risk.

Total Risk (Ingestion, Inhalation, Dermal) = Ingestion risk (Inorg) + (Ingestion risk (org) x 2)

Total CHI (Ingestion, Inhalation, Dermal) = Ingestion CHI (inorg) + (Ingestion CHI (org) x 2)

Total HI (Ingestion, Inhalation, Dermal) = Ingestion HI (inorg) + (Ingestion HI (org) x 2)

The contribution for inorganic chemicals of concern is only included for ingestion calculations.

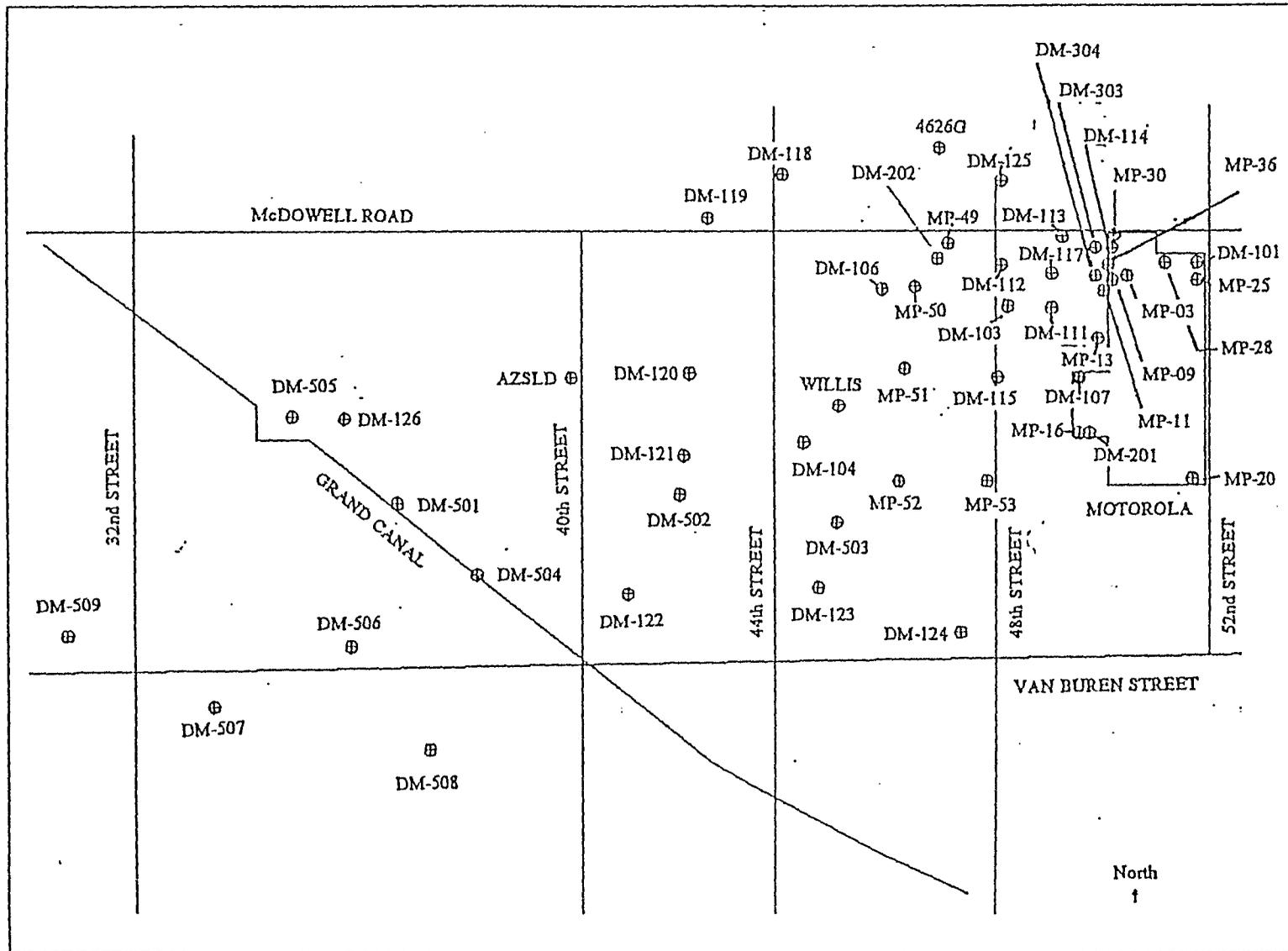


FIGURE 5.1 - Groundwater Sampling Sites for Motorola 52nd Street Study Area.

Table 5.4 - Contribution to total potential average alluvial risk by chemical for each well.

WELL	ARSENIC	BENZENE	BROMODICHLORO METHANE	CARBON TETRA CHLORIDE	CHLOROFORM	1,2-DICHLORO ETHANE	DICHLORO METHANE	TETRACHLORO ETHYLENE	TRICHLORO ETHYLENE	VINYL CHLORIDE
4626G	99.2									0.7
AZSLD	99.1								0.1	0.7
DM103			0.1	0.1					0.1	99.6
DM104	99.2							0.2	0.3	0.2
DM106			1.3	1.7		0.3	0.2	0.2	57	39.2
DM107	98.4		0.1						1.1	0.3
DM111	88.6		2.1	0.1	0.1			4.1	1.7	3.1
DM112	79.9		0.3	0.3		0.4	0.2	3.5	11.3	4.1
DM113	88.1							0.1	10.7	0.9
DM114	99.5							0.1	0.2	0.1
DM115	93.1		0.1	0.1		0.1	0.1	2.1	2.8	1.6
DM117	26.7								0.1	73
DM118	99							0.2	0.2	0.5
DM119			2.9	5.9	1	1.4	1.2	0.8	0.5	86.2
DM120	93.8		0.1	0.1		0.1		0.2	3.7	1.9
DM121			1.5	1.5	0.1	0.7	0.5	11.8	39.1	44.7
DM122	99.9									
DM123			4	4	0.2	2.8	28	1.6	0.3	59
DM124	99.4							0.2	0.2	0.1
DM125			3.6	5.3	2.5	2.1	5.5	1.2	2	77.8
DM126			2.8	5.5	0.1	1.2	0.8	4.2	5.1	80.4
DM201	58.6		1.1	1.1	0.1	2.3	0.6	19.9	0.3	16
DM202	55.8		0.8	0.8	0.1	0.5	0.4	0.8	23.4	12.3
DM303	81.1		0.4	0.4		0.3	0.2	2.6	8.8	6.1
DM304	85.4		0.2	0.2		0.4	0.1	2.8	7.6	3.3
DM501			1.2	1.2	0.2	1.7	0.7	4.2	38	52.8
DM502			1	1		2.9	0.6	24.2	55	15.2
DM503	99.3		0.1					0.1	0.2	0.2
DM504	48.8	0.5	1.6	0.8		0.6	0.5	9.2	26.3	11.7
DM505	94.9		0.2	0.2	0.2	0.2	0.1	0.4	0.5	3.3
DM506			4.7	0.6	1.7	0.4	0.3	29.7	53.9	8.7
DM507			2.2	2.2	0.3	1.5	1.3	6.1	54.6	31.9
DM508	99.1		0.4		0.1			0.1		0.3
DM509	80.2	0.2	1.6	0.1	0.2	0.1	0.1	0.1	16.2	1.3
MP03	6.5		3.5	3.2	0.1	7.5	1.4	2.8	22	53.1
MP09	29.4		1.1	1.1		0.6	0.5	17.2	33.3	16.9
MP11	96.1							2.7	0.4	0.6
MP13	77.6		0.3	0.3		0.2	0.1	1.4	16.2	4
MP16	93.6		0.1					5.3	0.4	0.6
MP20	99.6							0.1		0.2
MP28	93.5	0.6	0.1	0.1		0.1	0.1	0.6	3.2	1.6
MP30	99.8								0.1	0.1
MP36	78.4		0.3	0.4		0.2	0.2	3.2	10.2	7
MP49	32		0.8	1.2	0.1	0.6	0.7	1.1	45.6	18.1
MP50	79		0.6	0.6	0.1	0.9	0.3	0.5	9.4	8.6
MP51	76.5		0.3	0.4		0.2	0.2	1.2	14.4	6.8
MP52	99.3									0.6
MP53	99.9									0.1
WILLIS	50.7		0.8	0.8		0.5	0.5	4.6	30.6	11.5

Note: Well percentage totals have a rounding error of + or - 0.2%

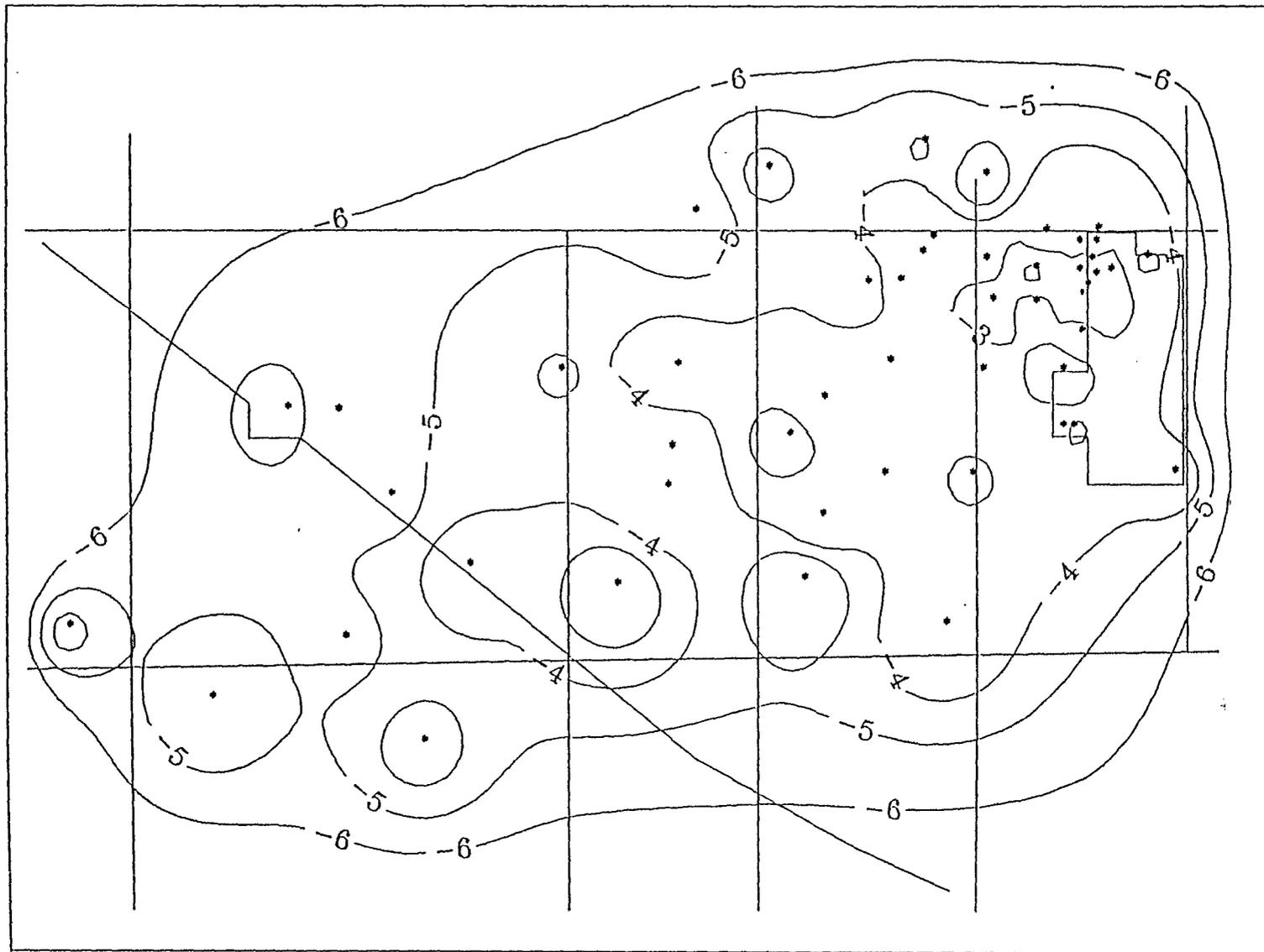
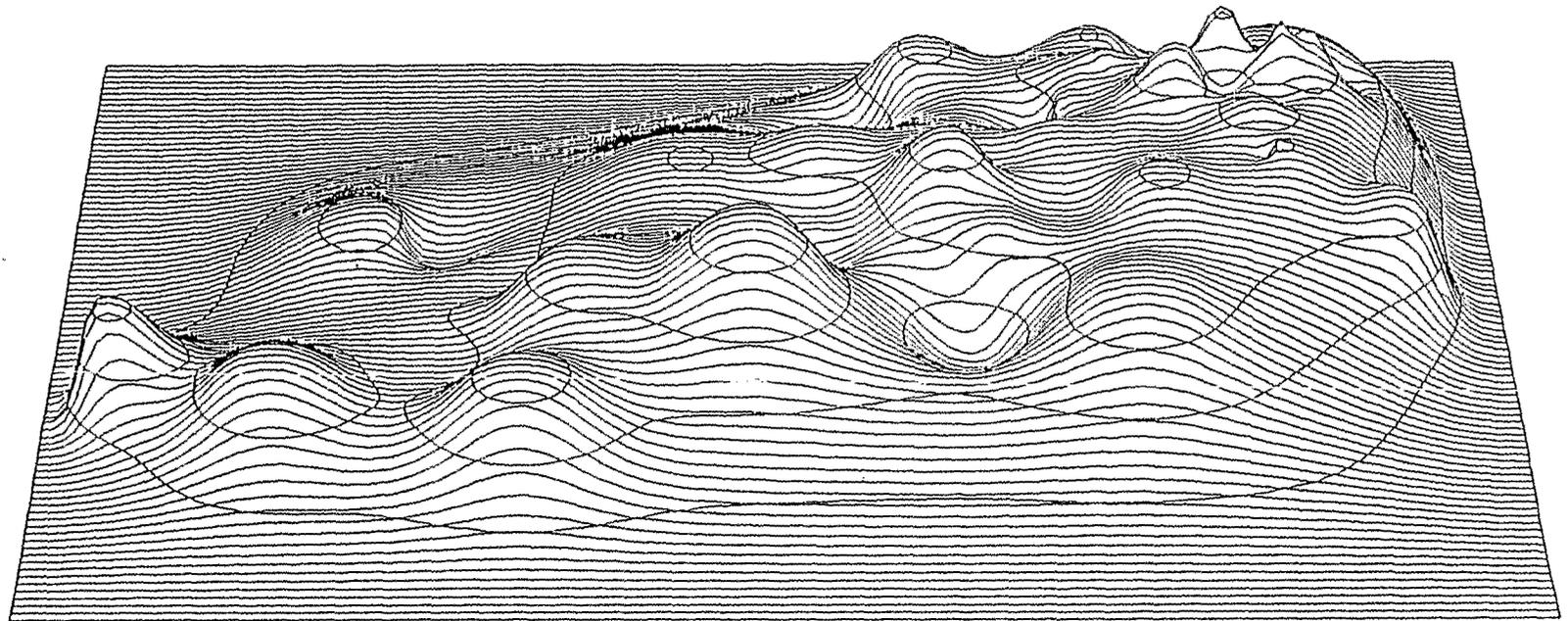


FIGURE 5.2 - Contour Map, Log10(Potential Total AVERAGE Risk), Alluvial Wells



3-D Surface Map

Log₁₀(Total AVERAGE Risk)

0
-1
-2
-3
-4
-5
-6

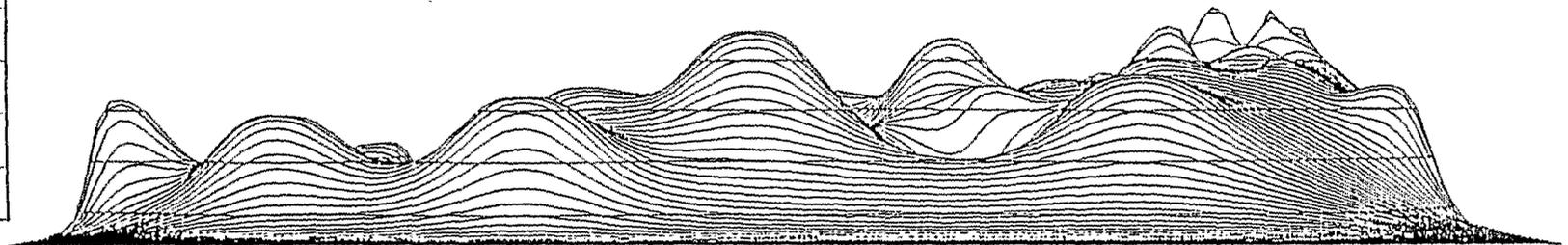


FIGURE 5.3 - SURFACE MAPS, Log₁₀(Potential Total AVERAGE Risk), Alluvial Wells

Arsenic is responsible for over 99% of the ELCR in 12 wells and for over 70% of the ELCR in 27 wells (Table 5.4). Vinyl chloride was responsible for more than 50% of the ELCR in six (6) wells. TCE contributed more than 25% of the ELCR in ten wells, with four of them over 50%. There was only one well in which PCE was responsible for more than 25% of the risk.

Potential RME Risk

The RME potential ELCR for alluvial groundwater was almost an order of magnitude greater than the average exposure estimates and followed a similar distribution over the study area. Potential risks are shown in Table 5.3 and graphically represented in Figures 5.4 and 5.5. Potential RME risk varied from a maximum of $7E-02$ at well MP11 to a minimum of $8E-06$ at DM119.

Arsenic dominated the risk estimates in a majority of the wells (Table 5.5), as with calculations for the average exposure. Arsenic was followed by VC, TCE and PCE in declining order of percent contribution to total ELCR by well.

The slope factor used for the estimation of ELCR due to arsenic has not been officially accepted by the USEPA for use in IRIS or HEAST. The slope factor is derived from a unit risk of $5E-05$ per $\mu\text{g/L}$ based on a Taiwanese study by Tseng (1977). It has been recommended for use with the caution that there is a high degree of uncertainty inherent in the estimate and that it may be revised downward by as much as an order of magnitude in the future. The MCL for arsenic in public drinking supplies is currently $50 \mu\text{g/L}$, which yields an ELCR of $2E-04$. A recent article by Smith et al. (1992) states that the ELCR may be as high as $2.6E-02$ at the MCL.

There was very little background data that could be used to determine if the arsenic concentrations were a result of activities at the Motorola 52nd Street site. However, when the mean concentrations for wells in the study area are plotted as in Figure 5.6, there appears to be some elevated concentrations in the vicinity of the Motorola 52nd Street facility.

At many of the wells much of the risk was due to vinyl chloride (VC) (Table 5.5 and Appendix Table 27). There are three reasons for this. The slope factor for VC is very high, therefore, low concentrations give high risk values. For example, TCE must be present at about 100 times the concentration of VC to have an equal risk estimate. In wells with low potential ELCR estimates, VC was often reported at the SQL, these values were included in the assessment because VC was detected in other wells. Finally, some wells had high concentrations of VC; these were the wells that show the highest potential carcinogenic risk for domestic use. They were on-site well MP09, and off-site wells DM103 and

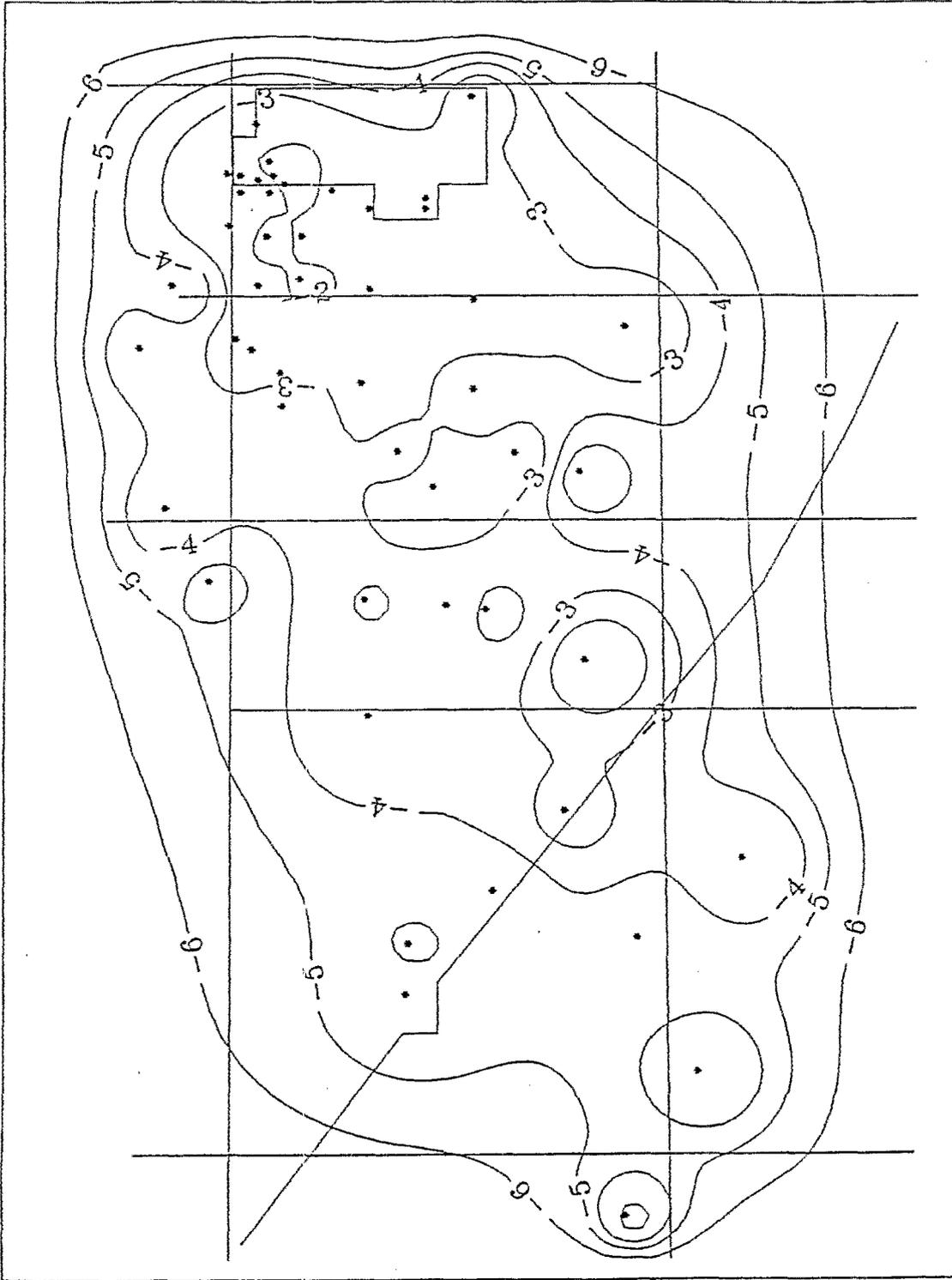
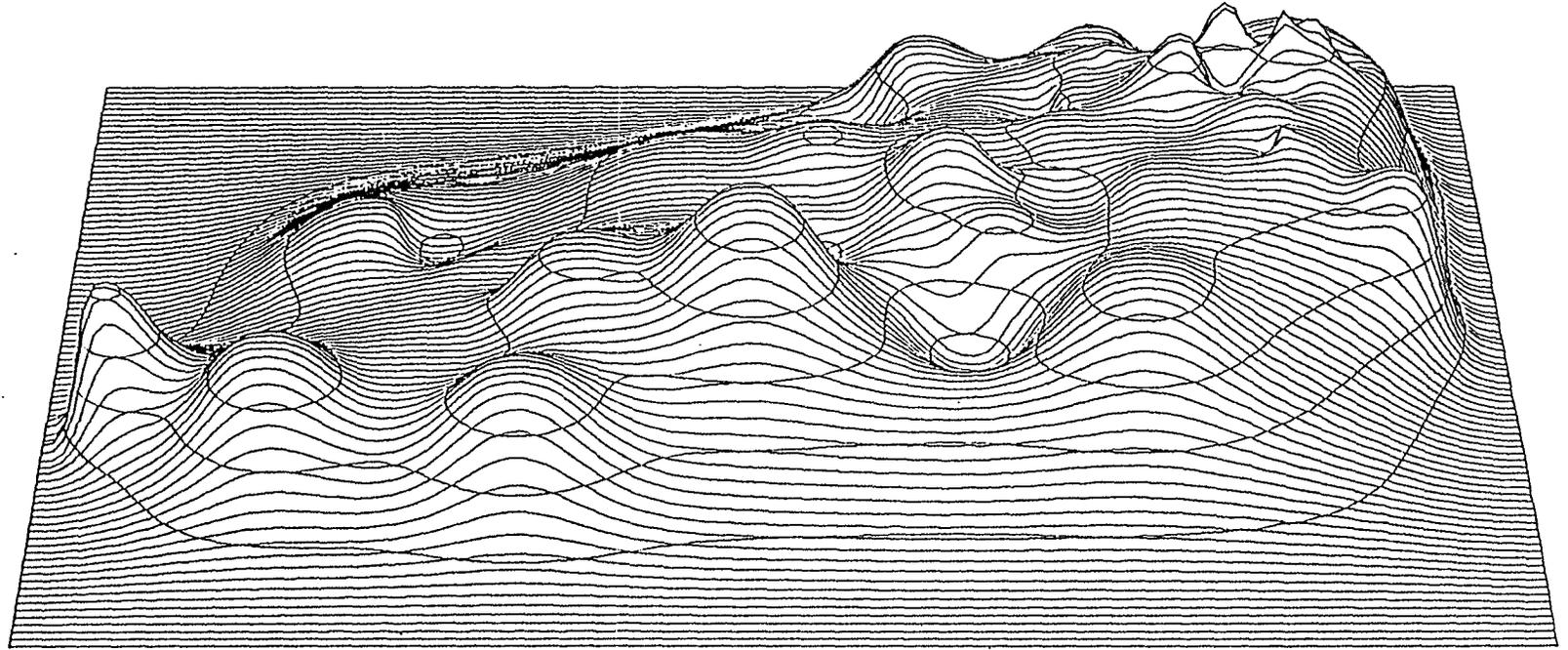


FIGURE 5.4 - Contour Map, Log10(Potential Total RME Risk), Alluvial Wells



3-D Surface Map

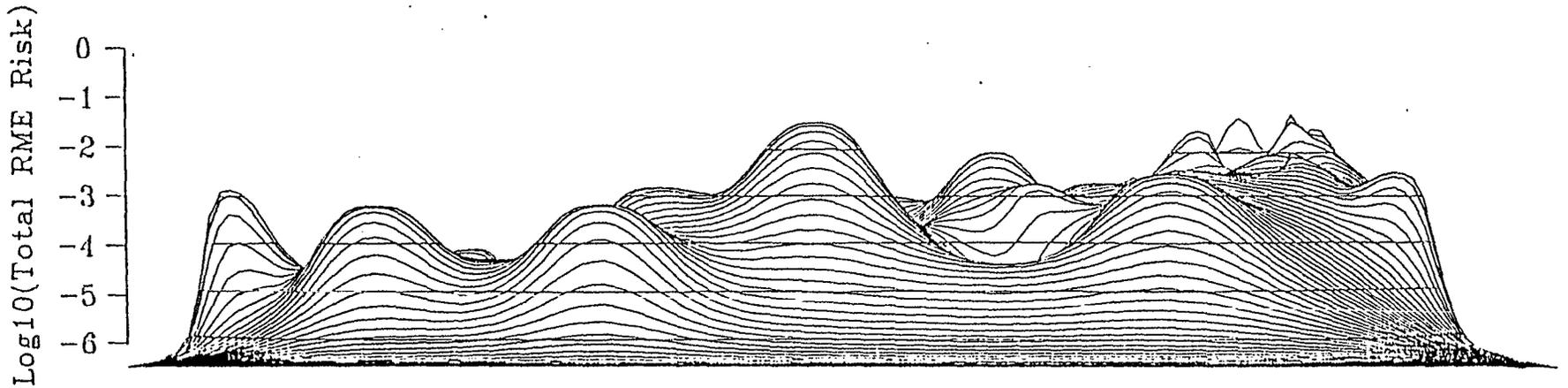
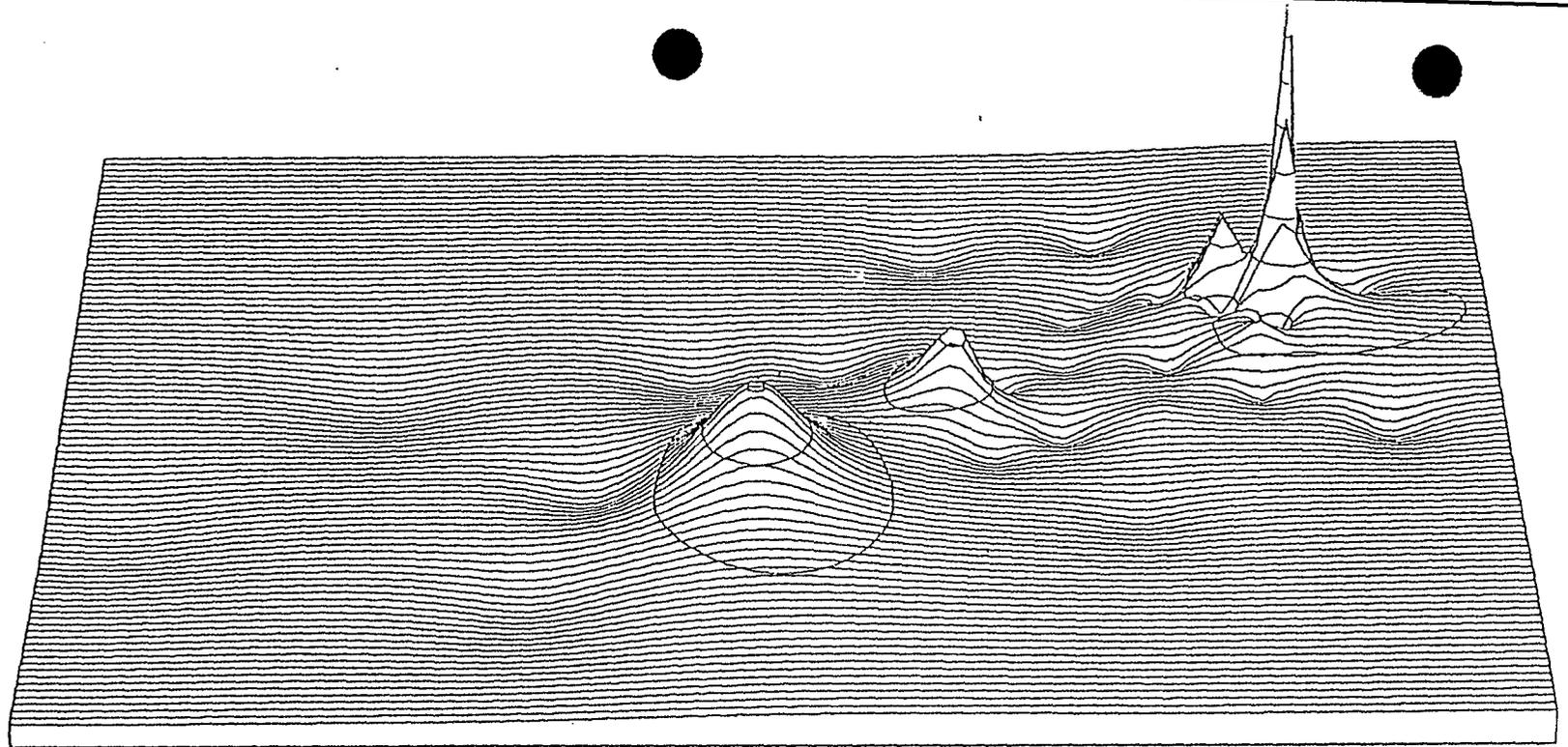


FIGURE 5.5 - SURFACE MAPS, $\text{Log}_{10}(\text{Potential Total RME Risk})$, Alluvial Wells

Table 5.5 - Contribution to total potential RME alluvial risk by chemical for each well.

WELL	ARSENIC	BENZENE	BROMODICHLOR METHANE	CARBON TETRA CHLORIDE	CHLOROFORM	1,2-DICHLORO ETHANE	DICHLORO METHANE	TETRACHLORO ETHYLENE	TRICHLORO ETHYLENE	VINYL CHLORIDE
4626G	98.7			0.1						1
AZSLD	98.9								0.2	0.7
DM103				0.1					0.1	99.7
DM104	98.7							0.5	0.6	0.2
DM106			1.2	1.5		0.3	0.2	0.2	58.2	38.4
DM107	97.8		0.1						1.5	0.5
DM111	79.4		4	0.2	0.3	0.1		7.3	3.2	5.6
DM112	58.8		1.6	1.6	0.1	1.5	0.9	3.3	8.9	23.3
DM113	65.8		0.1	0.1				0.3	31.1	2.6
DM114	99.1		0.1					0.4	0.4	0.1
DM115	89.1		0.2	0.2	0.1	0.1	0.1	4	3.8	2.5
DM117	23.6			0.1					0.1	76.1
DM118	98.3							0.5	0.7	0.4
DM119			2	5.9	1.9	1.4	1.1	0.8	0.5	86.5
DM120	93.6		0.1	0.1		0.1	0.1	0.3	3.6	2.1
DM121			1.5	1.5		0.5	0.4	17.9	46.3	32
DM122	100									
DM123			4	4	0.2	2.8	2.8	1.6	0.3	59
DM124	99.1							0.2	0.5	0.1
DM125			4.1	5.1	3.8	2.8	6.2	1.6	2.1	74.3
DM126			1.8	5.3	0.1	1.1	0.8	7.1	6.3	77.5
DM201	72.9		0.3	0.8		1.7	0.5	11.6	0.2	11.6
DM202	45.7		1.1	1.1	0.1	0.7	0.6	1.1	33.5	16.1
DM303	74.5		0.8	0.8		0.6	0.5	2.6	8.7	11.6
DM304	77.6		0.3	0.3		0.5	0.2	3.6	13	4.5
DM501			1	1	0.2	1.4	0.6	3.7	32.7	59.4
DM502			1.1	1.1	0.1	3.1	0.6	23	55	16.1
DM503	99.4		0.1					0.2	0.2	0.1
DM504	57.7	0.3	1.6	0.9		0.6	0.5	6.9	18.5	13
DM505	93.8		0.2	0.2	0.8	0.2	0.1	0.5	0.8	3.3
DM506			10.6	0.3	2.5	0.2	0.2	2.8	53.3	4.8
DM507			2.5	2.5	0.3	1.7	1.4	4.2	50.7	36.5
DM508	98.5		0.9		0.1			0.1		0.3
DM509	88.7	0.1	1	0.1	0.1	0.1	0.1	0.1	8.3	1.5
MP03	3.5		3.6	3.3	0.2	7.3	1.7	2.7	27.2	50.6
MP09	39.5		0.7	0.7		0.5	0.4	15.8	31.3	11
MP11	96.5							2.4	0.4	0.7
MP13	69.8		0.3	0.3		0.2	0.2	2.4	22.2	4.6
MP16	90.4		0.1					8.2	0.4	0.8
MP20	99.8							0.1		0.1
MP23	97.4	0.1	0.1	0.1				0.2	1.3	0.9
MP30	99.9								0.1	
MP36	75.2		0.5	0.5		0.3	0.2	3.9	11.4	8.1
MP49	25		1.1	1.4	0.1	0.8	0.8	1.3	4.8	21.4
MP50	76.9		0.7	0.7	0.2	1	0.4	0.6	9.5	10.1
MP51	68.1		0.6	0.6	0.1	0.4	0.3	1.5	18.3	10.1
MP52	99.3									0.6
MP53	99.9									0.1
WILLIS	42.6		0.7	0.7		0.5	0.4	8.4	37.2	9.6

Note: Well percentage totals have a rounding error of + or - 0.2%



3-D Surface Map

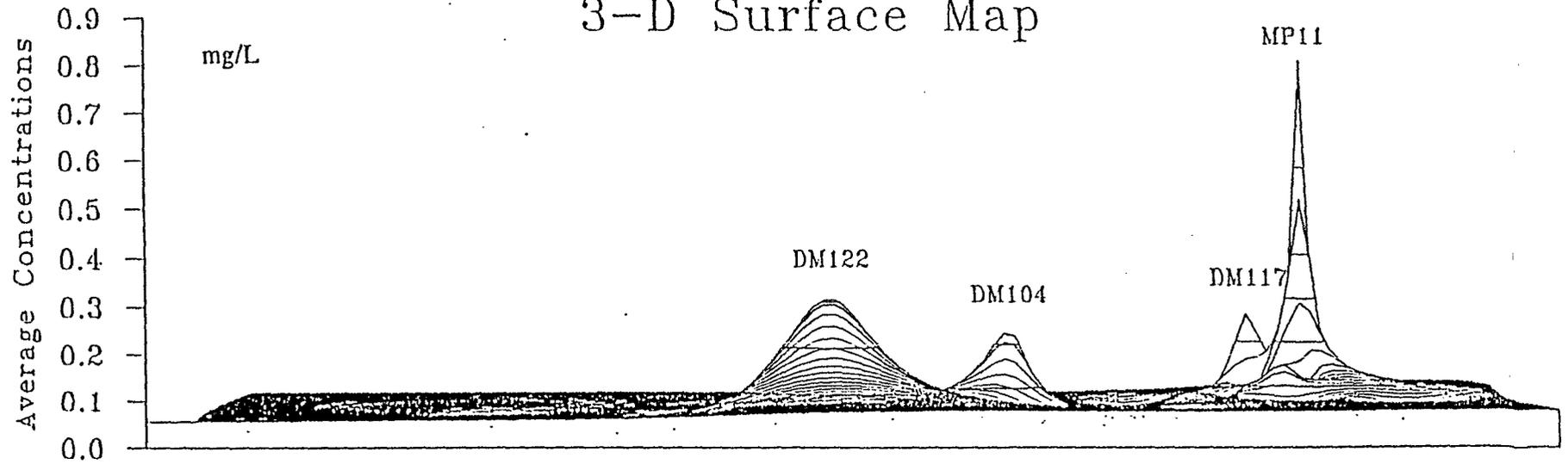


FIGURE 5.6 - SURFACE MAPS, Estimated Alluvial Arsenic AVERAGE Concentrations

DM117. Table 5.6 shows that six (6) alluvial and bedrock sampling locations at which VC was positively detected. Figure 5.7 represents the average potential ELCR due only to VC, the alluvial wells with a positive detections are labeled. The other areas of high potential ELCR are due to the use of one half the values of the SQLs in the computations. Vinyl chloride may or may not be present at these locations.

The other two most frequent sources of potential carcinogenic risk were tetrachloroethylene (PCE) and trichloroethylene (TCE). Their distribution in the alluvial groundwater followed the distribution of potential risk as represented on Figures 5.2 through 5.5.

Cancer Hazard Index

The potential cancer hazard quotients (CHQ) were calculated for chloromethane, dibromochloromethane, 1,4-dichlorobenzene, 1,1-DCA, 1,1-DCE and 1,1,2-TCA. Nine (9) alluvial wells were found to have CHI greater than one (Tables 5.4 and 5.5). The highest was 120 for on-site well MP03. There were seven wells in which the elevated CHI was due to 1,1-DCE. These were on-site-wells, MP03, MP09, and MP36 in the courtyard area, and, DM201 in the SWPL area; as well as off-site wells DM303, DM304, located along 50th Street; DM112, at 48th Street; and MP49, near the Crosscut Canal.

The RME potential CHI followed a similar pattern as the average CHI, but with 21 wells above the 1.0 level. Well MP03 was the highest at 790, with well DM201 at 500. Once again 1,1-DCE was a major contributor to the CHI. The occurrence of 1,1-DCE was widespread, probably as a result of the degradation of other chlorinated compounds.

Potential Noncarcinogenic Hazard Index

The potential noncarcinogenic chronic hazard index (HI) for average exposures was distributed similarly (Tables 5.4 and 5.5). The maximum average exposure potential HI was 370 for well MP03; 43 other wells had potential HI over 1.0. Major contributors were arsenic, fluoride, thallium, 1,1-DCE, 1,1,1-TCA, and TCE. Species with HI greater than one are listed in Table 5.7.

The RME potential HI was above 1.0 for 43 wells in the sampling area. All on-site wells had HI above 1.0. Only wells DM119, DM123, DM125, DM126, DM501, and DM506 had HI below 1.0. Again the wells with the highest values for HI were MP03 (820) and DM201 (160).

Table 5.6 – Vinyl chloride detections in the study area. ($\mu\text{g/L}$)

	Well	Detections	Range
Alluvial	DM103	8	20-1300
	DM117	7	720-1700
	DM501	1	2
Bedrock	DM103	20	70-4300
	DM121	1	1.4
	MP09	4	6000-20000

Figure 5.7 – Potential average excess lifetime cancer risk resulting from potential domestic ingestion, inhalation, and dermal exposures to vinyl chloride in groundwater.

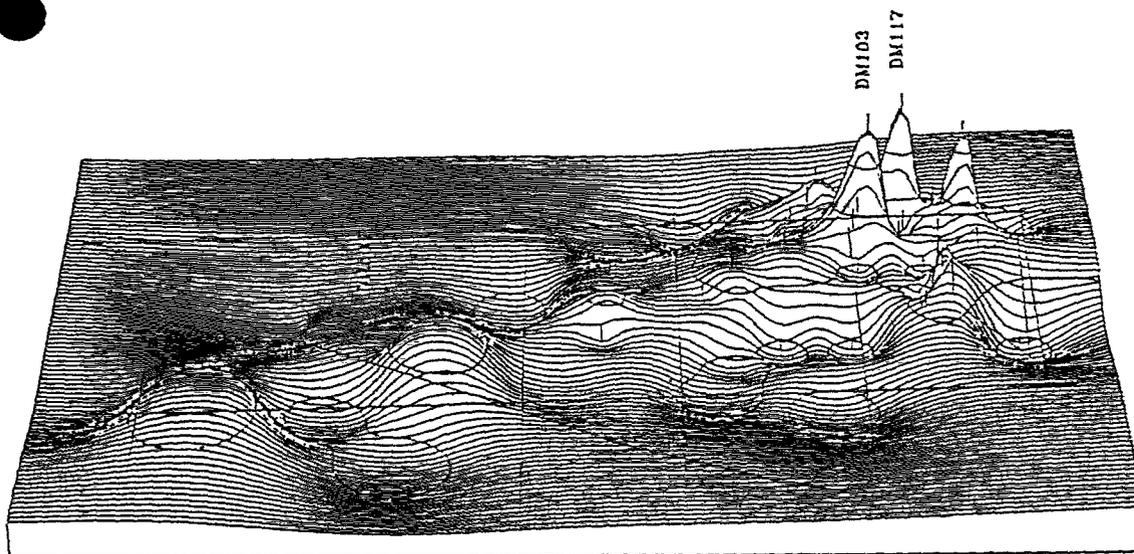


Table 5.9. -- Reasonable maximum risk estimates and hazard quotients for domestic ingestion of groundwater, well 2646G.

RISK ESTIMATES FOR REASONABLE MAXIMUM EXPOSURE (RME)									
Summary of Potential Exposure Concentrations & Health Risk Calculations for Alluvial Groundwater Ingestion.									
CONTAMINANT	EC 95% UCL (ug/L)	INTAKE CONSTANT		TOXICITY FACTORS - ORAL		INGESTION - GROUNDWATER			
		Cancer 1.2e-05	Noncancer 2.7e-05	SF (mg/kg/d) ¹	Rfd (mg/kg/d)	Cancer CDI (mg/kg/d)	Cancer Risk	N/C CDI (mg/kg/d)	Hazard Quotient
Arsenic (As)	8			1.8e+00	3.0e-04	9.4e-05	1.7e-04	2.2e-04	7.3e-01
Boron (B)	1100				9.0e-02	1.3e-02		3.0e-02	3.3e-01
Chloroform	0.5			6.1e-03	1.0e-02	5.9e-06	3.6e-08	1.4e-05	1.4e-03
Fluoride (F)	433				6.0e-02	5.1e-03		1.2e-02	2.0e-01
Lead (Pb)	65			NR	NR	NR	NR	NR	NR
Trichloroethylene	0.7			1.1e-02	6.0e-03	8.2e-06	9.0e-08	1.9e-05	3.2e-03
Total Cancer Risk							1.7e-04		
Hazard Index									1.3e+00
EXPOSURE ASSUMPTIONS:	Carcinogenic Intake Dose = (ED x EF x CR x WCF)/(BW x AT x TCF)								
	NonCarcinogenic Intake Dose = (ED x EF x CR x WCF)/(ED x BW x TCF)								
ED: Exposure Duration (years)	30			Constant x EC = Chronic daily intake (CDI)					
EF: Exposure Frequency (days/year)	350			EC = Exposure Concentration					
CR: Contact Rate (L/day)	2								
WCF: Weight Conversion Factor (mg/ug)	0.001								
BW: Body Weight (kg)	70								
AT: Average Time for cancer (years)	70								
TCF: Time Conversion Factor (days/year)	365								

after the well has been pumping for a representative period of time. The well is located near areas of higher levels of contamination and could be impacted to a greater degree in the future.

5.2.2 The Alluvium-Bedrock Interface

A separate assessment was performed for samples taken at or near the alluvium-bedrock interface (Appendix Table 27, Tables 5.2 and 5.3). The highest exposure concentrations and risk levels occur in this group of wells. The three on-site wells, MP03, MP09, and MP36 have the highest potential risk estimates for combined domestic use. The maximum is $9E-01$ for the RME potential risk for well MP03. This means 9 out of ten people using the water for 30 years might develop cancer as a result. At high levels of risk a one-hit exponential model may be used to estimate carcinogenic risk (USEPA 1989). In this case the use of the exponential model increases the maximum potential risk to $1E+00$.

Off-site wells that have high potential risks include DM103, DM104, DM121, DM507, MP11, MP49, MP50, and MP51. Well MP507 is one of the most distant monitor wells from the 52nd Street facility, other potential sources have been identified that may contribute to the contamination. The potential average and RME risks associated with domestic use of groundwater from the alluvium-bedrock interface is graphically shown in Figures 5.8 through 5.11.

5.2.3 Soil Gas

Extensive soil gas sampling on the Motorola site and the surrounding area was performed in 1984 and 1985. On-site sampling was also done in 1989 and 1991. Off-site sampling was performed in 1992. These data were presented in Chapter 2. Carcinogenic risk and chronic, systemic hazard was characterized for residential exposures for the 1984 and 1985 data. Occupational exposures were characterized for on-site sampling locations for the 1984, 1985, 1989, and 1991 data. Methods used to model releases of volatile compounds to outdoor and indoor air were discussed fully in Chapter 3. Exposure concentrations were calculated and shown in Tables 3.4 through 3.9. Chronic daily intakes are shown in Appendix Tables 12 through 26.

Residential, 1984-1985 Data

Both average and RME outdoor residential carcinogenic risk, associated with soil gas exposure as modeled from reported sampling results, were below the negligible risk level (Table 5.10). A maximum RME ELCR of $2E-08$ occurred at sampling point 2143. This sampling point is to the west of the site near the Old Crosscut Canal (Appendix Figure 2). Outdoor concentrations of VOCs released

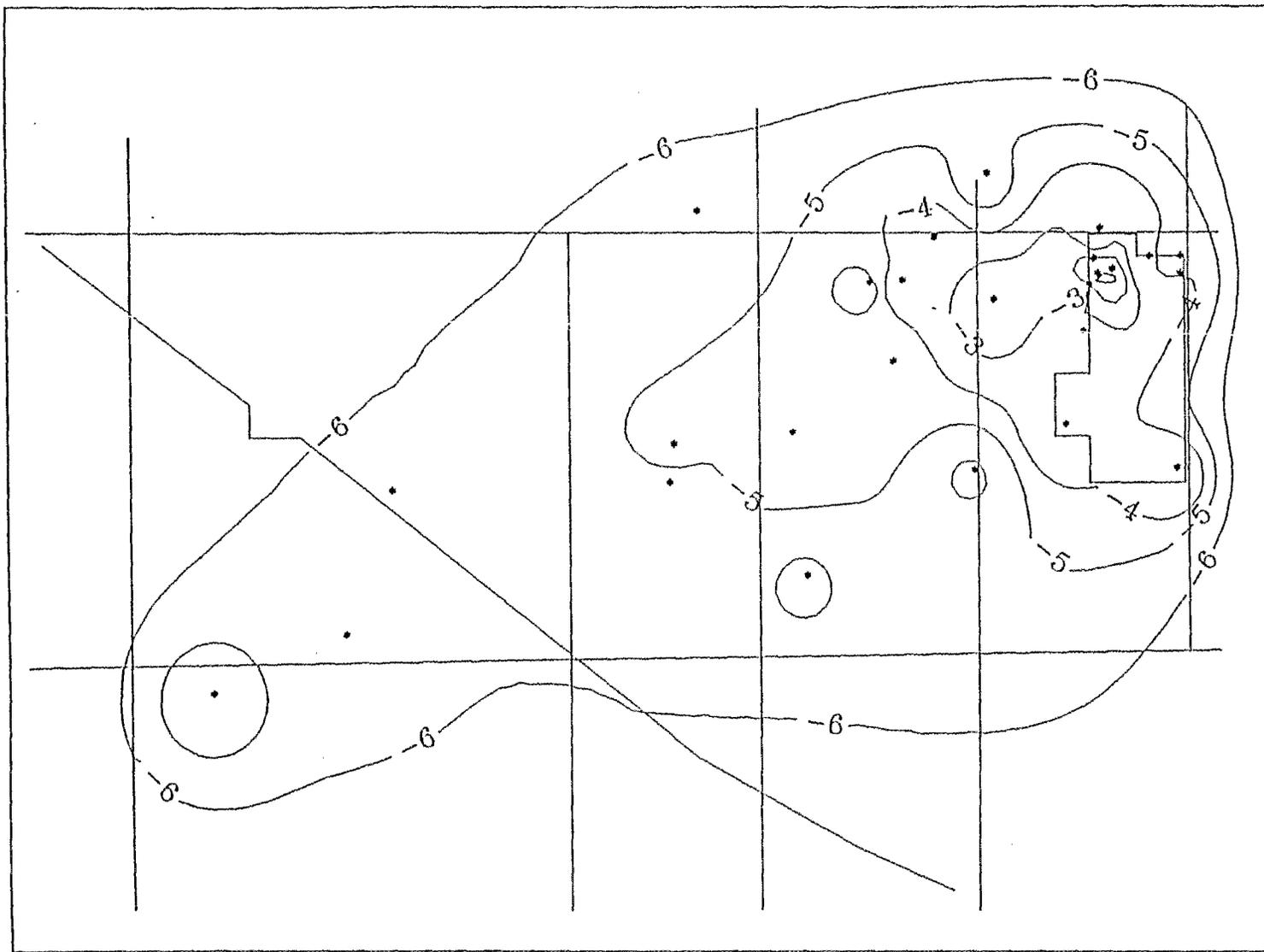
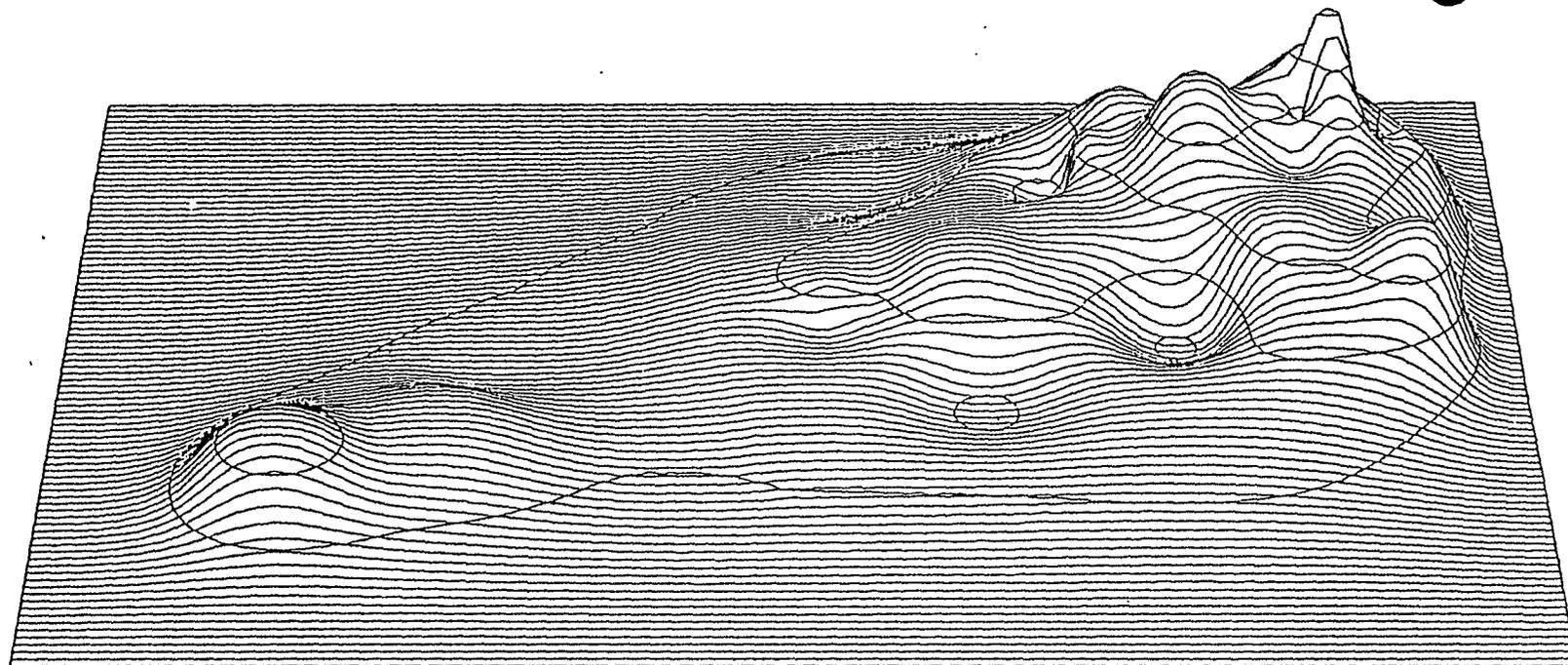


FIGURE 5.8 - Contour Map, Log10(Potential Total AVERAGE Risk), Bedrock Wells



3-D Surface Map

Log10(Total AVERAGE Risk)

0
-1
-2
-3
-4
-5
-6

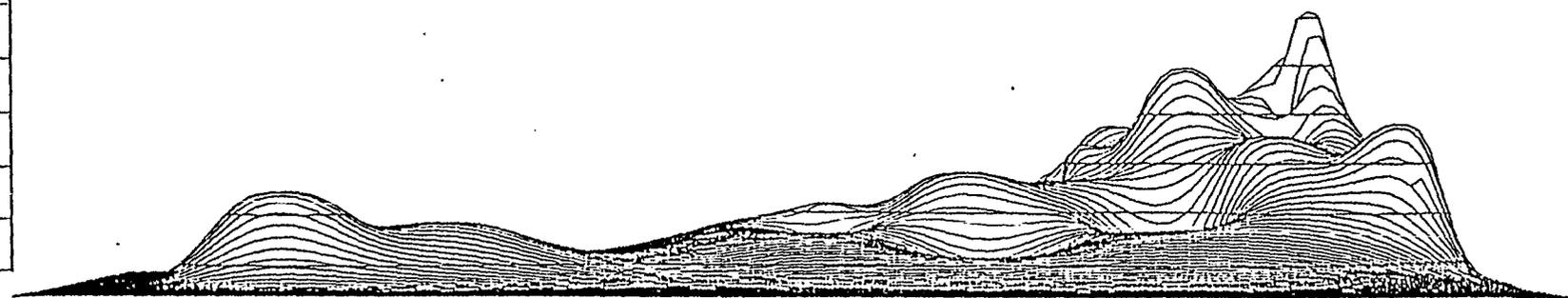


FIGURE 5.9 - SURFACE MAPS, Log10(Potential Total AVERAGE Risk), Bedrock Wells

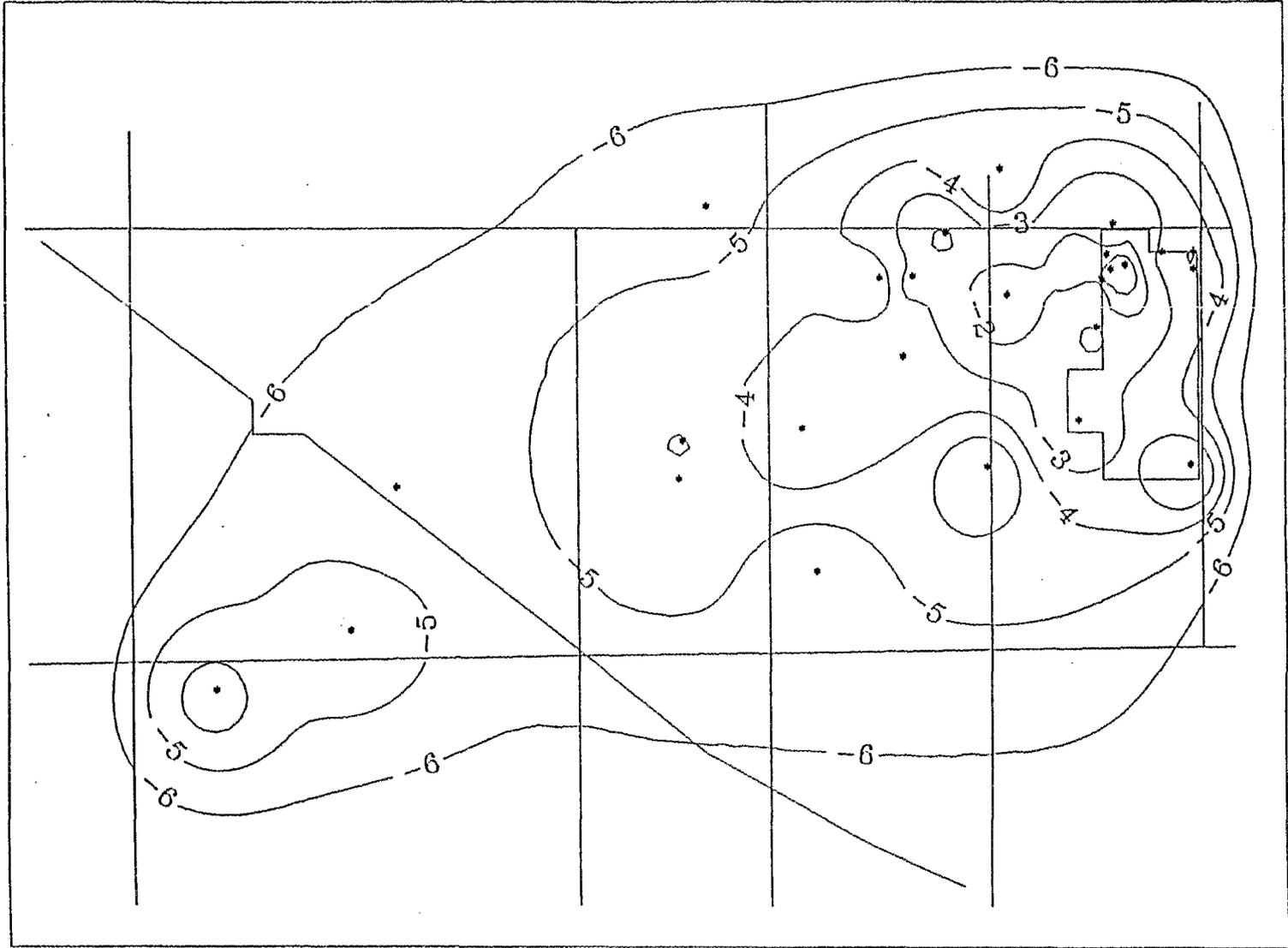
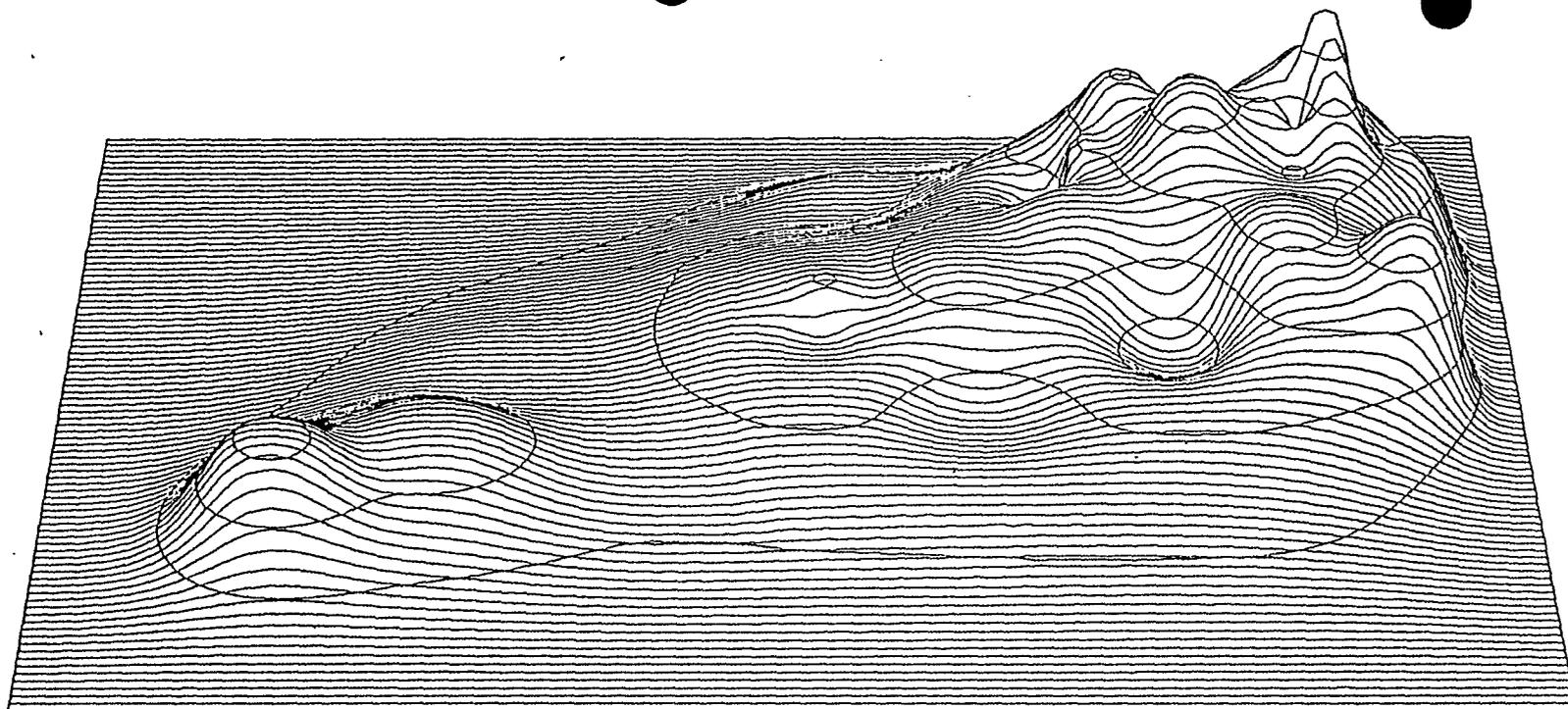


FIGURE 5.10 - Contour Map, Log₁₀(Potential Total RME Risk), Bedrock Wells



3-D Surface Map

Log10(Total RME Risk)

0
-1
-2
-3
-4
-5
-6

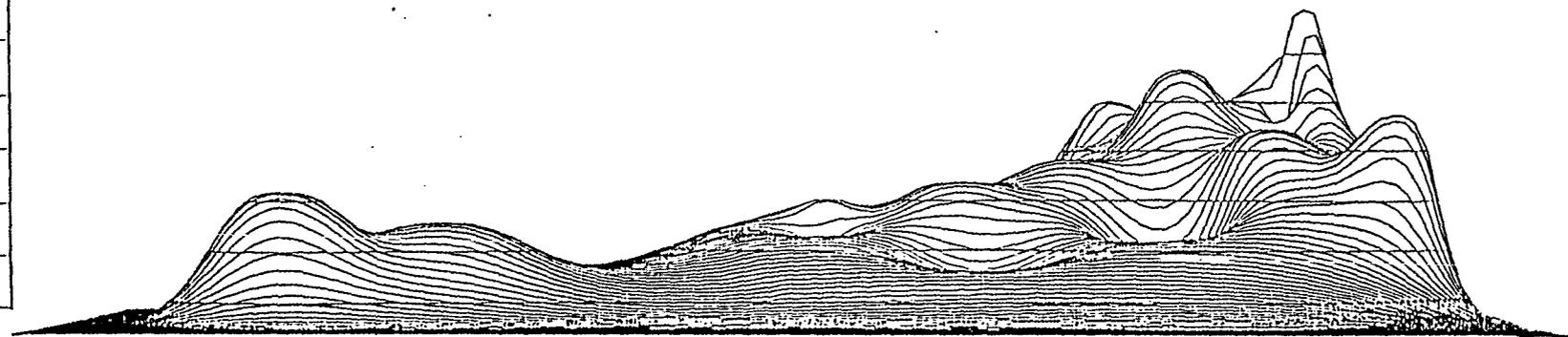


FIGURE 5.11 - SURFACE MAPS, Log10(Potential Total RME Risk), Bedrock Wells

Table 5.10 – Summary of carcinogenic risk from residential exposures to soil gas, 1984 - 1985.¹

LOCATION	OUTDOOR		INDOOR	
	AVG	RME	AVG	RME
1030	5e-10	7e-09	4e-08	2e-07
1031	5e-11	7e-10	4e-09	2e-08
1040	2e-11	3e-11	2e-09	9e-09
1094	4e-12	5e-11	3e-10	1e-09
2043	1e-09	2e-08	1e-07	5e-07
2045	3e-10	4e-09	3e-08	1e-07
2054	1e-10	1e-09	8e-09	4e-08
2055	4e-10	6e-09	4e-08	2e-07
2056	8e-11	1e-09	6e-09	3e-08
2057	3e-12	4e-11	3e-10	1e-09
2069	9e-11	1e-09	7e-09	4e-08
2072	3e-10	4e-09	2e-08	1e-07
2087	1e-10	2e-09	1e-08	5e-08
2088	2e-10	2e-09	1e-08	7e-08
2089	3e-11	4e-10	2e-09	1e-08
2090	2e-10	2e-09	1e-08	7e-08
2114	2e-11	3e-10	2e-09	1e-08
2120	2e-11	3e-10	2e-09	9e-09
2130	2e-10	3e-09	2e-08	9e-08

1. No location had a risk greater than the 1E-06 (one-in-one-million) level of concern.

from soil gas do not constitute a carcinogenic health hazard, based on levels detected in the 1984 and 1985 sampling data.

Indoor residential ELCR estimates were also below the negligible risk level ($1E-06$). The maximum for RME of $5E-07$ was determined at location 2143. On the basis of this risk assessment there does not appear to be a significant amount of risk associated with residential exposures to soil gas.

Chronic systemic hazard indices (HI) for both outdoor and indoor residential exposures were well below 1.0 for all sampling sites (Table 5.11). There is no indication from this assessment that residents to the west of the site are at risk of suffering chronic, systemic effects due to soil gas releases.

Occupational, 1984-1985 Data

Occupational risks were characterized for 1984 and 1985 on-site sampling locations (Table 5.12). Outdoor risks were very low, with only on-site location, 2133 (see Appendix Figure 2), having an ELCR for RME higher than one-in-a-billion ($1E-09$). Estimated indoor risks are higher but only location 2133 had an ELCR above the one-in-a-million ($1E-06$) level. ELCR due to average exposure was $4E-06$ and the RME maximum risk was $2E-05$.

Hazard indices were all below the 1.0 level of concern (Table 5.13). Predicted occupational soil gas exposures should not produce adverse chronic, systemic health effects.

Occupational, 1989 Data

Outdoor and indoor risk estimates for occupational exposures to soil gas emissions modeled from 1989 on-site sampling data are all below one-in-a-billion ($1E-09$) and not a cause for concern (Table 5.14). Air samples taken at the same time give higher risk levels, with one RME estimate at the $1E-06$ level. The higher risk estimates for the air sampling data may be due to other sources than soil gas for the compounds detected. VOCs are used in the manufacturing processes at the site and detectable concentrations are expected near the facility.

The HI for occupational exposures to 1989 concentrations of soil gas emissions and sampled air concentrations are all below 1.0 (Table 5.15). The exposure concentrations modeled and detected for the site are unlikely to produce chronic systemic effects.

Occupational, 1991 Data

Carcinogenic risk estimates characterized from the 1991 soil gas data are below the level of concern for both outdoor and indoor exposures (Table 5.16). The CHQ for 1,1-DCE also indicates that

Table 5.11 - Summary of noncarcinogenic hazard indices from residential exposures to soil gas, 1984 - 1985.¹

LOCATION	OUTDOOR		INDOOR	
	AVG	RME	AVG	RME
1030	5e-05	2e-04	1e-03	1e-03
1031	1e-05	4e-05	7e-04	1e-03
1040	1e-05	5e-05	1e-03	1e-03
1094	2e-06	9e-06	2e-04	3e-04
2043	1e-04	5e-04	3e-03	4e-03
2045	3e-05	1e-04	9e-04	1e-03
2054	7e-06	3e-05	2e-05	3e-05
2055	8e-05	3e-04	5e-03	7e-03
2056	1e-05	5e-05	7e-04	1e-03
2057	1e-06	5e-06	1e-04	1e-04
2069	3e-05	1e-04	3e-03	4e-03
2072	2e-05	9e-05	1e-06	2e-06
2087	6e-05	2e-04	5e-03	7e-03
2088	8e-05	3e-04	7e-03	1e-02
2089	1e-05	5e-05	1e-03	1e-03
2090	2e-05	7e-05	4e-04	6e-04
2114	9e-06	4e-05	7e-04	1e-03
2120	7e-06	3e-05	5e-04	8e-04
2130	2e-05	1e-04	7e-04	1e-03

1. No location had a hazard index above one (1.0), indicating that no chronic systemic effects are expected due to occupational exposures.

Table 5.12 - Summary of carcinogenic risk from occupational exposures to on-site soil gas, 1984 - 1985.

LOCATION	OUTDOOR		INDOOR	
	AVG	RME	AVG	RME
1021	1e-12	9e-12	5e-10	4e-09
1023	6e-13	4e-12	3e-10	2e-09
1024	3e-13	2e-12	1e-10	7e-10
1100	1e-12	6e-12	4e-10	3e-09
2122	2e-12	1e-11	8e-10	5e-09
2123	3e-11	2e-10	1e-08	9e-08
2127	7e-12	5e-11	3e-09	2e-08
2128	8e-11	6e-10	3e-08	2e-07
2131	7e-12	4e-11	3e-09	2e-08
2132	3e-09	2e-08	1e-06*	7e-06*
2133	9e-09	6e-08	4e-06*	2e-05*
2134	3e-13	2e-12	1e-10	8e-10
2135	7e-14	5e-13	3e-11	2e-10
2137	9e-12	6e-11	4e-09	2e-08
2138	9e-11	6e-10	4e-08	3e-07
2139	3e-11	2e-10	1e-08	8e-08
2140	3e-12	2e-11	1e-09	8e-09
2141	7e-12	4e-11	3e-09	2e-08
2143	6e-11	4e-10	3e-08	2e-07
2144	3e-13	2e-12	1e-10	7e-10

* Risks greater than or equal to the 1E-06 (one-in-one-million) level of concern.

Table 5.13 -- Summary of noncarcinogenic hazard indices from occupational exposures to on-site soil gas, 1984 - 1985.¹

LOCATION	OUTDOOR		INDOOR	
	AVG	RME	AVG	RME
1021	2e-07	5e-07	1e-04	2e-04
1023	4e-08	9e-08	2e-05	3e-05
1024	5e-08	1e-07	2e-05	4e-05
1100	4e-07	8e-07	2e-04	3e-04
2122	3e-07	6e-07	1e-04	2e-04
2123	1e-05	3e-05	6e-03	1e-02
2127	4e-09	8e-09	2e-06	3e-06
2128	2e-09	4e-09	9e-07	2e-06
2131	1e-06	2e-06	4e-04	8e-04
2132	1e-03	2e-03	4e-01	8e-01
2133	5e-04	9e-04	2e-01	4e-01
2134	9e-08	2e-07	4e-05	7e-05
2135	1e-08	2e-08	5e-06	9e-06
2137	3e-06	5e-06	1e-03	2e-03
2138	4e-05	8e-05	2e-02	3e-02
2139	1e-05	2e-05	4e-03	93-03
2140	1e-09	2e-09	5e-07	8e-07
2141	3e-07	5e-07	1e-04	2e-04
2143	3e-05	5e-05	1e-02	2e-02
2144	6e-08	1e-07	2e-05	5e-05

1. No hazard index is greater than one (1.0), indicating that no chronic systemic effects are expected due to occupational exposures.

Table 5.14 -- Summary of carcinogenic risk from occupational exposures to on-site soil gas, 1989.

LOCATION	OUTDOOR		INDOOR	
	AVG	RME	AVG	RME
Soil				
18-89-1	4e-12	3e-11	2e-09	1e-08
18-89-2	6e-12	4e-11	2e-09	2e-08
22-89-01	3e-13	2e-12	1e-10	8e-10
22-89-02	2e-12	1e-11	7e-10	5e-09
22-89-03	1e-12	8e-12	5e-10	3e-09
CY-89-02	6e-13	4e-12	2e-10	1e-09
CY-89-05	3e-11	2e-10	1e-08	7e-08
CY-89-06	1e-12	1e-11	6e-10	4e-09
CY-89-07	1e-10	7e-10	5e-08	3e-07
CY-89-08	5e-11	3e-10	2e-08	1e-07
CY-89-09	3e-11	2e-10	1e-08	9e-08
CY-89-10	9e-11	6e-10	4e-08	2e-07
SV89-01A	8e-13	5e-12	3e-10	2e-09
SV89-02	1e-12	6e-12	4e-10	3e-09
SV89-03	3e-12	2e-11	1e-09	8e-09
SV89-04	5e-13	3e-12	2e-12	1e-09
Air				
SV89	2e-07	1e-06*		
18-89	8e-08	5e-07		
89	2e-08	1e-07		
89	8e-08	5e-07		
CY-89	5e-08	3e-07		
CY-89	8e-09	5e-08		

* Risks greater than or equal to 1E-06 (one-in-one-million).

Table 5.15 -- Summary of noncarcinogenic hazard indices from occupational exposures to on-site soil gas, 1989.¹

LOCATION	OUTDOOR		INDOOR	
	AVG	RME	AVG	RME
Soil				
18-89-1	3e-08	6e-08	1e-05	2e-05
18-89-2	1e-08	3e-08	6e-06	1e-05
22-89-01	1e-07	3e-07	6e-05	1e-04
22-89-02	8e-07	2e-06	3e-04	6e-04
22-89-03	4e-07	7e-07	1e-04	3e-04
CY-89-02	1e-07	2e-07	5e-05	1e-04
CY-89-05	2e-07	4e-07	8e-05	2e-04
CY-89-06	3e-08	5e-08	1e-05	2e-05
CY-89-07	1e-05	3e-05	5e-03	1e-02
CY-89-08	1e-06	2e-06	4e-04	8e-04
CY-89-09	3e-06	5e-06	1e-03	2e-03
CY-89-10	1e-05	2e-05	4e-03	9e-03
SV89-01A	2e-07	5e-07	9e-05	2e-04
SV89-02	3e-07	6e-07	1e-04	2e-04
SV89-03	1e-06	2e-06	5e-04	9e-04
SV89-04	2e-07	3e-07	7e-05	1e-04
Air				
SV89	7e-02	1e-01		
18-89	3e-02	6e-02		
22-89	7e-03	1e-02		
22-89	3e-02	7e-02		
CY-89	2e-02	4e-02		
CY-89	2e-03	5e-03		

1. No hazard index is greater than one, indicating that no chronic systemic effects are expected due to occupational exposures.

Table 5.16 – Summary of carcinogenic risk and cancer hazard quotients from occupational exposures to on-site soil gas, 1991.

LOCATION	OUTDOOR				INDOOR			
	Cancer Risk		1,1-DCE CHQ ^a		Cancer Risk		1,1-DCE CHQ ^a	
	AVG	RME	AVG	RME	AVG	RME	AVG	RME
SG-138-01	1e-11	7e-11	3e-08	2e-07	4e-09	3e-08	1e-05	6e-10
SG-138-02	2e-11	1e-10	3e-06	2e-05	8e-09	5e-08	1e-03	7e-08
SG-138-03	7e-12	5e-11	1e-05	8e-05	3e-09	2e-08	5e-03	3e-07
SG-138-04	4e-11	3e-10	9e-06	6e-05	2e-08	1e-07	4e-03	2e-07
SG-138-05	7e-11	5e-10	1e-04	8e-04	3e-08	2e-07	5e-02	3e-06
SG-138-06	2e-12	1e-11	3e-06	2e-05	6e-10	4e-09	1e-03	7e-08
SG-138-07	5e-12	4e-11	6e-08	4e-07	2e-09	1e-08	3e-05	1e-09
SG-138-08	3e-12	2e-11	NR	NR	1e-09	9e-09	NR	NR
SG-138-09	4e-11	3e-10	4e-04	3e-03	2e-08	1e-07	2e-01	9e-06
SG-138-09B	5e-12	3e-11	7e-05	4e-04	2e-09	1e-08	3e-02	1e-06
SG-138-10A	2e-10	1e-09	7e-05	4e-04	8e-08	5e-07	3e-02	1e-06
SG-138-10B	3e-10	2e-09	1e-04	7e-04	1e-07	7e-07	4e-02	2e-06
SG-138-11	1e-11	7e-11	7e-05	4e-04	4e-09	3e-08	3e-02	1e-06
SG-138-12	5e-11	3e-10	2e-05	2e-04	2e-08	1e-07	9e-03	5e-07
SG-138-13	3e-13	2e-12	3e-07	2e-06	1e-10	7e-10	1e-04	7e-09
SG-138-14	8e-13	6e-12	2e-08	1e-07	3e-10	2e-09	9e-06	5e-10
SG-138-15	4e-12	3e-11	2e-05	1e-04	2e-09	1e-08	6e-03	3e-07
SG-138-16	9e-12	6e-11	2e-05	1e-04	4e-09	3e-08	8e-03	5e-07
SG-138-17	6e-12	4e-11	8e-07	5e-06	2e-09	2e-08	3e-04	2e-08
SG-138-18A	1e-12	9e-12	7e-10	4e-09	6e-10	4e-09	3e-07	1e-11
SG-138-18B	2e-12	1e-11	7e-10	4e-09	8e-10	5e-09	3e-07	1e-11
SG-138-19A	4e-13	3e-12	7e-10	4e-09	2e-10	1e-09	3e-07	1e-11
SG-138-01B	4e-13	2e-12	7e-10	4e-09	1e-10	1e-09	3e-07	1e-11
SG-138-20	3e-13	2e-12	7e-10	4e-09	1e-10	8e-10	3e-07	1e-11
SG-138-21	1e-12	7e-12	7e-08	5e-07	4e-10	3e-09	3e-05	2e-09
SG-138-22	8e-12	5e-11	7e-10	4e-09	3e-09	2E-08	3e-07	1e-11
SG-138-23	4e-12	3e-11	7e-10	4e-09	2E-09	1E-08	3e-07	1e-11

a: 1,1-DCE is a Class C carcinogen, a Cancer Hazard Quotient (CHQ) is calculated using a modified RfD approach.

The CHQ is not a probability or risk; it is comparable to the HQ for noncancer systemic effects, if below 1E+00 (1) there is no cause for concern.

xposures to soil gas releases for workers on site do not present health problems. The HI are below 1.0 and also do not indicate a cause for concern (Table 5.17).

Residential, 1992 Data

Excess cancer risk levels, CHQ values and HQ values for indoor and outdoor residential exposures are shown in Tables 5.18 and 5.19. All values are below the level of negligible risk. Carcinogenic risk is well below the one-in-one-million ($1E-06$) level. The CHQ for 1,1-DCE is below 1.0. The HQ values are all well below 1.0 as well. No health problems would be expected from the estimated levels of exposure. Residential populations do not appear to be at risk of negative health effects from exposures to soil gases in the area west of the Motorola 52nd Street facility.

A second sampling was performed at the same sites in July of 1992 (Malcolm Pirnie, 1992). These results are reported in Appendix Table 26. A separate assessment was not performed on the July data as they are in the same range as the March results. The second sampling confirmed the results from the March sampling.

5.3 FUTURE CONDITIONS

It is highly unlikely that the Motorola site will be developed for residential use in the foreseeable future due to zoning and changing land use patterns over the last 20 years (refer to Chapter 3). Residential risks were characterized for on-site soil gas data from 1984 and 1985. Two locations were determined to have elevated residential risk and HI. One was located in the courtyard area of the facility and the other off-site, near the western boundary, on 50th Street. Based on these results it was recommended that residential air sampling be conducted in the future to determine whether the population directly to the west of the facility has significant exposures.

5.4 UNCERTAINTIES IN THE RISK CHARACTERIZATION PROCESS

All risk estimates are based on a number of assumptions regarding contaminant concentrations and fate, exposures, doses, and toxicity information. There is uncertainty associated with the process at all stages. Although point estimates of risk are made, it should be recognized that each one represents a range of possibilities and is really only an indicator. Care is taken at each step to ensure that assumptions and estimates are upper bounds. It is unlikely that true risk is greater than the estimated risks, current and potential, that have been developed in this report.

Table 5.17 -- Summary of noncarcinogenic hazard indices from occupational exposures to on-site soil gas, 1991.¹

LOCATION	OUTDOOR		INDOOR	
	AVG	RME	AVG	RME
SG-138-01	5e-06	9e-06	2e-03	4e-03
SG-138-02	1e-05	2e-05	4e-03	9e-03
SG-138-03	1e-05	2e-05	5e-03	1e-02
SG-138-04	1e-05	3e-05	6e-03	1e-02
SG-138-05	1e-04	2e-04	5e-02	1e-01
SG-138-06	3e-06	6e-06	1e-03	2e-03
SG-138-07	2e-06	4e-06	9e-04	2e-03
SG-138-08	2e-06	3e-06	7e-04	1e-03
SG-138-09	3e-04	7e-04	1e-01	3e-01
SG-138-09B	5e-05	1e-04	2e-02	4e-02
SG-138-10A	5e-05	1e-04	2e-02	4e-02
SG-138-10B	9e-05	2e-04	3e-02	7e-02
SG-138-11	6e-05	1e-04	2e-02	5e-02
SG-138-12	2e-05	5e-05	1e-02	2e-02
SG-138-13	3e-07	6e-07	1e-04	2e-04
SG-138-14	3e-07	5e-07	1e-04	2e-04
SG-138-15	1e-05	2e-05	5e-03	1e-02
SG-138-16	2e-05	4e-05	8e-03	2e-02
SG-138-17	3e-06	6e-06	1e-03	3e-03
SG-138-18A	5e-07	1e-06	2e-04	4e-04
SG-138-18B	7e-07	1e-06	3e-04	5e-04
SG-138-19A	4e-08	9e-08	2e-05	4e-05
SG-138-019B	4e-09	8e-09	2e-06	3e-06
SG-138-20	2e-08	3e-08	7e-06	1e-05
SG-138-21	4e-07	8e-07	2e-04	3e-04
SG-138-22	1e-09	2e-09	4e-07	8e-07
SG-138-23	1e-08	2e-08	4e-06	8e-06

1. No hazard index is greater than one, indicating that no chronic systemic effects are expected due to occupational exposures.

Table 5.18 – Calculation of indoor CDI, risk, cancer hazard quotient (CHQ), and noncancer hazard index (HI), using maximum concentrations detected during the March, 1992 soil gas sampling.

CHEMICAL	INDOOR CONCENTRATION mg/m ³	AVERAGE CDI mg/kg/day	RME CDI mg/kg/day	AVERAGE RISK	RME RISK	AVERAGE CHI	RME CHI	AVERAGE HQ	RME HQ
BENZENE	7.56E-06	1.78E-07	8.9E-07	5.1E-09	2.6E-08				
TOLUENE	1.88E-06	4.41E-08	2.2E-07					7.7E-08	3.9E-07
ETHYLBENZENE	9.58E-07	2.25E-08	1.1E-07					8.0E-08	4.0E-07
XYLENES	2.97E-06	6.96E-08	3.5E-07					8.1E-07	4.0E-06
1,1-DCE	4.22E-04	9.91E-06	5.0E-05			1.1E-01	5.5E-01	1.1E-03	5.5E-03
1,2-t-DCE	6.96E-06	1.63E-07	8.2E-07					8.2E-06	4.1E-05
PCE	9.66E-05	2.27E-06	1.1E-05	3.4E-10	1.7E-09			2.3E-04	1.1E-03
TCE	2.18E-06	5.13E-08	2.6E-07	8.7E-10	4.4E-09			8.6E-06	4.3E-05
F-113	8.93E-04	2.10E-05	1.0E-04					2.7E-06	1.4E-05
TOTALS				6.4E-09	3.2E-08	1.1E-01	5.5E-01	1.3E-03	6.7E-03
EXPOSURE FACTORS	2.35E-02 AVE 1.17E-01 RME								

Inhalation	PCE	TCE	BENZENE	TOLUENE	ETHYL-BENZENE	XYLENES	1,1-DCE	1,2-t-DCE	F-113
WoE	B2	B2	A	D	D	D	C	D	D
Slope Factor (mg/kg/d) ¹	1.5E-04	1.7E-02	2.9E-02	NA	NA	NA	NA	NA	NA
Rfd (mg/kg/d)	1.0E-02	6.0E-03	NA	2.0E-01	NA	NA	9.0E-03	2.0E-02	NA
Rfc (mg/kg/d) ^a	NA	NA	NA	5.7E-01	2.8E-01	9.0E-02	NA	NA	7.7E+00

SF for PCE calculated from risk per concentration unit in air (HEAST, 1991)

Rfd and Rfc values are from IRIS, April 1992 or HEAST, 1991 with the exception of the Rfd for TCE.

This is a provisional oral Rfd issued by the Environmental Criteria and Assessment Office EPA in April 1992.

a. The Rfc values have been converted from concentrations to dosages.

Table 5.19 – Calculation of outdoor CDI, risk, cancer hazard quotient (CHQ), and noncancer hazard index (HI), using maximum concentrations detected during the March, 1992 soil gas sampling.

CHEMICAL	OUTDOOR CONCENTRATION mg/m ³	AVERAGE CDI mg/kg/day	RME CDI mg/kg/day	AVERAGE RISK	RME RISK	AVERAGE CHI	RME CHI	AVERAGE HQ	RME HQ
BENZENE	7.45E-07	2.19E-09	2.9E-08	6.3E-11	8.5E-10				
TOLUENE	1.85E-07	5.44E-10	7.2E-09					9.5E-10	1.3E-08
ETHYLBENZENE	9.43E-08	2.77E-10	3.7E-09					9.9E-10	1.3E-08
XYLENES	2.92E-07	8.58E-10	1.1E-08					1.0E-08	1.3E-07
1,1-DCE	4.16E-05	1.22E-07	1.6E-06			1.4E-03	1.8E-02	1.4E-05	1.8E-04
1,2-t-DCE	6.86E-07	2.01E-09	2.7E-08					1.0E-07	1.3E-06
PCE	9.52E-06	2.79E-08	3.7E-07	4.2E-12	5.5E-11			2.8E-06	3.7E-05
TCE	2.15E-07	6.32E-10	8.4E-09	1.1E-11	1.4E-10			1.1E-07	1.4E-06
F-113	8.80E-05	2.58E-07	3.4E-06					3.4E-08	4.5E-07
TOTALS				7.8E-11	1.0E-09	1.4E-03	1.8E-02	1.7E-05	2.2E-04
EXPOSURE FACTORS	2.94E-03 AVE 3.91E-02 RME								
Inhalation	PCE	TCE	BENZENE	TOLUENE	ETHYL- BENZENE	XYLENES	1,1-DCE	1,2-t-DCE	F-113
WoE	B2	B2	A	D	D	D	C	D	D
Slope Factor (1/mg/kg/d)	1.5E-04	1.7E-02	2.9E-02	NA	NA	NA	NA	NA	NA
RfD (mg/kg/d)	1.0E-02	6.0E-03	NA	2.0E-01	NA	NA	9.0E-03	2.0E-02	NA
RfC (mg/kg/d) ^a	NA	NA	NA	5.7E-01	2.8E-01	9.0E-02	NA	NA	7.7E+00

SF for PCE calculated from risk per concentration unit in air (HEAST, 1991)

RfD and RfC values are from IRIS, April 1992 or HEAST, 1991 with the exception of the RfD for TCE.

This is a provisional oral RfD issued by the Environmental Criteria and Assessment Office EPA in April 1992.

a. The RfC values have been converted from concentrations to dosages.

No public production wells have been influenced by the contaminant plume. There are no public production wells in the area likely to be affected by the groundwater plume. Although there is no statutory restraint on the drilling of private wells in the area, it is unlikely that new private wells will be drilled in the area due to the availability of relatively inexpensive publicly supplied water. It is doubtful that the potential risks associated with domestic groundwater use will ever be realized.

5.4.1 Data Uncertainties

The selection of chemicals of potential concern (COPC) is not an exact science. Decisions must be made to include or exclude compounds based on detected concentrations and frequencies. The ADHS has taken the most conservative approach to develop the list of COPC. Chemicals were included in the groundwater assessment of a given well although never detected at that location, based on its detection in the study area. For example, vinyl chloride, a class A carcinogen, was detected in three alluvial and three bedrock wells and has been included in the assessment for each well. Vinyl chloride is the main contributor to potential ELCR at wells in which it has never been detected, but in which it had high SQLs; however, it would not be prudent to exclude it from the assessment. Some inorganics were included in the list of COPC, although there are indications they may be present at background levels.

Mean concentrations were used in estimation of the average potential ELCR and the 95% UCL was used for RME calculations. This was done to better characterize the true range of probable potential risk. Assumptions used in the calculations also reflect this range from estimated mean values to upper bound estimates. True risk may be much less than calculated risk. This is done purposefully to be protective of public health.

The soil gas exposure route is an current pathway. For the soil gas assessment, all VOCs detected were used, in order to produce the most conservative estimates of risk. Conservative assumptions were used for modeling emissions from the soil to the air in an attempt to present a true upper bound estimate of risk from soil gas.

5.4.2 The Toxicity Assessment

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.

Slope factors, RfDs, and RfCs are not available for some chemicals. Arsenic, a class A carcinogen, has no approved slope factor. The slope factors for TCE and PCE have been withdrawn pending review and previously published values have been used. The RfD used for TCE was developed especially for this risk assessment by the USEPA Office of Environmental Criteria and Assessment.

5.4.3 Data Presentation

The potential risk estimates for residential ground water use was presented graphically in contour and three-dimensional representations. This was accomplished using Surfer (Golden Software, 1991), a graphics package designed to present geographic and environmental data. The program takes irregularly spaced data and converts it to a regularly spaced form that can be used to create surface representations. An inverse distance method was used for calculation of the plotted data. This method was chosen because it best retains the influence of each individual sampling point. Other methods that could have been chosen, such as Kriging, produce a smoother surface using statistical techniques. The integrity of the sampling points is not maintained. The method chosen, better represents the discrete sampling points, while still interpolating between sampling locations and extrapolating beyond the sampling area to produce an estimate of the potential risk distribution over the entire study area. The results are presented in Figures 5.1 through 5.9.

5.5 SUMMARY

At present, the contamination associated with the unpermitted and uncontrolled releases of chemicals to the groundwater at the Motorola Inc. 52nd Street facility Superfund site does not appear to expose the surrounding population to high levels of excess lifetime cancer risk (ELCR). The ELCR associated with the groundwater contamination is *potential*. There are two known exposure points, private well 4626G and SRP well 18E-5N. Current data does not indicate that either is presently a source of high risk. Both should be carefully monitored in the future as the situation could change due to movement of the contaminant plume.

The assessment of *current* ELCR associated with soil gas do not indicate high levels of risk. All indoor and outdoor residential risks were below the negligible risk level of one-in-one-million (1E-06) using both 1985 and 1992 data. ELCR due to occupational exposures to soil gas at the facility were above the 1-in-one-million level, but not high for an occupational setting. The estimates presented do not

represent total occupational risk which also includes exposures to compounds used in the manufacturing process. This report does not address total occupational risk.

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