HUMAN HEALTH RISK ASSESSMENT

for
Future Residential Tract 7003
621 Daggett Ave.
Union City, CA

Prepared for :
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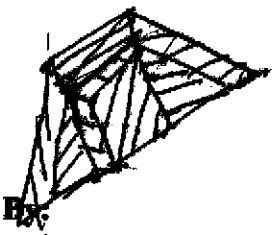

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September 14, 1998

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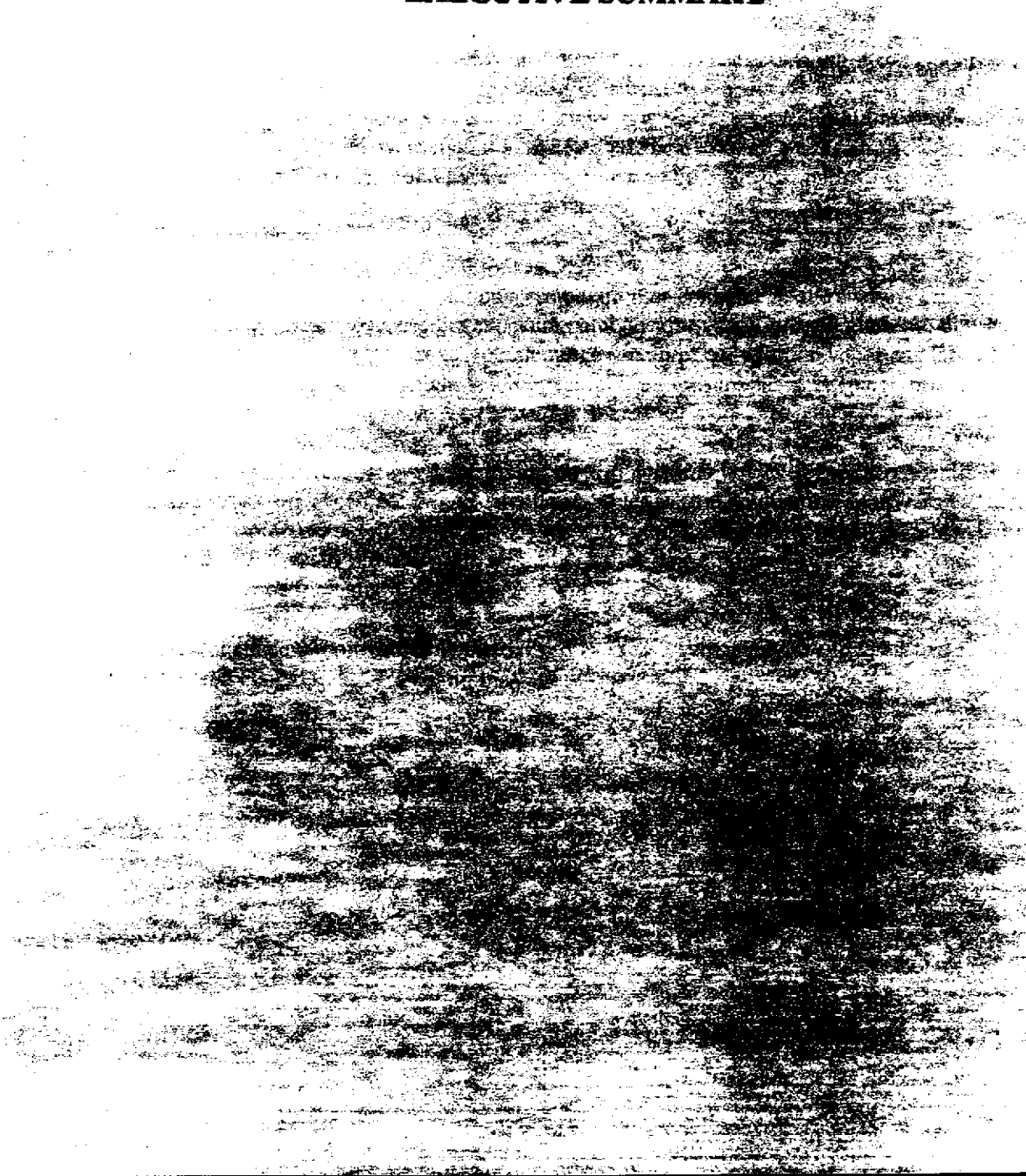
LIST OF ABBREVIATIONS

ADD	Average Daily Dose
ARARs	Applicable or Relevant and Appropriate Requirements
ASTM	American Society for Testing and Materials
bgs	below ground surface
BMD	Benchmark Dose
BTEX	Benzene, Toluene, Ethylbenzene and Xylenes
BW	body weight
Cal/EPA	California Environmental Protection Agency
COPC	Chemicals of Potential Concern
CPT	Cone Penetrometer Test
CSF	Cancer Slope Factor
CSM	Conceptual Site Model
DAF	Dermal Absorption Factor
DCA	Dichloroethane
DCE	Dichloroethene
DNAPL	Dense Non-Aqueous Phase Liquid
EBMUD	East Bay Municipal Utility District
ED	Exposure Duration
EF	Exposure Frequency
EPC	Exposure Point Concentration
ER	Emission Rate
F	Fahrenheit
foc	fraction of organic carbon
GC/MS	Gas chromatography/ Mass spectrometry
HEAST	Health Effects Assessment Summary Table
HHRA	Human Health Risk Assessment
HI	Hazard Index
HQ	Hazard Quotient
IR	intake rate
IRIS	Integrated Risk Information System
LADD	Lifetime Average Daily Dose
LMM	Linearized Multistage Model
LOAEL	Lowest Observed Adverse Effect Level
mg/kg	milligrams per kilogram
mg/kg-day	milligrams per kilogram per day
Mg/L	mg per liter
mg/m3	milligrams per cubic meter
MLE	Maximum Likelihood Estimate
msl	mean sea level
NAS	National Academy of Sciences
ND	non detect
NOAEL	No-Observed Adverse Effect Level
OSHA	United States Occupational Safety and Health Administration
PC	permeability constant
PCA	Tetrachloroethane

PCE	Tetrachloroethylene or Perchloroethylene
PEF	Pathway Exposure Factor
PEL	Permissible Exposure Limit
ppb	part per billion
ppb/v	parts per billion per volume
PRGs	Preliminary Remediation Goals
PVC	Polyvinylchloride
RCRA	Resource Conservation and Recovery Act.
RfC	Reference concentration
RfD	Reference Dose
RME	Reasonable Maximum Exposure
RWQCB	Regional Water Quality Control Board
SI	Supplemental Investigation
SQL	Sample Quantitation Limit
SVOC	Semi volatile Organic Compounds
TCE	Trichloroethylene
TDS	Total Dissolved Solids
TLV	Threshold Limit Value
TOC	Total Organic Carbon
TPH	Total Petroleum Hydrocarbon
UCL	Upper Confidence Limit
USEPA	United States Environmental Protection Agency
USGS	United states Geological Survey
VF	Volatilization factor
VOCs	Volatile Organic Compounds
µg/kg	micrograms per kilogram
µg/L	micrograms per liter

EXECUTIVE SUMMARY



EXECUTIVE SUMMARY

EXECUTIVE SUMMARY

This baseline human health risk assessment (HHRA) was prepared as a Supplemental Report (SR) to evaluate potential human health risks associated with the chlorinated solvents in the groundwater at 621 Daggett Avenue, Union City, California. Specifically, the overall objectives of this HHRA are to: (1) Provide an analysis of the actual and potential baseline risks, assuming no remedial action or institutional controls under both current and future land uses. (2) determine the need for remediation, (3) provide justification for the no-action alternative (if there are no significant risks) or for performing the remedial action (if remedial action is deemed necessary) and (4) determine which media need to be remediated.

Ecological impacts are expected to be negligible at this site given the fact that future land use involves clearing the land and the construction of residential housing.

ES 1 HUMAN HEALTH RISK ASSESSMENT

This HHRA was conducted in accordance with standard and customary practice as specified by the California Environmental Protection Agency (Cal/EPA) and U.S. Environmental Protection Agency (USEPA) guidelines for the performance of risk assessments (Cal/EPA, 1992 and 1994a; USEPA, 1989a).

ES 1.1 Identification of chemicals of Potential Concern

Although actual environmental sampling activities have not been conducted on the subject site, data are available from investigation of the neighboring site the McKesson Chemical Corp. property. This investigation indicates chlorinated hydrocarbon contamination of the groundwater, which is moving under the subject site. In effect, it is assumed that the groundwater under the 621 Daggett Ave. site is also contaminated with chlorinated hydrocarbons.

Results of past investigations indicate that volatile organic compounds (VOCs) in the form of chlorinated solvents, are found in the groundwater beneath the site. According to the May 1998 groundwater monitoring results from the McKesson property, the following chemicals (Table ES-1) have been identified:

Table ES-1. Identified Chemical of Potential Concern (COPCs) and their maximum reported concentrations in samples drawn from 10 monitoring wells surrounding the Daggett Avenue property.

Chemical of Concern	Maximum Concentration
1,1 Dichloroethylene (1,1-DCE)	210 µg/l
1,1,1 Trichloroethane (TCA)	150 µg/l
Trichloroethylene (TCE)	720 µg/l
Perchloroethylene (Perc)	1200 µg/l

ES 1.2 Toxicity Assessment

Oral cancer slope factors (CSFs), inhalation unit risk factors, oral reference doses (RfDs) and inhalation reference concentrations (RfCs) that have been developed by Cal/EPA (Cal/EPA, 1994c) and USEPA (USEPA, 1998) were used as indices of toxicity for the COPCs identified in this HHRA.

When available, Cal/EPA CSFs were selected for use. When Cal/EPA values were not available, USEPA values were used. Thus, the following hierarchy of available sources was used to select CSFs for each of the COPCs: California Cancer Potency Factors, USEPA's Integrated Risk Information System (IRIS)(USEPA, 1998) and USEPA's Health Effects Assessment Summary Tables (HEAST)(USEPA, 1997a). At the present time, CAL/EPA and USEPA have developed CSFs only for oral and inhalation routes of exposure. In the absence of toxicity values specific to the dermal route, oral factors adjusted by means of a dermal absorption factor, can be used to derive dermal toxicity values. However, dermal exposures were considered to be insignificant and not evaluated in this HHRA.

ES 1.3 Exposure Assessment

A conceptual site model (CSM) describing present site conditions and all complete/potentially complete exposure pathways was developed for this HHRA. Most of the area surrounding the Daggett Ave. property is destined for residential use. Single family residences are expected. The future or the property directly to the west, the McKesson property which is a Superfund site, is unknown.

Exposures to VOCs emissions from underground sources could occur during construction of housing foundations and building of the homes. Once completed, exposures to VOCs could occur via outside air emissions. Extraneous indoor air emissions could occur through cracks and faults in cement foundations. However, this is unlikely since the houses will be built on solid slab foundations poured over a visqueen barrier.

Although the houses will be supplied with municipal water so exposure through water use is an incomplete pathway, exposures via the drinking water were evaluated nevertheless.

Based on the physical setting of the site and anticipated land use, the three potential receptors addressed in this HHRA include construction workers; specifically infrastructure/utility workers (storm drain construction or utility), and future residents (adults and children).

The potential carcinogenic risks and non-carcinogenic health effects were assessed for each of the following complete or potentially complete exposure pathways.

- Inhalation of VOCs in shallow groundwater from excavation activities by construction/utility workers and on-site residents (tree planting).
- Inhalation of VOCs emanating from subsurface soils and shallow groundwater into indoor air by future on-site residents.
- Exposures attributed to domestic use of the groundwater by the future residents and their children.

To quantify human health risks for the exposure pathways identified in the CSM, the central tendency exposure (***CTE***) and reasonable maximum exposure (***RME***) estimates for each receptor were assumed. ***RME*** is defined as the maximum exposure that is reasonably expected to occur at the site (USEPA, 1989a). The ***RME*** for a given pathway was derived by combining the 95 percentile upper confidence limit (UCL) of the mean or the maximum detected concentration for each constituent with upper bound values describing the intake rate and extent, frequency and duration of exposure (USEPS, 1989a). The ***CTE*** scenarios were calculated by combining the average exposure point concentrations (EPCs) and exposure parameters.

EPCs for each COPC measured from shallow groundwater were estimated and modeled from the available well monitoring data. VOC concentrations in indoor air of future structures were estimated using models presented in the American Society for Testing and Materials (ASTM) *Standard Risk-Based Corrective Action Guidelines* (ASTM E1739;95). These specific models estimate volatilization from either soil, soil vapor, or shallow groundwater, upward transport of soil gas through soils, the foundation and floor of the building (primarily cracks and seams).

ES 1.4 Risk Characterization

Risk characterization is defined as the description of the nature and magnitude of actual and potential human health risks, including their inherent uncertainty. USEPA has determined that sites posing a cumulative lifetime cancer risk above 1×10^{-4} may pose an unacceptable risk and may require remedial action (USEPA, 1991c). Under most situations, carcinogenic risks in the range of 1×10^{-4} to 1×10^{-6} may or may not require remediation, depending on site-specific conditions. Non-carcinogenic hazard indices (***HIs***) of one or less are considered to be acceptable depending again upon site specific conditions. Risks above this range are generally considered unacceptable in which case remediation may be required.

Both ***CTE*** and ***RME*** estimates of chemical specific carcinogenic risks and non-carcinogenic ***HIs*** were estimated in this HHRA. According to USAEPA (1989a), multiple pathway ***RME*** risks could be best presented by a combination of single-pathway ***RME*** and other ***CTE*** risks, depending upon the site specific conditions. Thus, the ***HIs*** were estimated for the cumulative total for all compounds identified as COPCs.

With the anticipated residential development, the future inhabitants of these homes are unlikely to experience adverse carcinogenic health effects as a result of exposure to the chlorinated hydrocarbons found in the groundwater underlying the site. Under both ***CTE*** and ***RME*** scenarios, the carcinogenic cumulative risks for future residents due to vapor intrusion are 5.66×10^{-6} and 1.17×10^{-5} respectively. The majority of the cumulative carcinogenic risk is attributable to the inhalation of 1,1-dichloroethene.

The ***HIs*** attributed to vapor intrusions for future residential children is also negligible at the ***CTE***, but slightly above 1.0 at the ***RME***. The use of the groundwater for domestic purposes introduces a variable that has a dramatic effect and raises the ***HI*** for future residential children considerably. Hence, the ingestion of groundwater overwhelms any effect attributable to the vapor intrusion so that the cumulative non-carcinogenic ***CTE*** and ***RME HIs*** for future residential children are 3.86 and 13.1 respectively.

The ***HIs*** that are attributable to the ingestion of groundwater are likely to be overestimates due to the conservative nature of the assumptions employed in the quantitative estimates and the

overestimation may amount to several orders of magnitude. Nevertheless, this risk assessment indicates that, under current conditions, the use of the groundwater for domestic purposes should be avoided.

The non-carcinogenic ***HIs*** to site development workers is negligible since the values are in the 10^{-4} range which is considerably below the value of 1.0.

Future construction/utility workers were assumed to be exposed to constituents via direct contact pathways and inhalation of VOCs released during excavation activities. Workers involved in redevelopment activities are unlikely to experience adverse carcinogenic and non-carcinogenic health effects as a result of exposure to the constituents in subsurface soils and the shallow groundwater since the estimated carcinogenic risks for this receptor are less than 3.19 x 10-9 for the ***CTE*** scenario and 4.42 x 10-9 for the ***RME*** scenario. Similarly, the non-carcinogenic ***HIs*** are less than one for both exposure scenarios which indicates a negligible likelihood for any health effects.

Summaries of the ***CTE*** and ***RME*** carcinogenic risks and non carcinogenic ***HIs*** for each of the receptors under the hypothetical scenarios are presented in Table ESE-2. The cumulative carcinogenic risks and ***HIs*** are also presented in this table.

Table ES-2. Summary of Cancer Risk and Hazard Indices at 621 Daggett Ave.

Land Use Scenario	Receptor	Total Excess Cancer Risk		Total Non-Cancer Hazard Index	
		CTE	RME	CTE	RME
Residential	adult*	5.56E-6	1.17E-5	---(1)	---(1)
Residential	child*	---(2)	---(2)	1.79E-1	4.59E-1
Site Redevelopment	worker	3.15E-9	4.42E-9	1.65E-4	2.31E-4

* vapor intrusion only

(1) - children are more sensitive due to lower body weights.

(2) - the carcinogenic risk to children isexpressed during adult years

ES 1.5 Uncertainty.

To be health protective, the types of summations used in this HHRA were generally conservative. To the extent possible, site specific factors were incorporated in the HHRA. However, even the most site specific risk assessment is still subject to uncertainty. The conservative assumptions employed in the HHRA were biased in such a manner to cause risks to be overestimated rather than underestimated. Consequently, it is important that the magnitude of the uncertainties and biases be considered when interpreting the health risk results.

A principal source of uncertainty and potential bias associated with this HHRA are the models and assumptions employed to predict indoor air concentrations. The models employed conservatively assume that future buildings or residences will be constructed directly over the highest VOC locations and an infinite source of mass will be sustained over the exposure period duration. The models also assume that 100 percent of the constituents are migrating to the surface without any lateral migration, which would tend to reduce the estimated surface flux. In addition, the models assume that subsurface conditions beneath the site are uniform across the

entire site. It has been well documented that the potential migration of soil vapor into indoor air is highly variable and depends upon a number of conservative assumptions related to building air exchange rates and the area of the infiltration.

Another principal source of uncertainty is the treatment of 1,1-DCE as a carcinogen. This chemical is classified by USEPA (IRIS, 1998) as a Group C of possible human carcinogen, but Cal/EPA does not classify it as a carcinogen. In this HHRA, 1,1-DCE has been evaluated quantitatively as though it were carcinogenic. Thus, it is highly likely that the carcinogenic risks are greatly overestimated for this compound. In addition, since the largest portion of the total cancer risks are due to 1,1-DCE, it is also likely that total carcinogenic risk is also greatly overestimated.

In evaluating the pathways associated with shallow groundwater under future site conditions, the concentrations present in shallow groundwater were used to represent EPCs throughout the duration of exposure. No change in concentrations as a result of natural dilution/attenuation processes were considered. Consequently, the estimated carcinogenic risks and non-carcinogenic health effects from inhalation of VOCs in indoor air may be overestimated.

ES 1.6 Conclusions

Under the anticipated land use conditions, remediation of constituents the shallow groundwater is not necessary. This is because the exposure pathways for vapor intrusion into the future homes is estimated to fall into an acceptable range. In addition, addition vapor barriers will be installed during construction which would interrupt the exposure pathway (incomplete) and mitigate the exposure to intrusive VOCs even further.

Estimations of risks associated with drinking the groundwater however, produce quantitative numbers that are not acceptable. The calculated values for the non-carcinogenic ***HIs*** produce numbers that are in excess of "1" which suggests that non-carcinogenic health effects may be anticipated. As a result, the use of the groundwater for domestic purposes should be avoided under the current anticipated land uses.

The carcinogenic and non-carcinogenic risks to workers developing the site fall into a range that is considered insignificant and negligible.

SECTION 1.0

INTRODUCTION

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1.0 INTRODUCTION

This baseline Human Health Risk Assessment (HHRA) has been prepared by EnTox & Associates (EnTox) for Citation Homes in Santa Clara, California. This HHRA is intended to evaluate the potential human health associated with the presence of chlorinated hydrocarbon solvents in the groundwater beneath the site located at 621 Daggett Avenue in Union City, California. The site is destined for development into residential properties.

1.1 Purpose of the Risk Assessment

Risk assessment is generally defined as the scientific evaluation of human and environmental health impacts associated with exposure to a particular substance or mixture of substances. The purpose of this HHRA is to provide a health protective quantitative analysis of the potential adverse effects associated with human exposures to site-related constituents in the groundwater as a result of past chlorinated solvents releases from adjacent properties. The intent is to provide all interested parties with an understanding of the nature of the chemical releases , the pathways of human exposure and the degree to which such releases may pose a potential for adverse health effects.

Site specific objectives for this HHRA are to:

- Provide an analysis of the actual and potential baseline risks to the underground chemicals assuming no remedial action or institutional control is implemented under current or future land use conditions.
- Determine the need for remediation
- Provide justification for the no-action alternative (if there is no significant risk).
- Provide justification for performing a remedial action (if deemed necessary).
- Determine the media to remediate, if remediation is necessary.

Evaluation of exposure to ecological receptors will not be addressed in this risk assessment since the area is intended for development into residential use.

1.2 Risk Assessment Approach

This HHRA is consistent with the risk assessment paradigm originally proposed by the National Academy of Sciences (NAS, 1983) and follow standard and customary practice as specified in the California Environmental Protection Agency (Cal/EPA) and U. S. environmental protection Agency (USEPA) guidelines for the performance of risk assessments (Cal/EPA, 1994a;1994b; USEPA, 1989a). To the extent possible, recent improvements and refinements in the practice of risk assessment have also been incorporated into this HHRA.

1.3 Organization of the Report

This HHRA report is organized into subsections as follows:

- **Section 1.0** introduces the objectives of the HHRA.

- **Section 2.0** presents the process for identifying the chemical of potential concern (COPCs that are carried through the HHRA, including a discussion of facility history, results of previous environmental investigations and nature and extent of contamination.

- **Section 3.0** summarizes the toxicity information for the COPCs considered in this RA.

- **Section 4.0** discusses the exposure assessment, which includes the physical setting of the site, potential receptor, exposure pathways, exposure point concentrations and human intake doses.

- **Section 5.0** present the results of the HHRA in which the attendant health risks associated with the site related exposures are evaluated and described.

- **Section 6.0** discusses the uncertainties involved in estimating risks at this site, which should be considered when interpreting the potential risk estimates.

- **Section 7.0** provides a summary of the overall conclusion of the HHRA.

- **Section 8.0** provides the references use in the preparation of this HHRA.

SECTION 2.0

IDENTIFICATION OF THE CHEMICALS OF POTENTIAL CONCERN

2.0 IDENTIFICATION OF THE CHEMICALS OF POTENTIAL CONCERN

This section introduces a summary of site history and geology, previous investigations and nature and extent of contamination; presents the analytical data detected in various environmental media and discusses the selection of the COPCs that were evaluated in the HHRA. COPCs are generally defined as those chemicals that are attributed to site-related activities and are most likely to be of concern to human health or the environment.

2.1 History and Summary of the Site

Prior to 1960, the site was used only for agricultural purposes and remained undeveloped through 1977. In 1960, Higgens Lumber constructed their facilities across the street. In aerial photographs taken in 1977, four above ground storage tanks can be seen along the northeast border of the site on the adjoining property. In 1985 a large building was constructed on the site that was occupied by City Freight Lines. In 1990, Clipper Express took over the buildings and the site. Clipper Express was a freight forwarding business and did not own maintain, service or fuel trucks on the property. However, in December of 1994, a fuel truck ruptured its tank and spilled approximately 100 gallons of diesel fuel. This spill was reported to be contained and contaminated soils removed and disposed. (Terrasearch, 1998). Additionally, Higgens Lumber (across the street) had two underground diesel fuel tanks removed at some point in time but details of any remedial action associated with these USTs are not available. The product lines for these USTs still exist and lead from the former location of the USTs to the kiln. Other than these two diesel reports, no other significant chemical mishaps appear to have occurred on the subject property (Levine Fricke, 1995).

Several hazardous waste generators (HSGs) are located within a tenth of a mile of the subject property and are described below.

1. McKesson Chemical Co. located at 33950 7th Street is a large RCRA hazardous waste generator and up-gradient from the subject property. Two UST have apparently contaminated the groundwater. A remedial action of pumping and treating groundwater is currently ongoing, regulated by the California Regional Water Quality Control Board, San Francisco Bay Region (RWQCB) (Terrasearch, 1998).

2. Oxford Tire Recycling of Northern California located at 33950 7th Street is 0.02-mile west and cross gradient from the site. Although the operation is now closed, the site has been classified as a solid waste landfill (Terrasearch, 1998).

3. Magnaflux Surface Conditioners, Inc. (MSCI) located at 301 Daggett Ave. is approximately 300 ft directly east and cross gradient from the site. Although this operation is no longer there, this site has been designated as a large RCRA waste generator. Details regarding the wastes generated at this site are scanty but some of the files from MSCI indicate the storage of spent halogenated solvents including 1,1,1 TCA. A 1985 investigation detected TCE in surface soils on-site (Levine Fricke, 1995).

This site history suggests that the McKesson Chemical company and the Magnaflux Surface Conditioners, Inc. are the primary contributors to the groundwater contamination under the parcel at 621 Daggett Ave.

2.2 Nature and Extent of Contamination

Since January 1986, the McKesson property has been under an investigation and remedial work order to mitigate contaminated groundwater. These activities have been governed by Waste Discharge Requirements Order 86-3 and its successor, Site Cleanup Requirements Order 88-104, issued by the California Regional Water Quality Control board, San Francisco Bay Region (RWQCB).

2.2.1 Occurrence and Distribution of Chemicals in Shallow Groundwater

The current groundwater extraction system for the shallow aquifer consists of 21 shallow extraction wells. The system began operation in November of 1991. According to monitoring results, the direction of groundwater flow in the shallow aquifer is from the McKesson property toward the south and under the 621 Daggett Ave. property with a hydraulic gradient of approximately 0.004 foot per foot (ft/ft).

Table 2-1: Concentrations of chemicals (µg/L) detected in groundwater near 621 Daggett Avenue during 1998.

not where has been conducted on the referred property

Date Of Sampling	Well #	Depth to Groundwater (ft bgs)	1,1-DCE	TCA's	TCE	PCE
Feb 98	19	37.5	40	15	110	25
May 98	1	32.3	26	15	51	160
	3	33.1	190	150	130	540
	5	31.2	50	21	36	80
	8	31.5	75	28	100	220
	9	31.7	53	32	720	440
	10	35.4	20	13	190	47
	11	30.0	31	18	54	130
	12	31.8	210	90	250	1,200
	17	32.8	72	30	350	320
	19	33.1	43	17	150	25
Maximum		37.5	210	150	720	1,200
Minimum		30.0	20	13	36	25
Mean		32.8	74	37	199	338
95% UCL on Mean			104	53	277	408

Analytical data from the ten wells closest to the Daggett Avenue property were selected to represent possible exposures. The number of the selected wells, depth to groundwater and detected chemical concentrations are shown in Table 2-1. Four key organic chemical compounds were identified in sampling these wells in February and May 1998. These were 1,1 Dichloroethylene (1,1 DCE), 1,1,1-

trichloroethane (1,1,1-TCA), trichloroethylene (TCE) and Tetrachloroethylene, also known as perchloroethylene (PCE). The chemicals and their maximum concentrations detected in May of 1998 are also presented in Table 2.1.

2.2.2 Occurrence and Distribution of Chemicals in the Intermediate Groundwater Aquifer.

Ground water is being extracted and treated on the McKesson Chemical Co. property by means of two intermediate aquifer zone extraction wells. Monitoring results indicate that the same chemicals contaminate the shallow and intermediate groundwater zones.

2.2.3 Occurrence and Distribution of Chemicals in the Deep Groundwater Aquifer

The deep aquifer essentially had no detectable levels of halogenated hydrocarbons in the May 1998 report. The overall trend in monitoring results from February 1998 to May 1998 indicate a decrease in the detectable levels of halogenated solvent concentrations for the primary chemicals of concern. This could be due to recharge of groundwater from the surface following higher-than-average rainfall during the El Niño event of 1997-1998. Approximately 75% of the groundwater being extracted and treated is derived from the shallow aquifer. The remainder of the groundwater being treated is derived from the intermediate aquifer.

2.2.4 Summary

Analytical data form past and current investigations of neighboring properties have identified four key chlorinated solvents in the groundwater under the site located at 621 Daggett Avenue in Union City, California: 1,1 Dichloroethylene, (1,1 DCE), 1,1,1-Trichloroethane (TCA), Trichloroethylene (TCE) and Perchloroethylene (PCE). Data collected from monitoring wells indicate that the maximum concentration observed during 1998 for any of these chemicals was PCE at 1200 µ/l. The highest concentration of any contaminant occurred under the southeastern sector of the property (Property Lot 17).

2.3 Selection of Chemicals of Potential Concern

Although a number of constituents have been detected in the groundwater at the site, the USEPA Risk Assessment Guidance (USEPA, 1989a) presents a methodology for identifying which detected constituents should be included in a quantitative risk assessment as Chemicals of Potential Concern (COPCs). These COPCs are defined as chemicals potentially related to the Site and whose data are of sufficient quality for use in a quantitative risk assessment. It is important to recognize that the selection of a chemical as a COPC does not necessarily indicate that it poses a significant health risk. The guidance generally state that he list of COPCs should include all constituents that were:

- Positively detected in at least one sample,
- Detected above ambient levels,
- Tentatively identified but may be associated with the Site based on historical information.
- Transformation products of detected constituents.

However, the primary driving force in selecting the COPC for this risk assessment was the availability of suitable data. Because this site has not been investigated directly, and contamination has been demonstrated for neighboring sites, the data available in public documents were used as the only reasonable sources. As a result, the quarterly monitoring reports from the McKesson Chemical Company provided the data for this risk assessment and dictated the selection of the COPCs.

SECTION 3.0

TOXICITY ASSESSMENT

3.0 TOXICITY ASSESSMENT

Toxicity assessment is the process of using existing toxicity information from human or animal studies to identify potential health risks at various dose levels in exposed populations (USEPA, 1989a). To estimate these potential health risks, the relationship between exposure to a chemical (in terms of intake dose to individuals) and an adverse effect (in terms of bodily response to a specific intake dose level) must be quantified. Without the dose-response relationship embodied in toxicity criteria, risk-based decision making for human health protection purposes could not be achieved.

Toxicity assessments for contaminants found at hazardous waste sites generally consist of two steps; hazard identification and dose response assessment (USEPA, 1989a). Hazard identification is a qualitative process of determining whether exposure to a chemical agent can cause adverse health effects, especially in humans. The dose-response assessment step of the HHRA involves characterizing the relationship between administered dose and/or absorbed dose of a chemical agent and the magnitude or likelihood of the adverse health effects (USEPA, 1989a).

For chemicals that are known or suspected to cause cancer the dose-response assessment process defines the relationship between the dose of the risk agent and the probability of induction of carcinogenic effects in humans or animal species of interest. For systemic toxicants, or chemicals that give rise to toxic endpoints other than cancer and gene mutations (also called non-carcinogens), the dose-response assessment determines a threshold value below which the adverse non-carcinogenic effects are not expected in the general population, including sensitive subgroups. This HHRA will make use of toxicity assessments already performed by either Cal/EPA or USEPA.

The remainder of this section is divided into four subsections. Sections 3.1 and 3.2 provide a discussion to the carcinogenic and non-carcinogenic dose-response assessment methodologies, respectively. Section 3.3 discusses procedures for COPCs that lack toxicity criteria. Section 3.4 presents the toxicity summaries for the risk-driving COPCs.

3.1 Carcinogenic Dose-Response Assessment Methodology

3.1.1 Qualitative Assessment

Assessment of potential carcinogenicity begins with an assignment of a weight-of-evidence classification for the likelihood that a chemical agent causes cancer in humans. USEPA classifies chemicals with respect to carcinogenicity as Group A, B1, B2, C, D or E. These classifications are defined as follows:

- **Group A** includes known human carcinogens. These are agents for which sufficient evidence exists to support a causal association between exposure to the agents in humans and cancer.

- **Group B1** includes probable human carcinogens. These are agents for which evidence for carcinogenicity in humans is limited but evidence in animals is sufficient.

- **Group B2** includes probable human carcinogens also. These are agents for which evidence of carcinogenicity in animals is sufficient but evidence of carcinogenicity in humans is either inadequate or absent.

- **Group C** includes possible human carcinogens. These are agents for which there is a limited evidence of carcinogenicity in animals and the evidence in humans is inadequate or absent.

- **Group D** chemicals are not classifiable as to human carcinogenicity. For the most part, these are agents for which there is little or no data available.

- **Group E** includes chemicals that are not carcinogens. These are agents for whom human or animal studies exist to suggest no causal link between exposure and increased cancer incidence.

Cal/EPA maintains lists of chemicals known to the State of California to cause cancer. These include chemicals classified by USEPA in Groups A, B1 and B2. In addition, Cal/EPA includes chemical carcinogens identified by the International Agency for Research against Cancer (IARC) in their Groups 1, 2A, 2B, which are defined similarly to USEPA Groups A, B1 and B2, Cal./EPA evaluates Group C carcinogens (or IARC Group 3) on a case-by case-basis, finding that some merit classification as carcinogens while others do not.

Cal/EPA and USEPA classify the four COPCs identified to this risk assessment as follows:

Table 3-1; Carcinogenic Classifications of Chemicals of Potential Concern in groundwater at the Daggett Ave. site.

Chemical	USEPA Classification	Cal/EPA Classification
1,1 Dichloroethylene	Group C	Not listed
Perchloroethylene*	Group B2*	Carcinogenic
Trichloroethane	Group D	Not listed
Trichloroethylene*	Group B2/C*	Carcinogenic

* These classifications were published and withdrawn by USEPA.

3.1.2 Quantitative Assessment

Potential carcinogenic effects resulting from human exposure to chemicals are estimated quantitatively using cancer slope factors (CSFs) expressed in unit risk per milligrams of chemical intake per kilogram body weight per day (mg/kg-day-1). USEPA and Cal/EPA derive CSFs from chronic animal bioassays, human epidemiological studies or both. Animal bioassays are usually conducted at dose levels that are much higher than those likely to be the result of human exposure to environmental media. Such high levels were used in order to detect possible adverse effects in the relatively small animal test populations. Because humans are generally exposed to lower doses in the environment, the high dose date from animals studies are extrapolated to the low-dose region using mathematical models. Both EPA and Cal/EPA employ the linearized multi-stage model (LMM) to estimate the upper 95% upper confidence limit (UCL) on the slope of a straight line that describes the increasing cancer risk with increasing dose. This straight line descends through the origin of the graph because all doses of carcinogens are conservatively assumed to have at least some risk of ability to induce cancer. The 95% UCL on the slope of the dose-response curve is subjected to various adjustments including application of an inter-species scaling factor, to derive a CSF for humans. The (5% UCL slope of the dose-response curve is subjected to various adjustments and an inter-species scaling factor is usually applied to derived a CSF for humans. Dose-response data derived from human epidemiological studies are fitted to dose-response functions on a case-by-case basis.

In both types of analysis, i.e. either animals or epidemiological data, health protective assumptions are applied when data are absent. The models employed are believed to provide rough estimates of the upper limits on potential carcinogenic potency. The actual risks associated with exposure to a potential carcinogen are not likely to exceed the estimated risks and may be much lower or even zero (USEPA, 1989a).

Available CSFs are listed in Table 3-2 for oral and inhalation routes of exposure for the four halogenated solvents identified in the groundwater at the Daggett Ave. site. When available, Cal/EPA CSFs (Cal/EPA, 1994b) were identified and used in this HHRA. When Cal/EPA CSFs were not available, USEPA values were used. Thus, the following hierarchy of available sources was used to select CSFs for each of the COPCs: California Cancer Potency Factors (Cal/EPA, 1994b), USEPA's Integrated Risk Information System (IRIS)(USEPA, 1998) and USEPA's Health Effects Assessment summary Table (HEAST)(USEPA, 1997a as cited by USEPA Region IX, 1998). At the present time, Cal/EPA and USEPA have developed CSFs only for the oral and inhalation routes of exposure.

Table 3-2. Cancer Slope Factors (mg/kg-day)-1 for chemicals identified in groundwater at Daggett Ave.

	USEPA			Cal/EPA		
Chemical	Oral CSF	Inhalation CSF	Source	Oral CSF	Inhalation CSF	Source
1,1 DCE	6 E-1	1.8E-1	IRIS USEPAR9	Not listed	Not listed	Cal/EPA,1994
1,1,1-TCA	NA	NA	IRIS	Not listed	Not listed	Cal/EPA,1994
TCE	1.1E-2	6E-3	USEPAR9	1.5E-2	1.0E-2	Cal/EPA,1994
PCE	5.2E-2	2E-3	USEPAR9	2.1E-2	5.1E-2	Cal/EPA,1994

USEPAR9 = USEPA Region IX
NA = not available

In general, quantitative cancer risk characterization is performed for carcinogens in Groups A, B1 and B2. Quantitative risk characterization for Group C carcinogens is performed on a case-by case basis because the depth of evidence in support of an association between chemical exposure and cancer varies from chemical to chemical. In cases where the evidence is weal, a quantitative assessment is not recommended. In this HHRA, 1,1 DCE, a Group C carcinogen is evaluated quantitatively.

Currently, the USEPA is proposing significant changes to its carcinogenic risk assessment guidelines of 1986, resulting in a more flexible and less default driven cancer risk assessment. The Proposed Guidelines for Carcinogen Risk Assessment (USEPA, 1996a) could fundamentally change the way the agency assesses cancer risk. However, none of these proposals have been carried out to modify the current carcinogenic risk assessment methodology presented in this HHRA.

3.2 Non-carcinogenic Dose-Response Assessment Methodology

Typically, potential non-carcinogenic effects resulting from human exposure to chemical constituents can be estimated quantitatively using Reference Doses (RfDs) for ingestion route or Reference Concentrations (RfCs) for inhalation. As was the case for CSFs, RfDs and RfCs are only available for

oral and inhalation exposures. In the absence of criteria specific to the dermal pathway, the oral toxicity values and oral absorption adjustment factors may be used to evaluate the dermal route of exposure.

RfD and RfC values are developed by the USEPA RfD Work Group on the basis of a wide array of non-carcinogenic health effects. The RfD, expressed in units of mg/kg-day, is an estimate (with uncertainty spanning an order of magnitude or more) of the daily maximum level of exposure to human populations (including sensitive sub-populations) that is likely to be without an appreciable risk of deleterious effects during a designated time period of exposure (USEPA, 1989a).

The RfC is expressed in units of milligrams of chemical per cubic meter of air (mg/m3) and is an estimate of the maximum air concentration the can be present over a specified period of time without an appreciable risk of deleterious effects. Assuming a human adult body weight of 70 kg and an inhalation rate of 20 m^3/day, an inhalation RfD, in units of mg/kg-day, may be derived from the RfC value.

RfDs and RfCs are derived from either human studies involving work place exposures or from animal studies. They are based on the no-observed-adverse-effect level (NOAEL) or the lowest-observed-adverse-effect level (LOAEL) using generic uncertainty factors. The RfD provides a benchmark against which human intakes of chemicals resulting from exposure to environmental media are compared.

Exposure duration considered in the development of RfDs and RfCs is typically divided into three categories (USEPA, 1989a):

- **Acute** refers to exposure for short durations, measured in seconds, minutes, hours or days and to effects that appear promptly after exposure (USEPA, 1989a).

- **Subchronic** refers to exposures of intermediate duration from two weeks to seven years.

- **Chronic** refers to prolonged or repeated exposures and effects that develop only after exposures from seven years to a lifetime.

In this risk assessment, exposure duration for complete exposure pathways includes subchronic and chronic exposures. However, only chronic RfDs were used in the risk calculations. Cal/EPA has not derived RfDs for chemical constituents. Therefore, only the USEPA-derived chronic RfDs were used in this risk assessment; these values were found in IRIS (USEPA, 1998) or were derived by USEPA Region IX (1998).

3.3 Chemicals Lacking Toxicity Values

The IRIS database (USEPA, 1998) does not have RfDs for both the inhalation and oral routes of exposure for all the COPCs in this risk assessment. As a result, we have used the "Preliminary Remediation Goals" published by USEPA Region IX (1998), as a source for these missing values. When necessary, USEPA Region IX (1998) supplements these values with "temporary" RfDs available from USEPA's National Center for Environmental Assessment (NCEA). If no value was available by the inhalation route of exposure, then USEPA Region IX extrapolated from the oral route.

Table 3-3: Reference Doses (RfD) for Chemicals of Concern in Groundwater at the Daggett Avenue Site.

Chemical	Oral RfD (mg/kg-day)	Source	Inhalation RfD (mg/kg-day)	Source
1,1-DCE	9.0 E-3	IRIS	9.0 E-3	Extrapolated from oral RfD
1,1,1-TCA	3.5 E-2	NCEA	2.9 E-1	NCEA
TCE	6.0 E-3	HEAST	6.0 E-3	Extrapolated from oral value
PCE	1.0 E-2	IRIS	1.1 E-1	NCEA

3.4 Toxicity Profiles of COPCs

Four chemicals have been continuously monitored at the McKesson Chemical Co. property and are the subject of the required quarterly monitoring reports. The toxicity information for these chemicals are presented in Appendix 1.

SECTION 4.0

EXPOSURE ASSESSMENT

4.0 EXPOSURE ASSESSSMENT

Exposure is defined in the USEPA risk assessment guidelines as the contact of a receptor with a chemical or physical agent (USEPA, 1989a; 1992a). The exposure assessment determines the quantities or concentrations of the risk agents received by the potentially exposed populations (NAS, 1983;USEPA, 1992a). Because exposure assessment is used to determine the need for a remedial action to protect human health under both current and future land-use conditions, its emphasis is on calculating risk to individuals or small population groups (USEPA, 1990; 1992a) based on exposure scenarios evaluation. Thus, exposure assessment is generally performed by determining the concentrations of chemicals in a medium at a location of interest (termed exposure point concentrations [EPCs]) and linking this information with the time that individuals or populations contact the chemicals (time of contact). Exposure assessment also involves estimating human exposures from multiple routes, such as ingestion, inhalation and dermal contact (USEPA, 1989a;1992a), through a combination of direct measurements and mathematical models.

In accordance with risk assessment guideline (USEPA, 1989a;1992a), an exposure assessment consists of the following components:

- Characterization of the exposure setting
- Identification of complete/potentially complete exposure pathways and
- Quantification of exposure, including EPCs and daily intake doses.

4.1 Characterization of the Exposure Setting

Potential exposure to chemicals in the environment depends on a number of factors related to the physical characteristics of the site and its surroundings. These factors include site location, geology and hydrogeology, climatic conditions, demography and surrounding land uses. They also include factors related to potentially exposed populations such as current and future land uses of the property, which determines the types of human activities that might occur at the site, the degree to which the site is accessible to the general public and the mechanisms that might allow migration of chemicals to onsite and off-site locations. The information contained in the following sections was derived primarily from the Levine Fricke 1995 Phase 1 Evaluation and the Terrasearch, 1998 Phase I Evaluation.

4.1.1 Site Location and Description

The subject site is located on the western margin of the East Bay Hills immediately east of the San Francisco Bay within Union City, California (Figure 1). Currently, the site is located within a mixed commercial, light industrial and residential development The property is bounded to the southeast by Daggett Ave. and Higgens Lumber Co.. To the south west the property is bounded by 7th Street that is lined with light industrial operations (Figure 2). The former McKesson Chemical Co. property bounds the site to the northwest and vacant land, a residential development and mission boulevard bound it to the northeast.. The local topography of the area is relatively flat at 65 - 70 feet above mean sea level (msl). The foothills of the East Bay lie immediately east of the site and across Mission Blvd. Alameda Creek is located approximately 3,000 ft south of the site (Terrasearch, 1998).

Figure 1

map

Location of site within the township of Union City

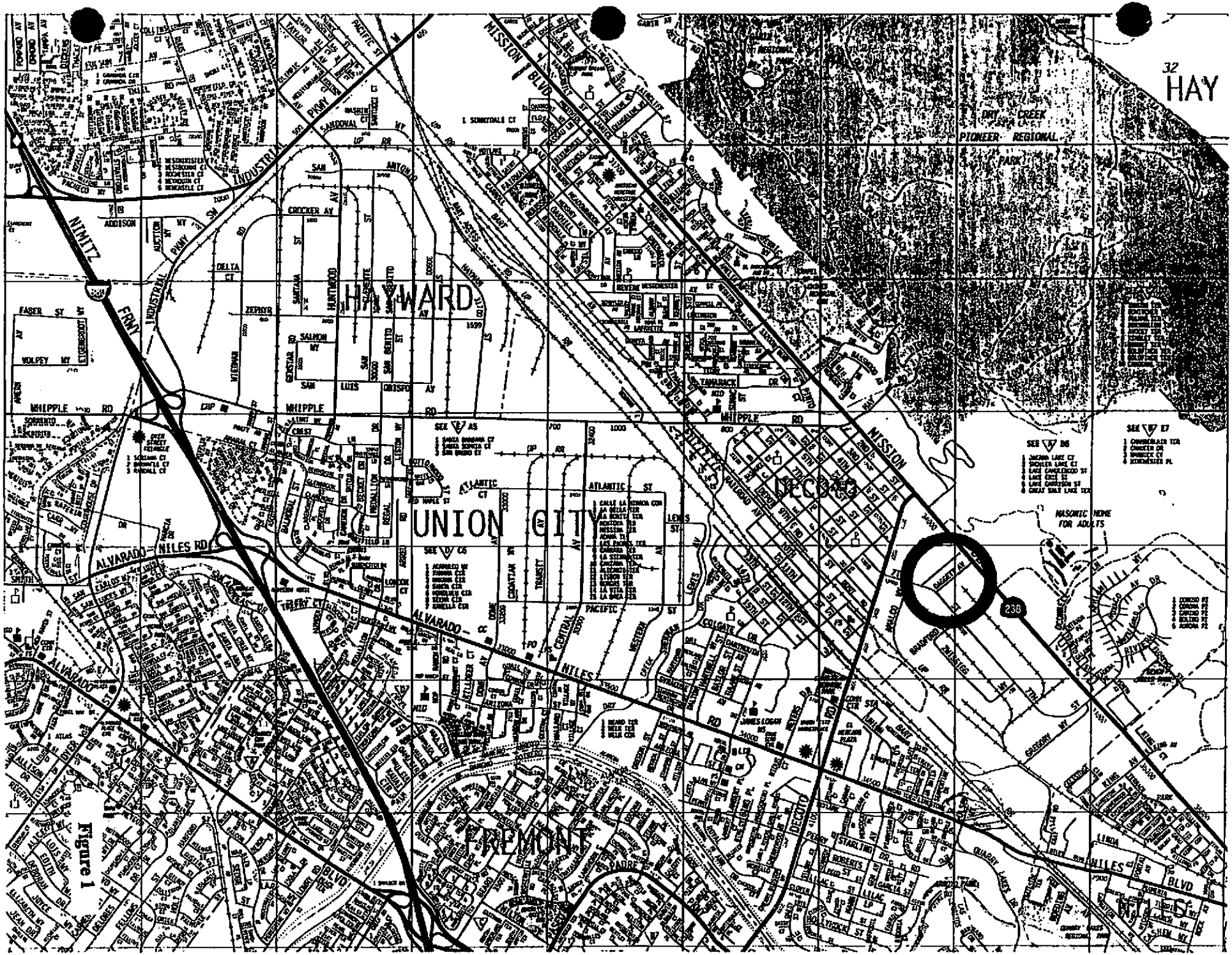

HAYWARD
UNION CITY
FREMONT
NILES
ALVARADO-NILES RD
WHIPPLE RD
MISSION BLVD
NIMITZ FWY
INDUSTRIAL PKWY
DRY CREEK PIONEER REGIONAL PARK
MASONIC HOME FOR ADULTS
880

Figure 1

Figure 2

map

Site plan for 621 Daggett Avenue

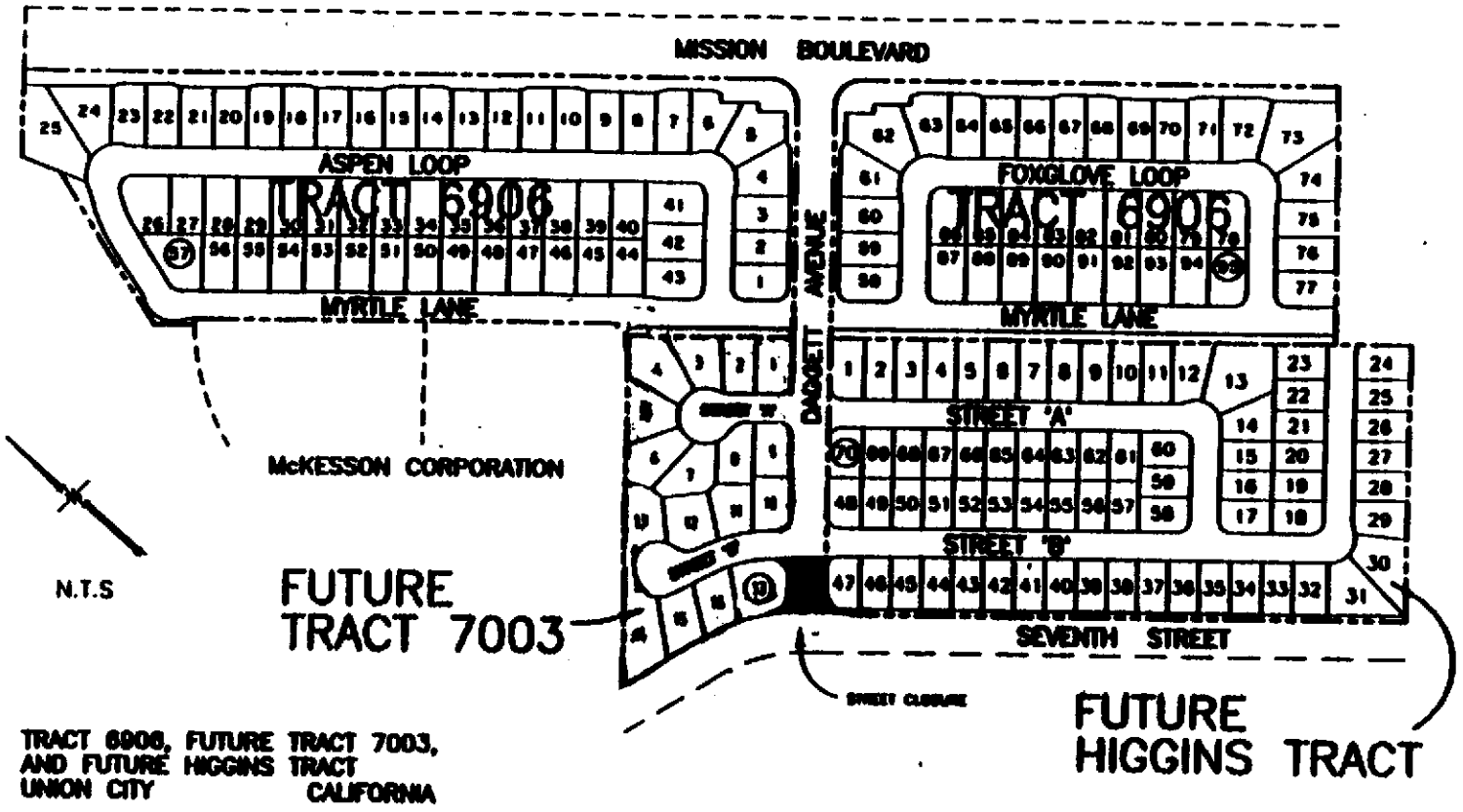

MISSION BOULEVARD
ASPEN LOOP
TRACT 6906
MYRTLE LANE
DAGGETT AVENUE
FOXGLOVE LOOP
TRACT 6906
MYRTLE LANE
STREET 'A'
STREET 'B'
McKESSON CORPORATION
N.T.S
FUTURE
TRACT 7003
SEVENTH STREET
STREET CLOSURE
FUTURE
HIGGINS TRACT
TRACT 6906, FUTURE TRACT 7003,
AND FUTURE HIGGINS TRACT
UNION CITY CALIFORNIA

Figure 2

4.1.2 Geology/Hydrology

The underlying sediments of the site consist of Pleistocene all, consisting of weakly consolidated, slightly weathered, poorly sorted irregular interbedded clay, silt, sand and gravel. These alluvial sediments were deposited were deposited by former streams flowing predominantly from Niles Canyon, located to the south of the property. These sediments comprise the Niles Cone and Dry Creek Cone alluvial fan (Levine-Fricke, 1995). Near surface sediments consist predominantly of clayey-silt to silty-clay to depths of approximately 50 ft bgs. Sediments encountered at the depth interval of 50 to 100 bgs are variable ranging from silty clay to sandy gravel.

The Hayward Fault is situated along the base of the Diablo Range less than 0.05 miles northeast of the site. The Hayward Fault is also considered active according to the Alquist-Priolo Earthquake Fault Zones Act.

First encountered groundwater beneath the site is approximately 40 ft bgs. This is based on wells located in the vicinity of the site; namely the McKesson Chemical Co. facility . The McKesson Facility files quarterly groundwater monitoring reports as part of their required remedial action. Based on the May 1998 report, groundwater is encountered in three aquifer zones labeled "shallow" with a depth in the range of 50 - 60 ft, "intermediate" with a depth in the range of 100 ft. and "deep" with a depth in the range of 150 ft. The Groundwater from all three zones flow to the south-south west at a gradient of approximately 0.004 ft/ft. (Omega, 1998).

4.1.3 Climatic conditions

Union City is located on the eastern border of San Francisco Bay, the foothills of the East Bay hills are located directly to the east of the city. Several ephemeral creeks flow from the hills and drain into the bay. The climate is characterized by windy, dry summers and cool, wet winters. The average annual temperature is in the range of 58 degrees F based on a winter temperature of 52 - 54 degrees F and an average summer temperature of 60 - 62 degrees F. The average rainfall is approximately 20 inches/year falling mostly between October and April. The prevailing winds are predominantly from the west-northwest with an average speed of 3.22 meters/sec (National Weather Service, 1997).

4.1.4 Demography and Anticipated Land Use

The general area of the subject site for this HHRA consists of agriculture, light industry and residential housing. The Higgens Lumber Company is located directly across the street and adjacent to the property is the former Oxford Tire Company. Other light industrial complexes in the vicinity include Air Liquid, Williams Bros. Construction Co., Pacific Lumber Coast Mill Works, the former Amalco Metals Company and the former McKesson Chemical Company. Residential multiunit housing is also located within a few blocks of the site.

Up until 1985, the site was used for agricultural purposed or left vacant. At that time, City Freight Lines built a large warehouse-like structure and operated a trucking company on the property. From 1990 to 1995, Clipper Express , also a trucking company operated from the site. In 1995 the land was bought by the Catellus Development Corporation in anticipation of turning the site into a residential housing area.

Access to the site is currently limited. The property is surrounded by a 6 foot cyclone fence and appears to be used to store excess soil. There are several large soil mounds on the site but there does not appear to be any active development at this time. Aside from the occasional construction worker, there are no other potential receptors on the property per se.

According to the Union City Building Department, the size of the site is 3.3 acres (143,748 ft^2.) and destined to be developed into 17 building/housing lots of varying size. The anticipated residences will consist of 2 story buildings with approximately 1,875 (Plan 1)[1] to 2,300 ft^2 (Plan 3) of living space (see Figure 3). The second story of the residences will contribute approximately one third of the available living space in all of the units. The foundation foot print of the housing units ranges from approximately 1,440 ft^2 (Plan 1) to 1,850 ft^2 (Plan 3). The residences will surround two Dead End street courts with 8-9 lots around each court. The paved street area is expected to occupy approximately 26, 000 ft^2. (estimated from Site Map, Figure 2) of the property parcel.

4.1.5 Characterization of Potentially Exposed Populations

Potential exposures to site related constituents are a function of the current and future land uses of the site and its surrounding area. Currently, the site is vacant and aside from an occasional construction worker, there are no current on-site receptors. However, a major residential development complex is under construction on neighboring properties and the anticipated use of the subject site is to be incorporated into this development. As a result, three potentially exposed populations have been identified for this HHRA.

The chemical contamination associated with this site is primarily associated with the migration of contaminated groundwater located approximately 60 feet bgs. However, volatile chemicals may migrate by emissions, specifically by emission from the groundwater through the soils and into the ambient air. Therefore two potential receptors can be identified; the Future Construction/Utility Worker and Future Residents.

- Future Construction/ Utility Worker: Future Construction/utility workers could be exposed to contamination in soils and air. Limited exposure to shallow groundwater and VOC emissions from shallow groundwater could also occur. Such exposures would be short term, such as during excavation for a building foundation or installation of underground utility lines (sewer, storm drains, etc.).

- Future Residents: In accordance with risk assessment guidance, the hypothetical residential receptor population was divided into adults and children. These receptors were selected for the future on-site residential land use scenario.

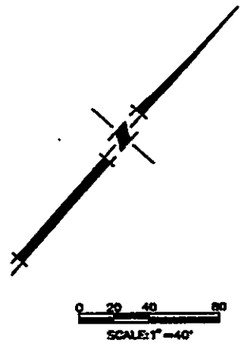
4.2 Identification of Exposure Pathways

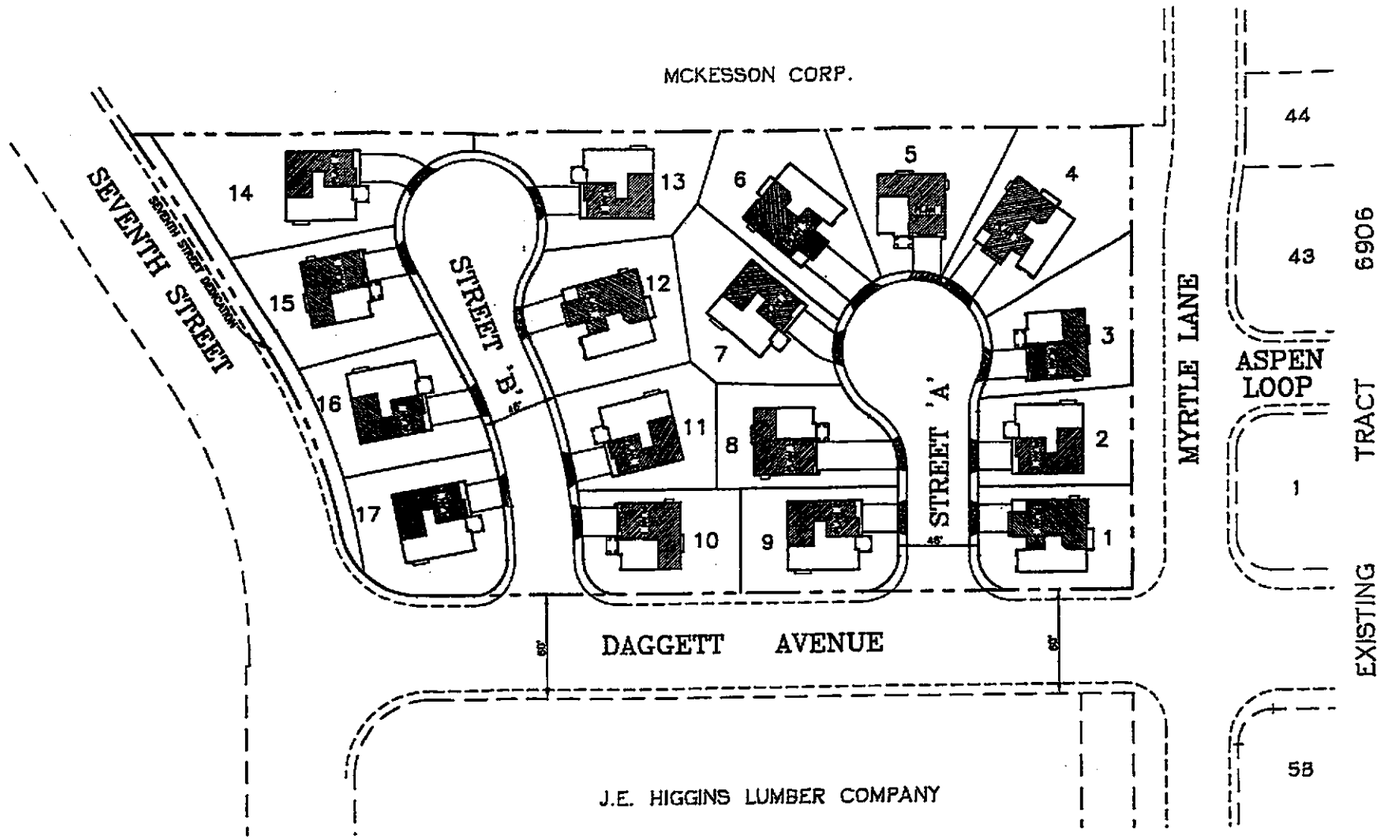
An exposure pathway is considered "complete" if there is direct evidence that a chemical has actually migrated from the source to a point of exposure where the receptors assimilate the chemical into their body. However, exposure does not actually have to be occurring. An exposure pathway is considered "potentially complete" if a source exists and conditions at the site indicate that such a chemical is expected to migrate to an exposure point in the future; no direct evidence of migrations or exposure is required. An exposure pathway is considered 'incomplete" if one of the above four elements is missing

[1] Plan refers to the architectural drawings for the different styles of housing units

Figure 3

Site Development Plan for Tract 7003





UNIT MIX

	NO. LOTS	%
PLAN 1	5	29.4
PLAN 2	8	47.1
PLAN 3	4	23.5
TOTAL	17	100

SITE SUMMARY

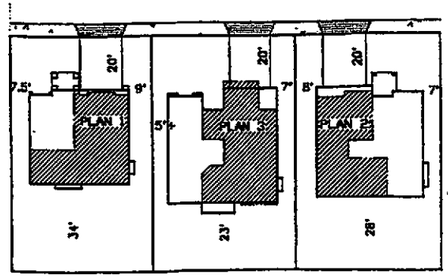
GROSS AREA	3.1 AC.
NET AREA	2.5 AC.
NUMBER OF UNITS	17
GROSS DENSITY	5.5 DU/AC.
NET DENSITY	6.8 DU/AC.

SETBACKS SHOWN ARE BASED ON 55'X 90' LOTS
MINIMUM SETBACKS PER THOSE ESTABLISHED
BY DIPSA AND THE DEVELOPMENT AGREEMENT.

FRONT YARD SETBACKS	20' MIN.
REAR YARD SETBACKS	15' MIN.
SIDE YARD SETBACKS	5' MIN.
STREET SIDE YARD SETBACKS	10' MIN.

BUILDING SEPARATION	10' BETWEEN ONE STORY ELEMENTS
BUILDING SEPARATION	15' BETWEEN TWO STORY ELEMENTS
BUILDING SEPARATION	12.5' BETWEEN COMBINATION ONE AND TWO STORY ELEMENTS

NOTE: UNIT MIX AND SITING SUBJECT TO CHANGE
PER DESCRETION OF DEVELOPER



TYPICAL SITING PLAN
NTS

Figure 3.

SITE DEVELOPMENT PLAN
TRACT 7003

LANDS OF CATELLUS RESIDENTIAL GROUP, INC.
CITATION HOMES — APPLICANT

UNION CITY — CALIFORNIA

DATE: JAN. 5, 1998
DRAWN BY: BGC
CHK'D BY: GD

MACKAY & SOMPS
CIVIL ENGINEERING • LAND PLANNING • LAND SURVEYING
PLEASANTON, CA. 94588-3333 (510) 225-0690

JOB NO.: 19018-40
DWG. NO.:
SHEET 1 OF 1

REV. 3/5/98

and that exposure cannot occur either now or in the future. Only complete or potentially complete pathways are evaluated in this HHRA.

The characterization of the potential exposure pathways at the subject site and whether each pathway is complete of potentially complete is presented in the Conceptual Site Model (CSM) for the site (Figure 4). Based on several possible release and transport mechanisms, potential receptors may be exposed to chemicals in the groundwater wither directly or indirectly as a result of chemical migration into air.

4.2.1 Sources/Retention/Transport Mechanisms of Release

The primary sources of chemicals in soils are from chemical spills or leaks from storage tanks. The released chemical seeps into the ground, through the soil column and into the groundwater. The chemical will then migrate according to its relationship with the groundwater. In general, VOCs in soil or groundwater could volatilize and migrate vertically and laterally through permeable zones of along man-made conduits. The vapors can migrate vertically upwards or downwards, depending upon their density relative to the air and their chemical diffusion gradients and potentially affecting the land surface. Vapors released at the surface may disperse into ambient air or migrate into the indoor air environment of existing or future structures.

VOCs can rapidly partition between the soil pore air and pore water and between gas, water and chemically active sites on the surfaces of soil particles. In the pore air, the VOCs can spread in all directions under advective and diffusional forces. Vapors released at the surface may disperse into ambient air or migrate into the indoor environment of existing or future structures. If contaminated groundwater were used as tap water, then exposures to COPCs could occur, both by ingesting the water directly and by inhaling constituents which volatilize during the normal daily use of water such as showering, toilets, dishwashing, *etc.* (USEPA, 1991b)

4.2.2 Exposure Points and Routes

Based upon the various migration pathways, there are several points of human contact with site related chemicals in the groundwater. The likelihood for potential receptors to contact site-related contaminants is summarized in the CSM. These potential receptors were identified based on current and future land uses for the site. When similar exposure routes exist for two or more receptors populations, the receptors with the greatest exposure (onsite location) was evaluated quantitatively as a bench mark for the others. If no significant risk was associated with the on-site receptor, then it was implicit that no significant risks was associated with off-site receptor and it was not necessary to explicitly quantify their exposures. Thus, under future land use conditions, only on-site construction workers and on-site residents were evaluated.

4.2.3 Summary of Exposure Pathways

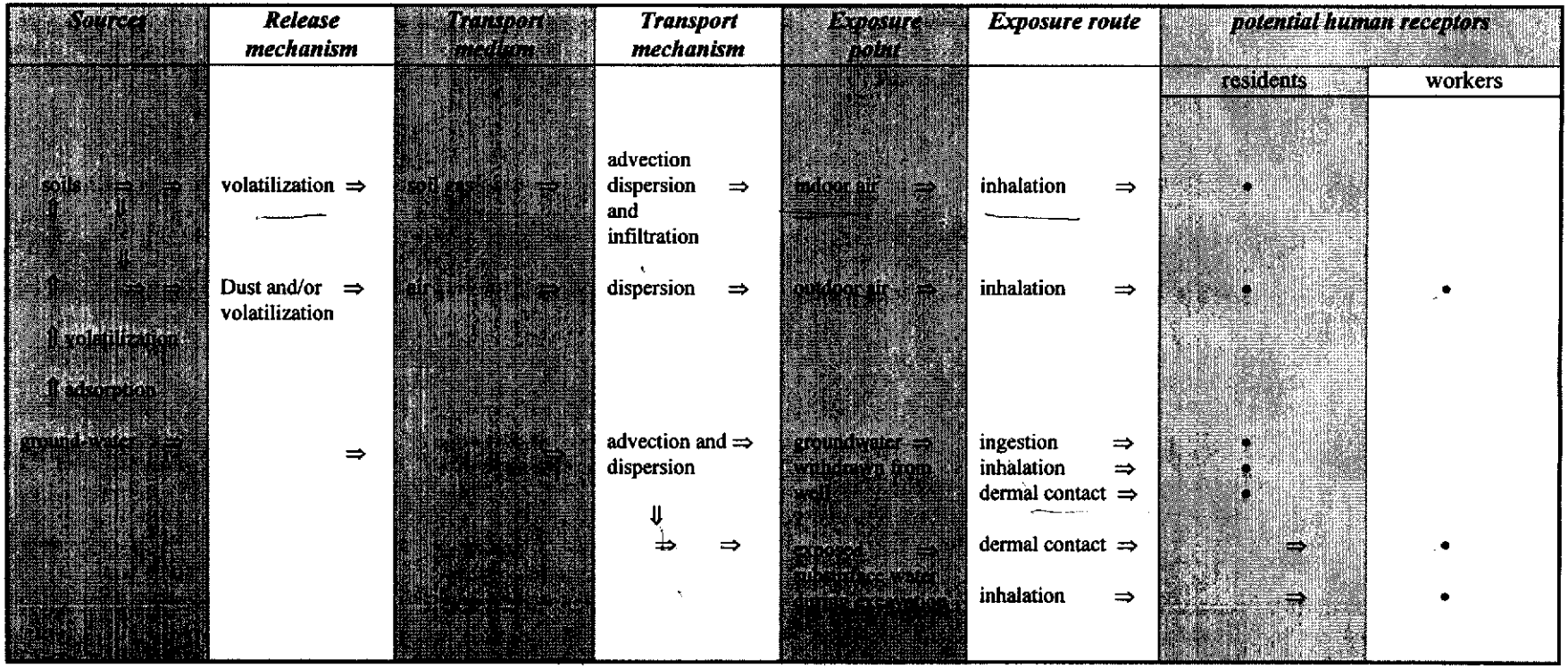
Given the characteristics of the COPCs and chemical release processes discussed above, the exposure pathways and their rationale for selection or exclusion are discussed in the following subsections.

Future on-site workers are potential receptors under the construction/utility workers involved with site redevelopment may be exposed to chemicals in the groundwater through direct dermal contact with the groundwater and by inhalation of emitted VOCs. Such exposure would be short term, such as during excavation of a building foundation or installation of site infrastructure (utilities and storm sewers).

Figure 4

CONCEPTUAL SITE MODEL

Figure 4. CONCEPTUAL SITE MODEL



surface soils - dermal contact / workers
inhal / ing

surface soils - dermal contact / residents -
ind / ing

Other potential receptors include future residents; both adult and child. Under this land use condition, the house, pavement, access streets or ornamental vegetation would cover a major portion of the surface area of the property. Nevertheless, these receptors are conservatively assumed to be exposed to chemicals by inhalation due to volatilization of the VOCs in the groundwater into the ambient air and indoor air. Compared to indoor air exposure, it is anticipated that the outdoor air pathway would be insignificant. Therefore, only the potential indoor air pathway was quantitatively evaluated .

It is conceivable that residents could drill their own wells and use the contaminated groundwater as tap water. This pathway is not considered likely since the local purveyor will provide potable water supply to the future residences and it is unlikely that the groundwater beneath the site will ever be used as a source of drinking water without prior treatment. Nevertheless, this exposure is quantified here for completeness. Residents could potentially be exposed by pathways that include ingestion of water, dermal contact with water and the inhalations of VOCs volatilized from water. Dermal exposure to tap water during domestic use is not quantified here for two reasons: (i) dermal exposure to tap water is not significant compared to ingestion and inhalation and (ii) quantification of dermal exposure from tap water is highly uncertain (USEPAR9, 1998). Because residents are expected to have greater exposure frequency and duration than other potential receptors, groundwater exposure was quantitatively evaluated only for residential receptors.

A summary of the exposure pathways applicable to each future land use and associated receptors for which risks were quantitatively evaluated is presented in Figure 4.

4.3 Quantification of Exposure

Potential exposure to chemicals in the environment is directly proportional to concentrations of the chemicals in environmental media and characteristics of exposure (e.g., frequency and duration). The chemical concentrations at exposure points are generally referred to as EPCs. Human exposure doses can then be estimated using EPCs and various exposure parameters. The following subsections describe how EPCs and exposure doses are determined in this HHRA.

4.3.1 Exposure Point Concentrations

USEPA defines EPCs as the representative chemical concentrations a receptor may contact at an exposure area over the exposure period (USEPA, 1989a). The typical concept of human exposure is that individuals contact the contaminated media on a periodic and random basis. Because of the repeated nature of such contact, the human exposures do not really occur at a fixed point but rather at a variety of points that are sampled at random and with equal likelihood that any given point within the exposure area will be the contact location on any given day. Thus, the EPC should be the arithmetic averages of the chemical concentrations at various points within the exposure area.

EPCs may be estimated using either direct measurement (i.e., soil concentrations from the sampling and analysis programs) or a combination of direct measurement data and the indirect results of fate and transport modeling. Direct measurement data are used when there had been human contact with the media sampled, such as ingestion of , or dermal contact with, soil or groundwater, by the future on-site residents. For these direct-contact pathways, site-specific measured chemical concentrations are used to estimate EPCs. If exposure occurs at a point removed from the contaminated source, concentrations at the exposure point can be estimated by modeling of fate and transport of contaminants from the source to the point of contact with the receptor.

Estimating EPCs to be used in the exposure assessment of the HHRA requires an understanding of the underlying distribution. For the purposes of this HHRA, it is assumed that chemical concentrations in environmental media are distributed log-normally (Gilbert, 1987; USEPA, 1992b). Because the geometric mean is a biased estimator of the mean of the two-parameter log-normal distribution and tends to underestimate the true mean (Gilbert, 1987; USEPA 1992b), arithmetic averages, such as the 95th UCL of the arithmetic mean and the maximum likelihood estimate (***MLE***) were used in estimating the reasonable maximum exposure (***RME***) and central tendency exposure (***CTE***) scenarios, respectively (USEPA, 1992b;1992c;Gilbert, 1987). According to USEPA (1989a), a combination of a single pathway ***RME*** and other ***CTE*** risks depending upon site-specific conditions could best present multiple pathway ***RME*** risks.

The 95th UCL of the arithmetic mean is a value that, when calculated repeatedly for randomly drawn subsets of data, equals or exceeds the true mean 95 percent of the time. Although the 95th UCL of the arithmetic mean provides a conservative estimate of the average (or mean) concentrations , it should not be confused with the 95th percentile of site concentration data. As sample size increases, the UCL of the mean moves closer to the true mean while the 95th percentile of the distributions remains at the upper end of the distribution (USEPA, 1992b). When the 95th UCL of the mean is calculated repeated for randomly drawn subsets of data, it equals or exceeds the true mean 95 percent of the time. In some instances, the 95th UCL of the mean may exceed the maximum detected value, due to high sample variation or small sample size. The maximum detected concentration in environmental media is then conservatively selected to the ***RME*** EPC (USEPA, 1992b).

For the ***CTE*** the geometric mean is used, whereas the 95% upper confidence limit (95%UCL) on the mean concentration is used for the ***RME***. Estimating mean concentrations and 95%UCLs requires an understanding of the mathematical distribution of the data on concentrations. For the purposes of this HHRA, it is assumed that chemical concentrations in environmental media are distributed log-normally (Gilbert, 1987; USEPA, 1992b). Data describe a log-normal distribution if their logarithms are distributed normally.

The ***CTE*** and 95%UCL (***RME***) on the mean of a log-normal distribution are calculated as follows: (Gilbert, 1987; USEPA, 1992b):

$$CTE = exp\left(y + \frac{\sigma y^2}{2}\right)$$

$$95\%UCL = exp\left(y + \frac{\sigma y^2}{2} + \frac{\sigma y\, H}{n-1}\right)$$

Where:

CTE	=	Estimate of the central tendency of concentration (mg/L)
95%UCL	=	95% upper confidence limit on the mean of a log-normal distribution
exp	=	antilog
y	=	mean of log-transformed data (the geometric mean)

σy	=	standard deviation of the log-transformed data
H	=	H statistic for one sided (upper) confidence limit (Gilbert, 1987)
n	=	number of samples

The geometric mean is a biased estimator of the mean of the log-normal distribution in that it tends to underestimate the true mean (Gilbert, 1987; USEPA 1992b). The purpose of the H-statistic is to correct for this underestimation.

For compounds detected at least one in the media of concern, all acceptable data without validation qualifiers and data with the J estimates qualifiers were included in the statistical analysis. Non detected values were calculated assuming on-half the SQL (SQL/2), called proxy concentrations. SQLs greater than two times the maximum detected values were eliminated from the statistical analysis to avoid using unrealistically high detection limits for non detected values (USEPA, 1989a).

Because shallow groundwater represents a mobile medium, the distribution of contamination observed should be a function of shallow groundwater flow. Shallow groundwater contamination that is introduced from a source on-site likely forms a plume that spreads laterally along with the shallow groundwater flow. Because such a plume spreads and disperses as it flows, concentrations within the plume vary as a function of distance down gradient from the source. Given this phenomena, The EPCs in local shallow groundwater are conservatively defined as the concentration of COPCs in the centralized portion of the plume (USEPA, 1993).

The manner in which the exposure points are defined for shallow groundwater is designed to be conservative, by estimating the exposure points based on detected concentrations within the center of the plume. Any locations further down gradient or off the center line of the plume (from which shallow groundwater might be withdrawn) will likely exhibit lower concentrations of even non-detects. Therefore for this HHRA, EPC in the shallow groundwater were conservatively estimated based on detected concentrations from the center of the plume. All non-detects were excluded in deriving the arithmetic mean.

The following subsections describe the data used to calculate EPCs for each exposure medium evaluated at this site.

4.3.1.1 Identification of Relevant Data

The pump and treat groundwater operation at the McKesson Chemical Co. site has been in operation since November 27, 1991. During this time, the concentrations of the groundwater contaminants has decreased from the ppm range to the ppb range. Quarterly monitoring reports confirm this decrease. As such, the latest quarterly monitoring results (May, 1998) were considered to be the most representative of the current conditions at the site and would reflect a the worst condition scenario for the future residents in housing yet to be built. The average and maximum detected concentration for the four COPCs monitored in the wells surrounding the site are presented in Table 2-1. Employing the most current data set to estimate potential exposures is a conservative approach since it assumes that the chemical concentrations will remain steady over the entire exposure duration and not be subject to attenuation by further treatment, dispersion or biodegradation.

4.3.1.2 Estimation of Chemical Concentrations

The chemical concentrations in the groundwater were estimated from direct measurement of the water samples collected for the May 1998 quarterly report (Omega, 1998) Since the HHRA is an evaluation of current and future conditions, it was determined that the most current shallow groundwater data set would conservatively represent concentrations to which receptors may be exposed. There may be some concern that the exclusion of data from earlier years may result in an underestimation of potential risks. However, it is unlikely that concentrations of the solvents could increase at some point in the future based on the comparison of concentrations trends at sample locations near source areas over he last few years. Employing the most current shallow groundwater data set to estimate potential exposures is still a conservative approach since the HHRA assumes that the chemical concentrations will remain constant over time and degradation or dilution will not occur.

4.3.1.3 Estimations of Chemical Concentrations in Outdoor Air

Potential future VOC air emissions were evaluated based on the assumption that subsurface soils may be excavated. For outdoor air, emissions of VOCs from soil were modeled by estimating a transfer factor from the groundwater to the soil and into air using a standard method recommended by the American society of Testing and Materials (ASTM, 1996). This transfer factor was used in conjunction with the data on concentrations in groundwater to estimate outdoor ambient air concentrations. Multiplication of the transfer factor by the concentration in groundwater yields a concentration in ambient air. Calculations for the transfer factors are given in Attachment A. EPC estimated for outdoor air are shown in Table 4-1.

Table 4-1: Estimated Concentrations of COPCs in Indoor Air and Outdoor Air (mg/m^3) for Contaminants in Groundwater at the Daggett Avenue Property (Based on 95% UCL on Mean Concentrations in Groundwater)

	Workers	Residents			
Chemical	Outdoor Air	Outdoor Air	Indoor Air due to Vapor Intrusion	Indoor Air from Tap Water	Total for Indoor Air
1,1-Dichloroethene	1.46 E-5	1.46 E-5	3.48 E-2	5.22 E-2	8.69 E-2
1,1,1-Trichloroethane	1.52 E-7	1.52 E-7	3.63 E-4	2.64 E-2	2.67 E-2
Trichloroethylene	2.46 E-6	2.46 E-6	5.86 E-3	1.38 E-1	1.44 E-1
Tetrachloroethylene	8.82 E-6	8.82 E-6	2.10 E-2	2.04 E-1	2.25 E-1

The concentrations of contaminants in outdoor air may be described as:

$$C_{AIR-OUT} = VF_{W-AMB} * C_w$$

Where:

$C_{AIR-OUT}$ = Concentration of contaminant in ambient air (mg/m^3)

VF_{W-AMB} = Volatilization factor for groundwater to ambient air [(mg/m^3)/(mg/L)]

C_W = Concentration of contaminant in groundwater (mg/L)

4.3.1.4 Estimation of Exposure Point Concentrations in Indoor Air

Inhabitants of the homes constructed on-site in the future could be exposed to VOCs that may infiltrate into the indoor environment by two mechanisms. First, vapors can migrate from the groundwater, through the soil column, through cracks in the foundations or walls of buildings, and finally into indoor air. A transfer factor for this mechanism is described in Attachment A. Concentrations in indoor air of future structures were estimated using the method recommended by ASTM (1996). The specific models employed describe volatilization from shallow groundwater, upward transport in soil gas through soils, foundations, and floors (primarily through cracks and seams), and mixing into indoor air. The indoor air is replaced by outdoor air at a fixed rate recommended by ASTM (1996).

Second, VOCs present in tap water can be released into indoor air through normal domestic uses of water, such as bathing, laundering, dishwashing, and toilets (USEPA, 1992b). The transfer for this mechanism is a single constant, converting concentrations of chemical in tap water to concentrations in indoor air. This constant is recommended by regulatory agencies (USEPA, 1992b; USEPAR9, 1998). The constant is derived originally from descriptions of the escape of radon, an inert gas, during domestic use of water. Chlorinated solvents can be considered to be inert with respect to chemical reactivity with water, so this model has been almost universally adopted (USEPA, 1992b, USEPAR9, 1998).

Chemicals detected in shallow groundwater were used as source inputs into an indoor air volatilization model. Any VOC detected in shallow groundwater was assumed to volatilize through the soil vadose zone and into indoor air of enclosed structures. Based on this assumption, the EPCs for each of the volatile COPCs were modeled indoor air concentrations. Risks and hazards were then estimated using the CTRE and RME estimates of concentrations in air. Discussions on the uncertainties of the models and assumptions are presented in Section 6.0 of this report.

Site chemical concentrations in indoor air are composed of two contributions, one from intrusion of vapors from soil and the other from liberation of vapors during domestic use of tap water:

$$C_{AIR-IN} = C_W * (VF_{W-ENC} + VF_{W-TAP})$$

Where:

C_{AIR-IN} = Concentration of contaminant in indoor air (mg/m^3)

VF_{W-ENC} = Volatilization factor for groundwater into indoor air [(mg/m^3)/(mg/L)]

VF_{W-TAP} = Volatilization factor for tap water into indoor air [(mg/m^3)/(mg/L)]

Predicted VOC concentrations in indoor and outdoor air for future workers and residents are shown in Table 4-1. Estimated outdoor air concentrations are 3-4 orders of magnitude lower than those for indoor air. For residents, the contributions from vapor intrusion and domestic use of water are roughly equal for 1,1-DCE and 1,1,1-TCA, but tap water use contributes the great majority for TCE and PCE.

4.3.1.5 Estimation of Exposure Point Concentrations in Shallow Groundwater

In keeping with the conservative nature of the HHRA, the groundwater underlying the site was evaluated

as if it were a beneficial resource in the future. Given the remote possibility that shallow groundwater will be used in the future as a drinking water source, potential human exposures were evaluated in this HHRA using concentrations currently observed in shallow groundwater. Modeling of shallow groundwater transport was not performed for this HHRA.

Concentrations in shallow groundwater were estimated from direct measurements based on the most current conditions at the site (Table 2-1). All detected chemical concentrations from the 1998 investigation were pooled into one data set to derive EPCs that are applicable to the pathways associated with tap water use.

4.3.2 Exposure Doses

Exposure dose is defined as the amount of a chemical that a receptor contacts. The dose of a chemical that an individual receives is a function of the concentration to which the individual is exposed, the duration and frequency of such exposure, and the mechanism(s) by which the chemical is assimilated into the body.

Exposure doses in the HHRA are classified as being either lifetime average daily doses (***LADD***s) or average daily doses (***ADD***s), depending on the effects being evaluated. ***LADD***s and ***ADD***s are expressed as the amount of a substance crossing the outer body boundary per unit body weight per unit time, or mg/kg-day. The ***LADD*** is averaged over a lifetime (70 years) for carcinogenic effects. The ***ADD*** is averaged over the expected exposure duration for non-carcinogenic effects, which varies depending on the type of receptors being evaluated. ***LADD***s and ***ADD***s are calculated from the exposure point concentrations, the exposure frequency, the exposure duration, and the physical characteristics of the receptor, such as intake rate or body weight. For this HHRA, default values assumed for each of these parameters are believed to be representative of potentially exposed populations at the site (USEPA, 1991b; 1997a; 1997b).

The methods of ASTM (1996) are designed to estimate an allowable concentration in an environmental media at a predetermined or fixed risk or hazard. However, the purpose of this HHRA is to quantify risk and hazard at known (given) concentrations. As a result, the ASTM equations were rearranged to solve for risk and hazard.

For risk assessment purposes, USEPA (1989a) recommends that ***LADD***s and ***ADD***s be estimated for both ***CTE*** and ***RME*** conditions. The ***RME*** is intended to place conservative upper bounds on the potential risks, meaning that each risk estimate is likely to be overestimated. For each chemical, the doses are combined by pathway for the ***RME*** by combining the exposure point concentrations by each route of exposure. This takes into account upper-bound values describing the intake rate and extent, frequency, and duration of exposure (USEPA, 1989a). The ***CTE*** represents the average case scenario; ***CTE*** doses are estimated by using the MLE exposure point concentrations and other average exposure parameters. The following equations are used to calculate ***LADD*** and ***ADD*** for the complete or potentially complete exposure pathways identified for the HHRA USEPA, 1992a).

These are the general equations for estimating doses from environmental exposures:

$$LADD = \frac{C \times CR \times ET \times EF \times ED \times CF}{BW \times AT}$$

$$ADD = \frac{C \times CR \times ET \times EF \times CF}{BW}$$

Where:

LADD	=	Lifetime Average Daily Dose of chemical for carcinogenic effects (mg chemical/kg body weight/day)
ADD	=	Average Daly Dose of chemical for non-carcinogenic effects (mg/kg-day).
CR	=	daily intake of environmental medium (L water/day or m^3 air/day)
ET	=	Exposure time (hr/day)
EF	=	Exposure frequency (days/year)
ED	=	exposure duration years)
BW	=	body weight (kg)
AT	=	Averaging time for carcinogenic effects (days)
CF	=	Conversion factor for units (as appropriate)

When calculating doses for non-carcinogenic effects, ***ED = AT***, so these factors are missing from the equation describing ***ADD***.

It is convenient to combine the ***CR, EF,*** and ***BW*** into a pathway exposure factor or ***PEF***, which is expressed in units of L/kg-day for drinking water and m^3/kg-day for air:

$$PEF = \frac{CR \times EF}{BW \times 365}$$

These factors are specific to an exposure setting and do not vary by chemical. The spreadsheets used to calculate risks and hazards in this HHRA made use of ***PEFs***. In calculating the ***LADD*** for residents, the value for is ***30*** yr. This is constructed as a weighted average of 6 yr. exposure as a child and 24 yr. exposure as an adult. ***ADDs*** use ***PEFs*** for children only. Workers are assumed to be adults.

4.3.2.1 Ingestion of Water by Residents

The ***LADDs*** and ***ADDs*** via ingestion of COPCs in soil and shallow groundwater are calculated from the following equation:

$$LADD = \frac{C_{WATER} * PEF_{WATER-RES} * ED_{RES}}{AT}$$

$$ADD = C_{WATER} * PEF_{WATER-CHILD}$$

Where:

C_{WATER} = Concentration of chemical in water (mg/L), from Table 2-1.

ED_{RES} = Exposure duration for a resident (yr)

$PEF_{WATER-RES}$ = $\frac{IRW_{RES} * EF_{RES}}{BW_{RES} * 365}$

and:

IRW_{RES} = Ingestion rate of water for a resident (L/day), weighted average

EF_{RES} = Exposure frequency for residents

BW_{RES}= Body weight for resident (kg), weighted average

365 = day/yr.

4.3.2.2 Inhalation of Outdoor Air by Residents

The ***LADD*** and ***ADD*** for inhalation of COPCs in outdoor air by residents is calculated as follows:

$$LADD = \frac{C_{AIR-OUT} * PEF_{RES-OUT} * ED_{RES}}{AT}$$

$$ADD = C_{AIR-IN} * PEF_{INH-CHILD-OUT}$$

Where:

$C_{AIR-OUT}$ = Concentration of chemical in outdoor air (mg/m^3) from Table 4-1.

$PEF_{RES-OUT}$ = Pathway exposure factor for inhalation of outdoor air by residents (m^3/kg-day)

$PEF_{INH-CHILD-OUT}$ = Pathway exposure factor for inhalation of outdoor air by resident children (m^3/kg-day)

VF_{W-AMB} is described in Attachment A. C_W is taken from Table 2-1. $PEF_{RES-OUT}$ is a weighted average of breathing rates, exposure frequencies, exposure times, and body weights for a combined residential exposure of 6 yr. as a child and 24 yr. as an adult:

$$PEF_{INH-RES-OUT} = \frac{\left(\frac{EF_{RES}}{365}\right)\left[\left(\frac{6 * BR_{RES-CHILD} * ET_{RES-CHILD-OUT}}{BW_{CHILD}}\right) + \left(\frac{24 * BR_{RES-ADULT} * ET_{RES-ADULT-OUT}}{BW_{ADULT}}\right)\right]}{6 * BW_{CHILD} + 24 * BW_{ADULT}}$$

Where:

EF_{RES}	=	Exposure frequency for residents (day/yr.)
365	=	Conversion factor (day/yr.)
$BR_{RES-CHILD}$	=	Breathing rate for resident children (m^3/hr)
$ET_{RES-CHILD-OUT}$	=	Exposure time outdoors for the resident child (hr/day)
BW_{CHILD}	=	Body weight of resident child (kg)
$BR_{RES-ADULT}$	=	Breathing rate for resident adult (m^3/hr)
$ET_{RES-ADULT-OUT}$	=	Exposure time outdoors for the resident adult (hr/day)
BW_{ADULT}	=	Body weight of resident adult (kg)

Residential exposure is divided into 6 yr. as a child and 24 yr. as an adult; hence, the values 65 and 24 in the equation above. The ***PEF*** for non-carcinogenic effects is simpler:

$$PEF_{INH-CHILD-OUT} = (EF_{RES} / 365) * \frac{BR_{RES-CHILD} * ET_{RES-CHILD-OUT}}{BW_{CHILD}}$$

Where:

$ET_{RES-CHILD-OUT}$	=	Outdoor exposure time of a resident child (hr/day)

4.3.2.3 Inhalation of Indoor Air by Residents

Contaminants in groundwater can reach indoor air by two mechanisms, so the estimation of dose has two components, one for intrusion of vapors from soil and the other for liberation of vapors from domestic use of tap water. Thus, the ***LADD*** and ***ADD*** are each made up of two terms:

$$LADD = \frac{C_{AIR-IN} * PEF_{INH-RES-IN} * ED_{RES}}{AT}$$

$$ADD = C_{AIR-IN} * PEF_{INH-CHILD-IN}$$

$PEF_{INH-RES-IN}$ and $PEF_{INH-CHILD-IN}$ are similar to their counterparts for outdoor exposure. The first is a weighted average and the second is for children only:

$$PEF_{INH-RES-IN} = \frac{\left(\frac{EF_{RES}}{365}\right)\left[\left(\frac{6 * BR_{RES-CHILD} * ET_{RES-CHILD-IN}}{BW_{CHILD}}\right) + \left(\frac{24 * BR_{RES-ADULT} * ET_{RES-ADULT-IN}}{BW_{ADULT}}\right)\right]}{6 * BW_{CHILD} + 24 * BW_{ADULT}}$$

Where:

$ET_{RES-CHILD-IN}$ = Exposure time outdoors for the resident child (hr/day)

$ET_{RES-ADULT-IN}$ = Exposure time outdoors for the resident adult (hr/day)

$$PEF_{INH-CHILD-IN} = (EF_{RES} / 365) * \frac{BR_{RES-CHILD} * ET_{RES-CHILD-IN}}{BW_{CHILD}}$$

4.3.2.4 Inhalation of Outdoor Air by Workers

For the purposes of this HHRA, construction/utility workers are exposed to outdoor air only. C_{AIR} is the same as for residents; but the ***PEF*** is simpler, because no weighting is needed:

$$LADD = \frac{C_{AIR-OUT} * PEF_{WORK} * ED_{WORK}}{AT}$$

$$ADD = C_{AIR-OUT} * PEF_{WORK}$$

$$PEF_{WORK} = \frac{BR_{WORK} * ET_{WORK} * EF_{WORK}}{BW_{WORK}}$$

Where:

ET_{WORK} = Exposure time at work (hr/day)

EF_{WORK} = Exposure frequency for workers (day/yr.)

ED_{WORK} = Exposure duration for the worker (yr.)

BW_{WORK} = Body weight of workers

The ***RME*** values for the construction worker are 250 day/yr for 1 yr, whereas the corresponding values for the utility are 10 day/yr for 25 yr. Thus, the two exposure settings are equivalent in terms of dose.

4.3.3 Exposure Parameters

Exposure parameters are used to estimate exposure doses in various media. The ***CTE*** and ***RME*** exposure parameters selected are based on site-specific factors, when available, or on regulatory guidance (Cal/EPA, 1992; USEPA, 1989b;1991b;1992e;1995;1997), or site-specific factors, when applicable.

Tables 4-2 and 4-3 present the ***CTE*** and ***RME*** exposure parameters for the worker and resident, respectively.

Ingestion of water by residents was taken to be 2 L/day for the ***RME*** adult and 1 L/day for the ***CTE***. Values for the child were one-half of these (USEPA, 1991a; DTSC, 1992). For the inhalation route, USEPA (1997b;1997c) has recently published values which are somewhat lower than previous recommendations (USEPA, 1991a). Hourly inhalation rates for the resident adult, resident child, and worker are 1.10, 0.33, and 1.4 m^3/day. In order to divide inhalation exposure for the resident between indoor and outdoor air, values from USEPA (1997b;1997c) were used. ***CTE*** and ***RME*** values for the resident adult are 21.5 hr/day indoors, 1.5 hr/day outdoors, and 1 hr/day in automobiles. This latter 1 hr/day was not included in this HHRA. Values for resident children are 6 hr/day outdoors and 18 hr/day indoors. ***CTE*** and ***RME*** values for ***ED, EF,*** and ***BW*** were taken from USEPA (1991a) and DTSC (1992), with one exception. The ***CTE*** value for ***EF*** for the resident is 137 day/yr, which is the mean of the distribution recommended by DTSC for use with Cal/TOX (DTSC, 1996). This HHRA includes no exposures via ingestion of or dermal contact with soil.

4.4 Summary

The calculations of estimated doses for the complete exposure pathways identified in this section are presented in Tables 4-4 through 4-7. These dose estimates will he combined with the toxicity values presented in the Toxicity Assessment (Section 3.0) in the Risk Characterization (Section 5.0) to estimate potential carcinogenic risks and non-carcinogenic health effects.

Table 4-2: Exposure Parameters for Workers

Parameter	Name	Units	*CTE*	*RME*	Sources/Comments
Active breathing rate	BR_{WORK}	m3/kg-hr	1.40 E+0	1.40 E+0	USEPA, 1997a
Exposure time for worker, active work	ET_{WORK}	hr/day	8.00 E+0	8.00 E+0	USEPA, 1997a
Exposure frequency for inhalation by worker	EF_{WORK}	day/yr.	3.00 E+1	2.50 E+2	USEPA 1991a
Pathway exposure factor for inhalation by worker	PEF_{WORK}	m3/kg-day	1.10 E-1	1.10 E-1	$= BR_{WORK} * ET_{WORK} * EF_{WORK}/365$
Exposure duration for worker, carcinogenic effects	ED_{WORK}	yr.	1.00 E+0	1.00 E+0	Assumed
Body weight for worker	BW_{WORK}	kg	6.20 E+1	7.00 E+1	USEPA 1991a
Averaging time for carcinogenic effects	*AT*	yr.	7.00 E+1	7.00 E+1	USEPA 1991a

Table 4-3: Exposure Parameters for Residents

	Name	Units	CTE	RME	Sources/Comments
1. Parameters Used for Both Inhalation and Tap Water					
Exposure frequency for residents	EF_{RES}	day/yr.	1.37 E+2	2.50 E+2	CTE - DATCAL.XLS; *RME* - USEPA, 1991a
Exposure duration for adult	ED_{ADULT}	yr.	2.40 E+1	2.40 E+1	USEPA 1991a
Exposure duration for child	ED_{CHILD}	yr.	6.00 E+0	6.00 E+0	USEPA 1991a
Exposure duration for resident, Carcinogenic effects	ED_{RES}	yr.	3.00 E+1	3.00 E+1	USEPA 1991a
Body weight of resident children	BW_{ADULT}	kg	6.20 E+1	7.00 E+1	CTE - DATCAL.XLS; *RME* - USEPA, 1991a
Body weight of resident adults	BW_{ADULT}	kg	6.20 E+1	7.00 E+1	USEPA 1991a
Averaging time for carcinogenic effects	AT	yr.	7.00 E+1	7.00 E+1	USEPA 1991a
2. Parameters Used for Inhalation					
Breathing rate for resident adult	$BR_{RES\ ADULT}$	m3/hr	1.10 E+0	1.10 E+0	USEPA, 1997b
Breathing rate for resident child	$BR_{RES\ CHILD}$	m3/hr	0.333	0.333	USEPA, 1997b
Exposure time outdoors for resident adult	$ET_{RES\ ADULT\ OUT}$	hr/day	1.50 E+0	1.50 E+0	USEPA, 1997c
Exposure time indoors for resident adult	$ET_{RES\ ADULT\ IN}$	hr/day	2.15 E+1	2.15 E+1	USEPA, 1997c
Exposure time for outdoors resident child	$ET_{RES\ CHILD\ OUT}$	hr/day	6.00 E+0	6.00 E+0	USEPA, 1997c
Exposure time indoors for resident child	$ET_{RES\ CHILD\ IN}$	hr/day	1.80 E+1	1.80 E+1	USEPA, 1997c
Pathway exposure factor for inhalation of outdoor air for residents, adults + children	$PEF_{INH\_RES\_OUT}$	m3/kg-day	3.42 E-4	5.28 E-4	$= (EF_{RES}/365)*((6 * BR_{RES\_CHILD} * ET_{RES\_CHILD\_OUT} / BW_{CHILD}) + (24 * BR_{RES\_ADULT} * ET_{RES\_ADULT\_OUT} / BW_{ADULT})) / (6 * BW_{CHILD} + 24 * BW_{ADULT})$
Pathway exposure factor for inhalation of indoor air for residents, adults + children	$PEF_{INH\_RES\_IN}$	m3/kg-day	2.75 E-3	4.07 E-3	$= EF_{RES} * ((6 * BR_{RES\_CHILD} * ET_{RES\_CHILD\_IN} / BW_{CHILD}) + (24 * BR_{RES\_ADULT} * ET_{RES\_ADULT\_IN} / BW_{ADULT})) / (6 * BW_{CHILD} + 24 * BW_{ADULT})$
Pathway exposure factor for inhalation of outdoor air by resident children	$PEF_{INH\_CHILD\_OUT}$	m3/kg-day	5.00 E-2	9.12 E-2	$= (EF_{RES} / 365) * BR_{RES\_CHILD} * ET_{RES\_CHILD\_OUT} / BW_{CHILD})$
Pathway exposure factor for inhalation of indoor air by resident children	$PEF_{INH\_CHILD\_IN}$	m3/kg-day	1.50 E-1	2.74 E-1	$= (EF_{RES} / 365) * BR_{RES\_CHILD} * ET_{RES\_CHILD\_IN} / BW_{CHILD})$

Table 4-3 (cont'd): Exposure Parameters for Residents

3. Parameters for Tap Water					
	Name	**Units**	***CTE***	***RME***	**Sources/Comments**
Ingestion rate for drinking water for resident adult	IRW_{ADULT}	L/day	1.00 E+0	2.00 E+0	***RME*** - USEPA Region IX, 1998: ***CTE*** - assumed
Ingestion rate for drinking water for resident adult	IRW_{CHILD}	L/day	5.00 E-1	1.00 E+0	***RME*** - USEPA Region IX, 1998: ***CTE*** - assumed
Pathway exposure factor for ingestion of water for resident children	PEF_{WATER_CHILD}	L/kg-day	1.25 E-2	4.57 E-2	$= (IRW_{CHILD} * EF_{RES}) / (BW_{CHILD} * 365)$
Pathway exposure factor for ingestion of water for residents	PEF_{WATER_RES}	L/kg-day	6.79 E-3	2.48 E-2	$= (6 * PEF_{WATER\_CHILD} + 24 * (IRW_{ADULT} * EF_{RES}) / (BW_{ADULT} * 365)) / 30$
Volatilization factor for volatile organic chemicals into indoor air from domestic use of tap water	VF_{W_TAP}	(mg/m3)/(mg/L)	5.00 E-1	5.00 E-1	USEPA Region IX, 1998

Table 4-4: Lifetime Average Daily Doses (*LADD*) for Carcinogenic Effects and Average Daily Doses (*ADD*) for Non-Carcinogenic Effects for Workers Exposed to Chemicals of Concern in Outdoor Air at Central Tendency Exposures (*CTE*) and Reasonable Maximum Exposures (*RME*)

	LADD for Workers Breathing Outdoor Air (mg/kg-day)		*ADD* for Workers Breathing Outdoor Air (mg/kg-day)	
Chemicals of Concern in Groundwater	***CTE***	***RME***	***CTE***	***RME***
1,1-Dichloroethene	1.61 E-8	2.28 E-8	1.13 E-6	1.60 E-6
1,1,1-Trichloroethane	1.69 E-10	2.38 E-10	1.18 E-8	1.67 E-8
Trichloroethylene	2.77 E-9	3.85 E-9	1.94 E-7	2.70 E-7
Tetrachloroethylene	1.15 E-8	1.38 E-8	8.02 E-7	9.66 E-7

Table 4-5: Lifetime Average Daily Doses (*LADD*) for Carcinogenic Effects and Average Daily Doses (*ADD*) for Non-Carcinogenic Effects for Residents Exposed to Chemicals of Concern in Outdoor Air at Central Tendency Exposures (*CTE*) and Reasonable Maximum Exposures (*RME*)

	LADD for Residents Breathing Outdoor Air (mg/kg-day)		*ADD* for Residents Breathing Outdoor Air	
Chemicals of Concern in Groundwater	***CTE***	***RME***	***CTE***	***RME***
1,1-Dichloroethene	1.51 E-9	3.30 E-9	5.14 E-7	1.33 E-6
1,1,1-Trichloroethane	1.58 E-11	3.45 E-11	5.39 E-9	1.39 E-8
Trichloroethylene	2.59 E-10	5.57 E-10	8.84 E-8	2.24 E-7
Tetrachloroethylene	1.07 E-9	2.00 E-9	3.66 E-7	8.04 E-7

Table 4-6: Lifetime Average Daily Doses (*LADD*) for Carcinogenic Effects and Average Daily Doses (*ADD*) for Non-Carcinogenic Effects for Residents Exposed to Chemicals of Concern in Indoor Air from Vapor Intrusion and Domestic Use of Tap Water at Central Tendency Exposures (*CTE*) and Reasonable Maximum Exposures (*RME*)

	LADD for Residents Breathing Indoor Air(mg/kg-day)		*ADD* for Residents Breathing Outdoor Air(mg/kg-day)	
Chemicals of Concern in Groundwater	***CTE***	***RME***	***CTE***	***RME***
1,1-Dichloroethene	2.97 E-5	6.21 E-5	9.19 E-3	2.38 E-2
1,1,1-Trichloroethane	7.20 E-7	1.41 E-6	2.84 E-3	7.32 E-3
Trichloroethylene	7.19 E-6	1.43 E-5	1.56 E-2	3.95 E-2
Tetrachloroethylene	2.43 E-5	4.26 E-5	2.80 E-2	6.15 E-2

Table 4-7: Lifetime Average Daily Doses (*LADD*) for Carcinogenic Effects and Average Daily Doses (*ADD*) for Non-Carcinogenic Effects for Residents Exposed to Chemicals of Concern by Ingestion of Tap water Central y Exposures (*CTE*) and Reasonable Maximum Exposures (*RME*)

	LADD for Residents Ingesting Tap Water (mg/kg-day)		*ADD* for Residents Ingesting Tap Water (mg/kg-day)	
Chemicals of Concern in Groundwater	***CTE***	***RME***	***CTE***	***RME***
1,1-Dichloroethene	4.07 E-6	1.88 E-5	5.00 E-4	4.77 E-3
1,1,1-Trichloroethane	2.06 E-6	9.50 E-6	2.53 E-4	2.41 E-3
Trichloroethylene	1.10 E-5	4.98 E-5	1.35 E-3	1.26 E-2
Tetrachloroethylene	1.87 E-5	7.34 E-5	2.30 E-3	1.86 E-2

SECTION 5.0

RISK CHARACTERIZATION

5.0 RISK CHARACTERIZATION

Risk characterization is the culmination of the risk assessment process (USEPA 1992a). It integrates the results of the identification of COPCs, toxicity assessment, and exposure assessment to describe the risks to individuals and populations in terms of extent and severity of probable adverse health risks under both current and future land-use conditions. The overall quality of the assessment is also presented, including the confidence on the risk estimates. In this HHRA, the risk characterization process involved integrating the exposure doses and toxicity values to estimate two types of potential health effects, carcinogenic and non-carcinogenic. Because carcinogenic and non-carcinogenic effects are assumed to occur by different mechanisms of action, different methods must be used to evaluate these effects. This section is divided into four subsections. Sections 5.1 and 5.2 discuss the risk characterization methodology for both carcinogenic and non-carcinogenic effects, respectively. Section 5.3 presents the summary discussion and tabulation of risks.

Various demarcations of acceptable risks have been established. Chemical-specific carcinogenic risks in excess of 1×10^{-5} per chemical have been deemed unacceptable pursuant to the California Safe Drinking Water and Toxic Enforcement Act of 1986, otherwise known as. Proposition 65 (California Health and Safety Code, Sec. 252495 *et seq.*; 22 California Code of Regulations, Sec 12703(b)). USEPA has recommended that sites posing a cumulative carcinogenic risk above 1×10^{-4} probably require remedial action (USEPA, 1991c), while carcinogenic risks in the range of 1×10^{-4} to 1×10^{-6} fall into the "risk management range". In this range, cancer risks might be acceptable or might require remedial action, depending upon site-specific conditions. Potential non-carcinogenic health effects are not considered to be of concern by both California and USEPA if the Hazard Index (***HI***) is equal to or less than 1.0.

5.1 Carcinogenic Risk Characterization

In order to estimate the theoretical upper-bound excess lifetime carcinogenic risk associated with exposure to a chemical, the product of the chemical- and the pathway-specific CSF and the ***LADD*** is determined, as shown below:

$$Risk = LADD * CSF$$

Where:

Risk = Probability of cancer occurrence in excess of background rates (unitless)

LADD = Lifetime average daily dose (mg/kg-day)

CSF = Cancer Slop factor (kg-day/mg)

This approach to estimating carcinogenic risk assumes that the increased risk resulting from exposure to a chemical is linearly proportional to the amount of chemical intake averaged over a lifetime. The potential carcinogenic risks associated with the exposures to multiple chemicals were estimated by summing the chemical-specific risks to yield pathway-specific risks. Implicit in this approach is the assumption that potential carcinogenic risks from multiple chemical exposures are additive, such that the total pathway-specific risk is equal to the sum of the individual chemical-specific risks. Similarly, the excess lifetime carcinogenic risks for each carcinogenic compound were also summed for all exposure pathways. The resulting multi-

pathway chemical-specific risks represent the upper-bound potential risk of developing cancer from exposure to a chemical present in various media.

5.2 Non-carcinogenic Effects

Adverse non-carcinogenic effects are evaluated by comparing the estimated ***ADD*** of a chemical to its associated ***RfD***. The ***RfD*** is the point of reference for evaluating the potential effects of non-carcinogenic chemical exposures. Exposure doses equal to or less than the ***RfD*** are not likely to be associated with adverse health effects and are, therefore, not of regulatory concern. However, ***ADD***s which exceed the RD are considered to present likelihood for adverse effects. The relationship is expressed numerically using the Hazard Quotient (***HQ***). The ***HQ*** is obtained by dividing a chemical-specific ***ADD*** by its respective ***RfD*** as presented below.

$$HQ = \frac{ADD}{RfD}$$

Where:

HQ	=	Hazard Quotient for one chemical by one route of exposure (unitless)
ADD	=	Average daily dose for a chemical by one route of exposure (mg/kg-day)
RfD	=	Reference dose chemical-and route-specific (mg/kg-day)

Chemical-specific and pathway-specific ***HQ***s for all chemicals across multiple pathways are summed to yield the hazard Index (***HI***):

$$HI = \sum_{i}^{n} HQ_i$$

Where:

HI	=	Hazard Index (unitless)
HQ_i	=	Hazard Quotient for the *i*th chemical via the *i*th route of exposure (unitless)

HQs and ***HI***s are not statistical probabilities, such as excess cancer risks, and the level of concern does not increase linearly as the RfD is approached or exceeded. For regulatory purposes, when $HI < 1.0$ is considered to be an acceptable level of non-carcinogenic hazard (USEPA, 1989a:1991c). If the pathway-specific or cumulative exposure $HI > 1.0$, segregation of the ***HQ*** may be considered, based on the type of effects or mechanisms of action (USEPA 1989a).

5.3 Risks and Hazards for the Daggett Avenue Property

Potential carcinogenic risks and non-carcinogenic hazards associated with exposures to COPCs found in shallow groundwater were estimated for two future land use scenarios, construction/utility workers and future residents. Estimates associated with the ***CTE*** and ***RME*** exposures are presented in Tables 5-1 through 5-4.

5.3.1 Residential Land Use Scenario

Potential receptors under the residential land use scenario are adult and child residents. They are assumed to be exposed to COPCs via inhalation of ambient air containing vapors of volatile COPC which have migrated upwards through soil from shallow groundwater. In the unlikely event that future residents would use shallow groundwater as a source of tap water (in lieu of connecting to municipal water supplies), ingestion of COPC in tap water and inhalation of COPC liberated into indoor air are also evaluated. Contact with soil is a potentially complete pathway for residents, but no COPC are known to be present in soils at the site.

For this HHRA, it was assumed that residential exposures might occur anywhere within the site. Because on-site residents are unlikely to be exposed to shallow groundwater directly beneath the site, the risks associated with shallow groundwater exposure pathways are presented separately from the risks and hazards due to inhalation of outdoor air. Both ***CTE*** and ***RME*** estimates of chemical-specific carcinogenic risks for the complete and potentially complete exposure pathways are summarized below.

5.3.2 Risks and Hazards to Residents Due To Intrusion of Vapors Indoors and Outdoors

Residential risks are estimated for a weighted average of 6 yr as a child and 24 yr as an adult. Non-carcinogenic hazards are estimated for children only. Carcinogenic risks and non-carcinogenic hazards for the ***CTE*** and ***RME*** are given in Table 5-1 for the inhalation of indoor and outdoor air with COPC which have migrated from shallow groundwater. The exposure of residents to outdoor air is similar to that of workers, as described in Chapter 4. Comparison of Tables 5-1 and 5-4 shows that outdoor air makes only a small contribution to total risk and hazard.

Table 5-1 shows that risks at both the ***CTE*** and ***RME*** are greater than the "point of departure" of 1×10^{-6}, but less than the probable remedial action level of 1×10^{-4}. These risks fall into the "risk management range" described above. 1,1-DCE contributes nearly 100% of the contribution to total risk, but this solvent is a Class C carcinogen. Non-carcinogenic hazard is less than the benchmark of 1.0 for the ***CTE***, but greater than 1.0 for the ***RME***. Hazards do not necessarily increase proportionally as ***HI*** exceeds 1.0. Nevertheless, any level greater than 1.0 is cause for concern.

5.3.3 Risks and Hazards to Residents Due To Use of Shallow Groundwater as Tap Water

It is unlikely that shallow groundwater at the Daggett Avenue site will be used as tap water. However, Table 5-2 present a summary of risks and hazards to residents ingesting tap water and inhaling indoor air with volatile COPC liberated during the domestic use of tap water.

Risks at both the ***CTE*** and ***RME*** are similar in magnitude to those posed by intrusion of vapors. Once again, 1,1-DCE contributes more than the other carcinogenic COPC, but not as much as that seen for vapor intrusion only (Table 5-1).

The non-carcinogenic hazard is greater than the benchmark of 1.0 for both the ***CTE*** and the ***RME***. The greatest contributions to ***HI*** come from TCE, although individual hazards at the ***RME*** exceed 1.0 for 1,1-DCE and PCE. Hazards do not necessarily increase proportionally as

HI exceeds 1.0. Nevertheless, any level greater than 1.0 is cause for concern.

5.3.4 Risks and Hazards for Residents Considering All Pathways

Table 5-3 presents a similar pattern to that of Table 5-2. Risks at the ***CTE*** and ***RME*** range from about 9×10^{-6} to 3×10^{-5}. 1,1-DCE is the principal driver of carcinogenic risk for all receptors. *HI* exceeds 1.0 for 1,1-DCE, TCE, and PCE, with the total *HI* in excess of 14.

5.3.5 Risks and Hazards To Construction/Utility Workers

As described in Chapter 4, the construction worker and the utility worker are exposed in different time patterns, but their total risks and hazards are numerically the same. These workers are exposed to outdoor air only. Table 5-4 shows that risks and hazards are very low and insignificant.

5.4 Summary

The groundwater under the property located at 621 Daggett Ave. in Union City, California has been contaminated with chlorinated solvents that appear to have migrated from neighboring properties. Chlorinated solvents are highly volatile materials and have the potential to migrate vertically from the groundwater into overlying structures. As such, they have the potential to pose an inhalation hazard to humans living or working in such structures. However, this HHRA has determined the following:

1. Risks and hazards attributable to the inhalation of chlorinated solvents do not exceed benchmarks for the construction/utility worker.

2. For the future resident exposed to vapor intrusion only, cancer risks are estimated for the ***RME*** at about 1×10^{-5} for vapor intrusion only and 2×10^{-5} for residents using tap water. The total for residents exposed via all pathways is also about 3×10^{-5}. The principal risk driver is 1,1-DCE, which is a Group C carcinogen.

3. Also for the future resident, non-carcinogenic hazards exceed the benchmark of 1.0 for vapor intrusion combined with the use of tap water. Drivers are TCE, PCE, and 1,1-DCE.

Table 5-1: Risks and Hazards for Future Residents Exposed to Volatile Chemicals of Concern in Shallow Groundwater That Have Migrated to Indoor And Outdoor Air.

	Excess Cancer Risk for Residents from Vapor Intrusion		Hazard Index for Resident Children from Vapor Intrusion	
Chemicals of Concern in Groundwater	**CTE**	***RME***	**CTE**	***RME***
1,1-Dichloroethene	5.20 E-6	1.09 E-5	4.09 E-1	1.06 E+0
1,1,1-Trichloroethane	0.00 E+0	0.00 E+0	1.33 E-4	3.43 E-4
Trichloroethylene	2.98 E-8	6.13 E-8	1.05 E-1	2.67 E-1
Tetrachloroethylene	4.31 E-7	7.69 E-7	2.38 E-2	5.23 E-2
Total Risk/Hazard	**5.66 E-6**	**1.17 E-5**	**5.38 E-1**	**1.38 E+0**

Table 5-2: Risks and Hazards for Future Residents Exposed to Chemicals of Concern in Shallow Groundwater by Ingestion of Drinking Water and Inhalation of Vapors Liberated during Domestic Use of Tap Water.

	Excess Cancer Risk for Residents Ingesting Tap Water and Inhaling Indoor Air		Hazard Index for Resident Children Ingesting Tap Water and Inhaling Indoor Air	
Chemicals of Concern in Groundwater	**CTE**	***RME***	**CTE**	***RME***
1,1-Dichloroethene	2.59 E-6	1.16 E-5	6.69 E-1	2.12 E+0
1,1,1-Trichloroethane	0.00 E+0	0.00 E+0	1.69 E-2	9.37 E-2
Trichloroethylene	1.35 E-7	5.73 E-7	2.71 E+0	8.42 E+0
Tetrachloroethylene	1.05 E-6	3.94 E-6	4.60 E-1	2.37 E+0
Total Risk/Hazard	**3.78 E-6**	**1.61 E-5**	**3.86 E+0**	**1.30 E+1**

Table 5-3: Summed Risks and Hazards for Future Residents Exposed to Chemicals of Concern in Shallow Groundwater by Inhalation of Indoor and Outdoor Air and Ingestion of Tap Water

	Cancer Risk for Residents Via All Pathways		Hazard Index for Resident Children Via All Pathways	
Chemicals of Concern in Groundwater	**CTE**	***RME***	**CTE**	***RME***
1,1-Dichloroethene	7.79 E-6	2.25 E-5	1.08 E+0	3.17 E+0
1,1,1-Trichloroethane	0.00 E+0	0.00 E+0	1.70 E-2	9.41 E-2
Trichloroethylene	1.64 E-7	6.34 E-7	2.82 E+0	8.69 E+0
Tetrachloroethylene	1.48 E-6	4.71 E-6	4.84 E-1	2.42 E+0
Total Risk/Hazard	**9.44 E-6**	**2.78 E-5**	**4.40 E+0**	**1.44 E+1**

Table 5-4: Risks and Hazards for Construction/Utility Workers Exposed to Volatile Chemicals of Concern in Shallow Groundwater That Have Migrated to Outdoor Air

	Excess Cancer Risk for Workers from Vapor Intrusion		Hazard Index for Workers from Vapor Intrusion	
Chemicals of Concern in Groundwater	**CTE**	***RME***	**CTE**	***RME***
1,1-Dichloroethene	2.90 E-9	4.11 E-9	1.25 E-4	1.78 E-4
1,1,1-Trichloroethane	0.00 E+0	0.00 E+0	4.07 E-8	5.76 E-8
Trichloroethylene	1.66 E-11	2.31 E-11	3.23 E-5	4.49 E-5
Tetrachloroethylene	2.41 E-10	2.90 E-10	7.29 E-6	8.78 E-6
Total Risk/Hazard	**3.15 E-9**	**4.42 E-9**	**1.65 E-4**	**2.31 E-4**

SECTION 6.0

UNCERTAINTIES

6.0 UNCERTAINTIES

The purpose of this section is to describe the uncertainties associated with the health risk estimates presented in this HHRA in order to assist risk managers in environmental decision making. The procedures used in the HHRA result in conditional estimates of risk that incorporate numerous assumptions concerning chemical toxicity and human exposure and unavoidably uncertainties. Although measurements or survey techniques may be used to better quantify a variety of site-specific factors that can replace some generic assumptions, even the most site-specific risk assessment is still subject to uncertainty.

To be health protective, the types of assumptions typically incorporated into a risk assessment are conservative. They are biased in such a manner as to cause risks to be over-estimated rather than under-estimated. However, the degrees to which biases are introduced into risk assessments are not comparable from one assessment to the next. Consequently, it is important that the magnitude of uncertainties and biases that are incorporated in risk assessments are considered when interpreting the health risk results.

Uncertainty and bias are potentially introduced into a risk assessment during each of the following major stages including:

- Identification of COPCs;
- Toxicity assessment;
- Exposure assessment; and
- Risk characterization.

The sources of uncertainty and bias associated with each of these HHRA stages are discussed in detail below.

6.1 Identification of Chemicals of Potential Concern

The predominant sources of uncertainty and potential bias associated with COPC identification derive both from the procedures employed for site investigation (including sampling plan design and the methods employed for sample collection, handling, and analysis) and from the procedures employed for data evaluation. The most important data that were used to support this HHRA were the measured chemical concentrations in the shallow groundwater. These data were used to derive EPCs for potential receptors under both current and future land use conditions.

The sampling data included in this risk assessment are likely biased towards higher concentrations due to the selection of the sampling locations. Only wells surrounding the site were selected since there were no wells directly on the site. As a result, the wells chosen may not provide a representative estimate of the chemical distribution at the site. It is, therefore, likely that the estimated mean and 95% UCL of the mean based on these sample values will result in an overestimate of the true mean concentrations of COPCs present.

Another source of uncertainty is the assumption that shallow groundwater beneath the Daggett Ave. property will be used for domestic purposes, particularly as a potable water source. This is

unlikely since current planning indicates that water will be supplied by the local municipal utility district. Thus, the estimated risks from potential exposures to constituents observed in shallow groundwater to future on-site residents represent extremely conservative estimates of risk.

6.2 Toxicity Assessment

There are a number of inherent uncertainties associated with the estimation of potential toxicity to humans associated with the low-leveled exposure that typically occurs at a contaminated site.

Unlike Cal/EPA, the USEPA classifies 1,1,DCE as a Group C material (i.e. a possible human carcinogen) and provides a CSF (IRIS, 1998). Since the predominant risk associated with the site is contributed by 1,1 DCE and the carcinogenic status is uncertain, 9t is highly unlikely that the cancer risk has been underestimated in this HHRA.

In another arena, the CSFs are considered to be plausible upper bounds of risk at a 95% confidence level. Thus, there is a high probability that the true risks do not exceed these levels and are much lower than the estimated values. The USEPA Carcinogenic Assessment Group (USEPA, 1986a) states that the use of upper-bound risk estimates is appropriate for estimating the upper-bounds of cancer risk, but that the lower limit of risk may be as low as zero (USEPA, 1989a).

The USEPA modeling approach to determine COPC-specific CSFs includes the assumption that there is no threshold for carcinogenicity. Thus, even a single molecule of a potential carcinogen is assumed to produce an increase in cancer risk. Carcinogenicity is also assumed to be independent of the exposure period, meaning once exposed, people remain at risk for the remainder of their lives.

For chronic non-carcinogenic effects, there are protective mechanisms in the body that must he overcome before a chemical can exert harmful effects on human health. Thus, the approach is to identify this tolerance range that will protect the most sensitive human receptors, or a "safe" dose called RfD. The derivation of a RfD incorporates uncertainty factors indicating the degree of confidence that can he assigned to the animal experimental data from which the RfD was derived. Virtually all of the uncertainty adjustments for non-carcinogenic oral RfDs and inhalation RfCs have large "safety factors". For example, the NOAELs from animal experiments have been lowered by several arbitrary factors of 10 (often totaling 1,000 to 10,000 fold) to account for uncertainty (e.g., variations in human sensitivity, animal-to-human extrapolations, deficiencies in available animal data). The RfD values are thought by USEPA to contain an overall uncertainty of an order-of-magnitude or more (USEPA, 1989a). Due to the degree of conservatism employed in determining RfDs and RfCs, the net result is likely to be an over-estimation of the potential non-carcinogenic health effects.

6.3 Exposure Assessment

Potential exposure to chemicals at any site is a function of current and future land uses. The current land use at the Site is vacant agricultural and the future land use is expected to be residential. Therefore the residential land uses were evaluated.

Estimation of EPCs is critical to the exposure assessment. Assuming that the concentration in the bulk medium (soil, shallow groundwater, air) is the same as the EPC is a clear source of

potential error in the exposure analysis. In this HHRA, chemical concentrations were determined by measuring the levels in the bulk media rather than at the point of contact (USEPA, 1989a and 1992a). EPCs at the Site were assumed to be constant over the exposure period; hence, the estimated exposure doses are more likely to be overestimated than underestimated. In addition, the use of 95th UCL or maximum detected values for the ***RME*** concentration term presents one of the most conservative and health protective steps in this HHRA. This degree of conservatism can be up to three orders of magnitude when comparing ***CTE*** with ***RME*** risk values.

The method by which the EPCs of COPCs in shallow groundwater was derived represents a source of uncertainty. Average and ***RME*** concentrations were calculated for the chemicals of potential concern (COPCs) as reported in the quarterly monitoring report from the McKesson Cemical Corp. and the potential exposures from shallow groundwater were calculated based on the assumption that the current chemical concentrations and site conditions would remain constant throughout the exposure period. For this HHRA, the concentrations currently observed in shallow groundwater are assumed to be the same as levels that potential receptors could be exposed to over the next 30 years. No decrease in concentrations as a result of natural attenuation was considered. Consequently, the estimated risks associated with shallow groundwater exposures may be over-estimated.

The exposure assumptions used in the HHRA were designed to quantify the ***RME*** scenario (consistent with USEPA guidance) and are very conservative. For example, the residential exposure scenario assumes that an adult will remain at the Site for 30 years. Both assumptions are conservative and have a low probability of occurring. Exposure characteristics depend largely on activity patterns that are not as easy to generalize as population characteristics. For example, it is assumed that future on-site residents and workers will have daily contact with site soils. Actual exposures to soils may be less frequent, resulting in an overestimation of risks.

The models used to predict indoor air concentrations as a result of volatilization likely over-estimated the total amount of chemicals being emitted. They conservatively assume an infinite source over the duration of the exposure period. The models also assume that 100 percent of COPCs are migrating to the surface, without accounting for potential lateral migration (which would tend to reduce the estimates of chemical flux at the surface) and biodegradation. Furthermore, the models assume that subsurface conditions are uniform across the entire Site. These assumptions may under or over-estimate the calculated health risks.

In addition, it has been well documented that the potential migration of soil vapor into indoor air is highly variable and depends on a number of site-specific factors. To account for this uncertainty, the modeling effort employed in the HHRA incorporated a number of conservative assumptions related to building type, air exchange rates, and areas of infiltration. The current indoor air infiltration model for a house assumes a slab-on-grade house construction. The area through which VOCs could pass was assumed to be the area of cracks or other breaches in the concrete. Clearly this parameter is highly dependent on specific house characteristics. Well-defined estimates of the fraction of floor space with cracks are not available. Therefore, a conservative estimate of the area (1%) was employed. Actual infiltration rates are expected to be much lower than those assumed in the HHRA.

The fate and transport indoor models are based on the assumption of steady-state conditions. Chemical concentrations used to estimate EPCs are based on measured soil or groundwater data.

The inherent assumption is that future chemical concentrations are the same as those measured during the recent field investigations. The assumption ignores the effects of various fate and transport mechanisms, which will alter the composition and distribution of chemicals present in the various media. In general, the assumption of steady-state conditions usually results in an overestimation of chemical concentrations and resulting exposure doses.

Another conservative bias introduced in this HHRA is the default volatilization constant used to estimate chemical concentrations in air from tap water use. No site-specific fate and transport modeling was performed. Of all the exposure pathways that are associated with contaminants in shallow groundwater, only one pathway (inhalation of VOCs in indoor air) is potentially complete under current or future site conditions. All other shallow groundwater pathways are only complete if shallow groundwater in the vicinity of the Site is used as a potable source of water which is extremely unlikely since the planned construction includes a municipal water supply to the homes. Therefore, the risks associated with shallow groundwater exposure represent hypothetically conservative levels.

6.4 Risk Characterization

Predicting adverse effects as a result of exposure to chemical mixtures is a major data gap in the HHRA. While some information is available regarding most individual chemicals, there is almost no information available regarding the health effects of chemical mixtures. USEPA (1986b) recommends that the cancer risks associated with exposure to multiple chemicals simultaneously be summed, thus assuming additivity. The methodology used in this risk evaluation for chemicals potentially released shallow groundwater has incorporated a number of conservative assumptions. Many of these assumptions lead to an overestimation of the most probable risks. The most probable risks are likely to be much less, perhaps zero, and probably not measurable in the potentially exposed populations.

Calculation of the dose is based upon a linear relationship between chemical concentrations and other intake parameters. According to USEPA, when constructing the exposure estimate from a series of factors (environmental concentrations, intake rates, individual activities, etc.), not all factors should be set at values that maximize exposure or dose, since it will almost always lead to an estimate that is much too conservative (USEPA, 1992a). USEPA risk guidance requires the use of upper bound values for at least three variables (contact rate, exposure frequency, and exposure duration) for the ***RME*** scenarios (USEPA, 1991b). Using a simple relationship of probability, the multiplication of three 95th percentile numbers (assuming log normal distribution) yields a value at the 99.78th percentile. This translates to an estimated risk that is a factor often to 20 or more above the intended ***RME*** risk using the Monte Carlo simulation results (Burmaster and Harris, 1993).

Given that both the estimates of exposure and toxicity employed in this report are conservative, it is highly likely that all risk estimates in this assessment are higher than risks that may actually or potentially occur. Given the attendant uncertainties discusses above, it is likely that estimates are conservative by several orders of magnitude, as depicted by the range of ***CTE*** and ***RME*** risks. Thus, the estimated risks for this site must be interpreted with caution when making risk management decisions.

SECTION 7.0

CONCLUSIONS

7.0 CONCLUSIONS

This HHRA was performed to evaluate the actual and potential risks to human health due to the chlorinated solvent at the Site, in the absence of any remedial actions and institutional controls. This evaluation was accomplished by first reviewing sampling data and identifying COPCs. The quantitative portion of the HHRA was primarily based on subsurface water data collected by Omega Inc. in May 1998. To characterize the human health risks, the results of the exposure assessment were coupled with the toxicological evaluation of the COPCs. This section summarizes the results of the HHRA.

The four chemicals in groundwater that are routinely monitored on a quarterly basis were identified as the COPCs for this site. As part of the toxicity assessment, the potential for each COPC to elicit adverse health effects was qualitatively evaluated, and toxicity values (CSFs and RfDs) determined for each COPC were summarized for use in the risk characterization process.

Site-specific potentially exposed populations identified include: future residential adults and children, and future construction/utility workers. Complete and potentially complete exposure pathways included the following:

- Incidental ingestion and dermal absorption of chemicals in on-site surface soils by future construction/utility workers;
- Inhalation of VOCs volatilized from subsurface soils and shallow groundwater to indoor air by future on-site residents.
- Inhalation of VOCs from excavation activities by future construction/utility workers;
- Inhalation of VOCs in shallow groundwater from tap water use by future residents.

The risk characterization step combines the results from the exposure and toxicity assessments to evaluate ***CTE*** and ***RME*** individual risks associated with the complete and potentially complete exposure pathways. Risk characterization results suggest that exposure to chemicals detected in soil and shallow groundwater at this site will not pose a significant risk to human health. With one exception, for the receptors considered in this HHRA, the cumulative ***CTE*** and ***RME*** risks are either near or below the trigger levels for remediation (an excess cancer risk of 1 x 10-4. And a segregated HI of one).In addition, the potential risks to these receptors are expected to be lower due to the conservative nature of the HHRA.

The only noted exception to acceptable risk is associated with the use of groundwater as a potable water supply. In this hypothetical situation, the HI for children becomes significantly elevated. However, such conditions are considered to be unlikely at this site since the planned utilities include a municipal water supply.

Uncertainty associated with the risk characterization results was qualitatively evaluated for each step of the HHRA process and then integrated to qualitatively evaluate the overall uncertainty. Based on a consideration of uncertainty associated with each step of the HHRA process, the overall uncertainty is moderate to high with a bias toward overestimation of risks.

The following notes are general qualifiers for the health risk results estimated in this HHRA:

- Under current land use conditions, remediation of constituents in shallow groundwater is not necessary. This is because the exposure pathways for water, and vapor to potential receptors are either not complete or the carcinogenic risks and non-carcinogenic health effects associated with chemicals measured in indoor air in the interior of anticipated residences are within the acceptable risk ranges. The chemical contributing the most to the cumulative carcinogenic risk and non-carcinogenic HI is 1,1 Dichloroethene.

- **Remediation of the shallow groundwater does not appear to be warranted to protect against the inhalation of VOCs in indoor air. The carcinogenic risks from inhalation of VOCs in indoor air are within the acceptable risk range of 1 x 10-4 to 1 x 10-6 under a residential scenario. Because the estimated risks may be overestimated due to the conservative nature of the assumptions and models employed to predict indoor air concentrations, the likelihood of actual potential health risks is probably low.**

- However, remediation of shallow groundwater based on future exposure pathways may be necessary, if the groundwater is to be used as a potable water supply. This is because the Hazard Index for residential children becomes significantly elevated and would trigger a regulatory response.

SECTION 8.0

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8.0 REFERENCES

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ATTACHMENT A

MODELING OF FATE AND TRANSPORT

A.1 INTRODUCTION

This section of the risk assessment describes the modeling of fate and transport used to estimate exposure point concentrations (EPC) for contaminants of potential concern (COPCs) in from groundwater to ambient air and indoor air. This method of modeling is taken from ASTM (1996). Transfer factors are defined and calculated to link concentrations of chemicals in soil to concentrations in outdoor and indoor air. This section does not describe the liberation of volatile chemicals from domestic use of tap water into indoor air. This latter process is described by a single constant, according to USEPA Region IX (1998).

The equations that are part of ASTM (1996) solve for concentrations in environmental media at target values for risk or hazard. Concentrations in groundwater are known for this risk assessment, and the object is to estimate risk and hazard. Thus, the equations were re-arranged to solve for risk or hazard. These rearrangements were programmed into Microsoft Excel spreadsheets (Office 97).

Based on the potential migration pathways identified in the Conceptual Site Model (CSM), presented in Section 4.0, the pathways for potential migration of chemicals include:

- Migration of vapors from groundwater, through subsurface soils, and into ambient air, or
- Migration of vapors from groundwater, through the soil column, through cracks in foundations of buildings, and into indoor air.

The results of these models are used to estimate EPCs that, in turn, serve as inputs to derive the intake of chemicals via inhalation. For residents, who can be exposed to indoor and outdoor vapors, the contribution from intrusion of vapors to indoor air is by far the larger of the two.

The following are the principal assumptions in this modeling exercise:

- The concentrations of contaminants in shallow groundwater are constant over the period of exposure, *i.e.* the source of contaminants is not depleted over time.
- Continuous, equilibrium partitioning occurs between shallow groundwater and chemical vapors at the shallow groundwater table.
- The chemical and physical properties of the contaminants are adequate to described the processes of volatilization and diffusion.
- Steady-state vapor- and liquid phase diffusion occurs through the capillary fringe, the vadose zone, and floor cracks and seams.
- Infiltrating soil gas is uniformly and instantaneously mixed with the entire volume of indoor air.
- No loss or dilution of chemical occurs before entering the building.

A.2 CONCENTRATIONS OF VOLATILE CHEMICALS IN OUTDOOR AIR

The equations below describe the movement of volatile chemicals from solution in groundwater, through the capillary fringe of the water table, into the air- and water-filled spaces of soil, and finally to the ground surface, where they are dispersed by wind. The process depends on both chemical-specific and landscape-specific properties. The overall process is described as follows:

$$VF_{W-AMB} = \frac{H'}{1 + \left[\frac{U_{AIR}\ MH\ L_{GW}}{W\ D_{W-S}^{EFF}}\right]} x\ 1{,}000$$

Where:

$VF_{W\text{-}AMB}$	=	Volatilization factor for chemicals migrating from groundwater to ambi ent air[$(mg/m^3)/(mg/L)$]
U_{AIR}	=	Average annual wind velocity (cm/sec)
MH	=	Mixing height (cm)
L_{GW}	=	Depth to groundwater from the surface (cm)
W	=	Width of site (cm), estimated with the square root of area of site
D_{W-S}^{EFF}	=	Effective diffusion coefficient between groundwater and soil surface (cm^2/sec)
$1{,}000$	=	Conversion factor (L/m^3)

The quantities U_{AIR} and MH represent the parameters of a "box model" to estimate the dispersion of emitted contaminants into ambient air.

The quantity D_{W-S}^{EFF} is actually composed of two diffusion coefficients, one for transit through the capillary fringe of the groundwater table, and the other for transit through the unsaturated or vadose zone of soil to reach the surface:

$$D_{W-S}^{EFF} = (h_{CAP} + h_{VAD}) \left[\frac{h_{CAP}}{D_{CAP}^{EFF}} + \frac{h_{VAD}}{D_{SOIL}^{EFF}}\right]^{-1}$$

Where:

h_{CAP}	=	Height of the capillary fringe (cm)
h_{VAD}	=	Distance from the top of the capillary fringe to the soil surface (cm)

D_{CAP}^{EFF} = Effective diffusion coefficient through the capillary fringe (cm^2/sec)

D_{SOIL}^{EFF} = Effective diffusion coefficient in soil based on vapor- concentration (cm^2/sec)

D_{CAP}^{EFF} is defined as follows:

$$D_{CAP}^{EFF} = D_{AIR}\left[\frac{\theta_{A-CAP}^{3.33}}{\theta_{TOTAL}^{2}}\right] + D_{WATER}\left[\frac{1}{H'}\right]\left[\frac{\theta_{W-CAP}^{3.33}}{\theta_{TOTAL}^{2}}\right]$$

Where:

D_{AIR} = Chemical-specific diffusion coefficient of chemical in air (cm^2/sec)

D_{WATER} = Chemical-specific diffusion coefficient of chemical in water (cm^2/sec)

θ_{A-CAP} = Air-filled porosity in capillary fringe (unitless)

θ_{A-CAP} = Water-filled porosity in capillary fringe (unitless)

θ_{TOTAL} = Total porosity of soil (unitless)

The ratio of the air- or water-filled porosity raised to the 3.33 power to the total porosity squared represents the "tortuosity" of the path followed by any molecule of vapor through the pore spaces of soil.

D_{SOIL}^{EFF} is defined in a manner similar to D_{CAP}^{EFF} :

$$D_{SOIL}^{EFF} = D_{AIR}\left[\frac{\theta_{A-SOIL}^{3.33}}{\theta_{TOTAL}^{2}}\right] + D_{WATER}\left[\frac{1}{H'}\right]\left[\frac{\theta_{W-SOIL}^{3.33}}{\theta_{TOTAL}^{2}}\right]$$

Where:

θ_{A-SOIL} = Air-filled porosity in soil column (unitless)

θ_{A-SOIL} = Water-filled porosity in soil column (unitless)

The last step is to multiply the volatilization factor by the concentration of contaminant in water to reach a concentration in air:

$$C_{AIR} = C_{WATER} x VF_{W-AMB}$$

Where:

C_{AIR} = Concentration of contaminant in air (mg chemical/m^3 air)

C_{WATER} = Concentration of contaminant in groundwater (mg chemical/L water)

A.3 CONCENTRATIONS OF VOLATILE CHEMICALS IN INDOOR AIR

The equations below describe the movement of volatile chemicals from solution in groundwater, through the capillary fringe of the water table, into the air- and water-filled spaces of soil, through cracks in the foundation of a building, and finally into indoor air. The principal difference from $VF_{W\text{-}AMB}$ is the presence of walls or foundations to form barriers to the entry of soil vapors into indoor air. As before, the calculation depends on both chemical-specific and landscape-specific properties. The overall process is described as follows:

$$VF_{W-ENC} = \frac{H'\left[\frac{D_{WS}^{EFF}/L_{GW}}{ER\,L_B}\right]}{1+\left[\frac{D_{WS}^{EFF}/L_{GW}}{ER\,L_B}\right]+\left[\frac{D_{WS}^{EFF}/L_{GW}}{\eta\left(D_{CRACK}^{EFF}/L_{CRACK}\right)}\right]} \times 1{,}000$$

Where:

$VF_{W\text{-}ENC}$ = Volatilization factor for chemicals migrating from groundwater into indoor air [$(mg/m^3)/(mg/L)$]

ER = Exchange rate of indoor air (L/sec)

L_B = Ratio of the volume of enclosed space to the area where infiltration occurs (cm)

η = Fraction of foundations or walls occupied by cracks or spaces (unitless)

D_{CRACK}^{EFF} = Effective diffusion coefficient through cracks in walls or foundation (cm^2/sec)

L_{CRACK} = Thickness of foundation or walls (cm)

$1{,}000$ = Conversion factor (L/m^3)

D_{CRACK}^{EFF} is defined similarly to other effective diffusion coefficients above:

$$D_{CRACKL}^{EFF} = D_{AIR}\left[\frac{\theta_{A-CRACK}^{3.33}}{\theta_{TOTAL}^{2}}\right] + D_{WATER}\left[\frac{1}{H}\right]\left[\frac{\theta_{W-CRACK}^{3.33}}{\theta_{TOTAL}^{2}}\right]$$

Where:

$\theta_{A\text{-}CRACK}$ = Air-filled porosity in cracks (unitless)

$\theta_{W\text{-}CRACK}$ = Water-filled porosity in cracks (unitless)

A.4 PARAMETER VALUES

Table A-1 presents the chemical and physical properties of the COPCs which are needed to estimate concentrations in indoor and outdoor air. Table A-2 presents the landscape-specific used to calculate these values. Table A-3 shows the calculated values for the various diffusion coefficients and volatilization factors.

Table A-1: Values for Chemical-Specific Properties of Contaminants of Concern in Groundwater at the Daggett Avenue Property

Chemical	Henry's Law Constant (H') (unitless)	Diffusivity in Air (D_{AIR}) (cm^2/sec)	Diffusivity in Water (D_{WATER}) (cm^2/sec)
1,1-Dichloroethylene	6.31 E+0	7.60 E-2	1.23 E-5
1,1,1-Trichloroethane	1.13 E-1	8.00 E-2	1.03 E-5
Trichloroethylene	3.66 E-1	8.10 E-2	1.07 E-5
Tetrachloroethylene	9.43 E-1	7.80 E-2	1.05 E-5
Reference:	PHYSCHEM.XLS USEPA Region IX (1998)	DATCAL.XLS DTSC (1996)	DATCAL.XLS DTSC (1996)

Table A-2: Values for Landscape-Specific Parameters at the Daggett Avenue Property

Landscape Parameter	Name of Variable	Units	Value	Source/Comment
Area of site	A	cm^2	9.27E+5	=143,780 ft^2 x $(2.54)^2$ cm^2/ft^2
Width of the site	W	cm	9.63E+2	= $A^{0.5}$
Mixing height	MH	cm	2.00E+2	Default (ASTM, 1996)
Average wind velocity	U_{AIR}	cm/sec	3.22E+2	Nat'l. Weather Service, 1977
Depth to groundwater	L_{GW}	cm	1.00 E+3	95%UCL on geometric mean for 11 wells
Height of capillary fringe	h_{CAP}	cm	5.00E+0	Default (ASTM, 1996)
Height of vadose zone	h_{VAD}	cm	9.95E+2	= L_{GW}- h_{CAP}
Volumetric air content of capillary fringe	$\theta_{A\text{-}Cap}$	(unitless)	3.80E-2	Default (ASTM, 1996)
Volumetric water content of capillary fringe	$\theta_{W\text{-}CAP}$	(unitless)	3.40E-1	Default (ASTM, 1996)
Volumetric air content of soil	$\theta_{A\text{-}SOIL}$	(unitless)	2.60E-1	Default (ASTM, 1996)
Volumetric water content of soil	$\theta_{W\text{-}SOIL}$	(unitless)	1.20E-1	Default (ASTM, 1996)
Total porosity of soil	θ_{TOTAL}	(unitless)	3.80E-1	= $\theta_{A\text{-}SOIL}$ + $\theta_{W\text{-}SOIL}$
Volumetric air content of foundation cracks	$\theta_{A\text{-}CRACK}$	(unitless)	2.60E-1	Default (ASTM, 1996)
Volumetric water content of foundation cracks	$\theta_{W\text{-}CRACK}$	(unitless)	1.20E-1	Default (ASTM, 1996)
Enclosed space air exchange rate	ER	L/sec	1.40E-4	Default (ASTM, 1996)
Enclosed space volume/infiltration area ratio	L_B	cm	2.00E+2	Default (ASTM, 1996)
Enclosed space foundation or wall thickness	L_{CRACK}	cm	1.50E+1	Default (ASTM, 1996)
Areal fraction of cracks in foundation or wall(s)	η	(unitless)	1.00E-2	Default (ASTM, 1996)

Table A-3: Calculated Diffusion Coefficients and Volatilization Factors for Chemicals of Concern in Groundwater at for the Daggett Avenue Property

Chemical	Diffusion Coefficients				Volatilization Factors	
	D_{CAP} cm^2/sec	D_{CRACK} cm^2/sec	D_{SOIL} cm^2/sec	D_{WS} cm^2/sec	$VF_{W\text{-}AMB}$ $(mg/m^3)/(mg/L)$	$VF_{W\text{-}ENC}$ $(mg/m^3)/(mg/L)$
1,1-Dichloroethylene	9.84 E-6	5.93 E-3	5.93 E-3	1.48 E-3	1.40 E-4	3.33 E-1
1,1,1-Trichloroethane	1.17 E-5	6.24 E-3	6.24 E-3	1.71 E-3	2.89 E-6	6.88 E-3
Trichloroethylene	1.09 E-5	6.32 E-3	6.32 E-3	1.62 E-3	8.88 E-6	2.12 E-2
Tetrachloroethylene	1.02 E-5	6.09 E-3	6.09 E-3	1.53 E-3	2.16 E-5	5.16 E-2

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Attachment B

Summary Table 1: Excess Cancer Risk and Hazard Index for Site Development Workers

Summary Table 2: Excess Cancer Risks for Future Residents and Hazard Index for Resident Children

Summary Table 1. Excess Cancer Risk and Hazard Index for Site Development Workers.

	Excess Cancer Risk for Workers		Hazard Index Via Inhalation for Workers	
Chemicals of Concern in Groundwater	From Mean Concentration in Groundwater	From 95%UCL Concentration in Groundwater	From Mean Concentration in Groundwater	From 95%UCL Concentration in Groundwater
1,1-Dichloroethene	2.90 E-9	4.11 E-9	1.25 E-4	1.78 E-4
1,1,1-Trichloroethane	0.00 E+0	0.00 E+0	4.07 E-8	5.76 E-8
Trichloroethene	1.66 E-11	2.31 E-11	3.23 E-5	4.49 E-5
Tetrachloroethene	2.41 E-10	2.90 E-10	7.29 E-6	8.78 E-6
Total Risk/Hazard	**3.15 E-9**	**4.42 E-9**	**1.65 E-4**	**2.31 E-4**

Summary Table 2. Excess Cancer Risks for future residents and Hazard Index for Resident Children.

	Excess Cancer Risk for Residents Due to Vapor Intrusion		Hazard Index for Resident Children Due to Vapor Intrusion	
Chemicals of Concern in Groundwater	From Mean Concentration in Groundwater	From 95%UCL Concentration in Groundwater	From Mean Concentration in Groundwater	From 95%UCL Concentration in Groundwater
1,1-Dichloroethene	5.20 E-6	1.09 E-5	1.36 E-1	3.52 E-1
1,1,1-Trichloroethane	0.00 E+0	0.00 E+0	4.43 E-5	1.14 E-4
Trichloroethene	2.98 E-8	6.13 E-8	3.51 E-2	8.91 E-2
Tetrachloroethene	4.31 E-7	7.69 E-7	7.93 E-3	1.74 E-2
Total Risk/Hazard	**5.66 E-6**	**1.17 E-5**	**1.79 E-1**	**4.59 E-1**
	Excess Cancer Risk for Residents Due to Domestic Use of Tap Water		Hazard Index for Resident Children Due to Domestic Use of Tap Water	
Chemicals of Concern in Groundwater	From Mean Concentration in Groundwater	From 95%UCL Concentration in Groundwater	From Mean Concentration in Groundwater	From 95%UCL Concentration in Groundwater
1,1-Dichloroethene	2.59 E-6	1.16 E-5	6.69 E-1	2.12 E+0
1,1,1-Trichloroethane	0.00 E+0	0.00 E+0	1.69 E-2	9.37 E-2
Trichloroethene	1.35 E-7	5.73 E-7	2.71 E+0	8.42 E+0
Tetrachloroethene	1.05 E-6	3.94 E-6	4.60 E-1	2.37 E+0
Total Risk/Hazard	**3.78 E-6**	**1.61 E-5**	**3.86 E+0**	**1.31 E+1**
	Excess Cancer Risk for Residents Exposed to Vapor Intrusion and Domestic Use of Water		Hazard Index for Children Exposed to Vapor Intrusion and Domestic Use of Water	
Chemicals of Concern in Groundwater	From Mean Concentration in Groundwater	From 95%UCL Concentration in Groundwater	From Mean Concentration in Groundwater	From 95%UCL Concentration in Groundwater
1,1-Dichloroethene	7.79 E-6	2.25 E-5	8.05 E-1	2.47 E+0
1,1,1-Trichloroethane	0.00 E+0	0.00 E+0	1.69 E-2	9.38 E-2
Trichloroethene	1.64 E-7	6.34 E-7	2.75 E+0	8.51 E+0
Tetrachloroethene	1.48 E-6	4.71 E-6	4.68 E-1	2.39 E+0
Total Risk/Hazard	**9.44 E-6**	**2.78 E-5**	**4.04 E+0**	**1.35 E+1**

APPENDIX 1

TETRACHLOROETHYLENE

1,1,1-TRICHLOROETHANE

TRICHLOROETHYLENE

1,1-DICHLOROETHYLENE

VINYL CHLORIDE

Toxicology Brief

TETRACHLOROETHYLENE

Synonyms:	Perchloroethylene; Carbon Dichloride; Carbon Bichloride; PCE, Perc.
CAS No:	127-18-4
Boiling Point:	121.4°C
Conversion Factor:	1 ppm = 6.8 mg/m^3
Flammability:	nonflammable
Henry's Law Const:	2.59×10^{-2} atm-m^3/mol
K_{oc}:	137 - 364 ml/g
Log K_{ow}:	2.6
Melting Point:	-22.7°C
Molecular Formula:	CCL_2CCL_2
Molecular Weight:	165.83
Odor:	Chloroform-like, sweet, threshold at 27 ppm; recognition at 50 ppm
Specific Gravity:	1.62
Vapor Density:	5.7 (air = 1)
Vapor Pressure:	14 mm Hg @ 20°C
Viscosity	0.839 cp @ 25°C
Water Solubility:	150 mg/l

(references: ATSDR, 1987; Verschueren, 1983; Merck, 1989; Amoore and Hautala, 1983)

GENERAL DATA

Tetrachloroethylene is a colorless liquid with chloroform-like odor. It is generally used as a dry cleaning solvent and may be handled in the presence or absence of air, water, and light with any of the common construction materials at temperatures up to 140°C. This material is extremely stable and resists hydrolysis. When heated to decomposition it emits highly toxic fumes of chlorine gas (ATSDR, 1987; Verschueren, 1983).

FATE AND TRANSPORT

The majority of tetrachloroethylene used in the U.S. is released to the **atmosphere** by volatilization, although that which has become heavily contaminated with grease and oil is disposed of in the form of liquid or solid waste, which is discharged directly to the land or surface water. When released to surface **water**, volatilization occurs rapidly, and when released to **soil**, tetrachloroethylene either volatilizes or leaches to the groundwater, due to its medium-to-high soil mobility, and low adsorption potential. Tetrachloroethylene in the atmosphere may be transported to the soil or surface water by wet deposition. Tetrachloroethylene has a low bioconcentration potential, with a bioconcentration factor (BCF) of 31 (EPA, 1986).

In the **atmosphere**, tetrachloroethylene reacts with photochemically produced hydroxyl radicals producing hydrogen chloride, phosgene and chloroacetylchlorides. Tetrachloroethylene's half- life in air is 47 days (EPA, 1986). In surface water and soil, biodegradation and hydrolysis appear to be the most important transformation processes, although these occur slowly. In surface water, a half-life from 1-30 days is reported (EPA, 1986). In groundwater, where volatilization cannot occur, tetrachloroethylene can be very persistent. Under certain conditions, tetrachloroethylene in groundwater may slowly biodegrade to trichloroethylene, and then to dichloroethylene and vinyl chloride (EPA, 1987).

TOXICITY DATA

Human Toxicology

Acute inhalation of high concentrations of tetrachloroethylene in air has been shown to produce central nervous system (CNS) effects leading to dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, and possibly unconsciousness and death. Stewart et al. (1970) reports that headache, dizziness, difficulty in speaking and sleepiness occurred in 11 individuals exposed to 101 ppm tetrachloroethylene for 7 hours. Inhalation also produces liver and kidney toxicity after chronic exposure.

The effects seen following ingestion appear to parallel those observed after inhalation. Tetrachloroethylene was previously used as a treatment for hookworm infestation, and therapeutic oral exposure produced narcotic effects, inebriation, and exhilaration at doses of 2.8 to 4 ml (4.2 to 6 g). Acute ingestion has also produced hepatic and renal toxicity. No data are available on human dermal exposure to tetrachloroethylene.

Pharmacokinetics

Tetrachloroethylene is readily absorbed following inhalation (62 - 64%) and ingestion (80 - 100%). In contrast, absorption following dermal exposure to either the vapor or the liquid is poor and not considered to add significantly to the overall toxicity (Steward and Dodd, 1964; Tsurata, 1975). The adipose tissue appears to be the primary site of tetrachloroethylene distribution. The primary urinary metabolites of tetrachloroethylene in humans appear to be trichloroacetic acid and trichloroethanol. The main excretion pathway for unmetabolized tetrachloroethylene is through expired air with a triphasic decay curve with half-lives of 12 - 16 hours, 30 - 40 hours and 50 - 55 hours (Monster et al., 1979). Urinary excretion of metabolites account for only a small percentage of the absorbed dose.

In laboratory animals, tetrachloroethylene exhibits similar pharmacokinetics as in humans with the exception that oxalic acid is also found in the urine. The metabolism of tetrachloroethylene is believed to be mediated by the cytochrome P-450 catalyzed oxidation reaction involving the formation of an epoxide intermediate.

Significant Animal Studies

The acute oral LD_{50} (single-dose 14 day) for tetrachloroethylene in male rats is 3835 mg/kg, while in female rats the value is 3005 mg/kg. Tremors, ataxia, and CNS depression preceded death, and hemorrhagic lungs and adrenals were observed in some animals (Hayes, et al., 1986). An oral LD_{50} of 8100 mg/kg was reported for mice (Wenzel and Gibson, 1951). A 4-hour inhalation LC_{50} of 5200 ppm for mice was reported by Friberg et al., (1953). Toxic nephropathy was observed in mice exposed to 386 and 1072 mg/kg tetrachloroethylene in corn oil via gavage, 5 days/week for 78 weeks. Rats exposed to 230 to 470 ppm tetrachloroethylene 8 hours/day, 5 days/week for 7 months exhibited swelling of the liver and kidney, and congestion in the spleen. The 70 ppm treatment group showed no effect.

Neurotoxicity

Damage to the CNS by tetrachloroethylene has been associated with hypertrophy and/or proliferation of astroglial cells. Rosengren et al. (1986) reported an increase in brain level S-100 (an astroglial protein) after continuously exposing guinea pigs to 320 ppm tetrachloroethylene for 3 months. Briving et al. (1986) reported a tetrachloroethylene-induced increase in free glutamine in the hippocampus, and a significant decrease in brain taurine content in gerbils exposed to 120 ppm tetrachloroethylene continuously for 12 months. Taurine is a known non-specific membrane stabilizer, thus its reduction may lead to alterations in nerve impulse transmission and could be responsible for tetrachloroethylene's neurotoxic effects.

Rosengren et al. (1986) reported a decreased DNA content in the brain of gerbils exposed continuously to tetrachloroethylene concentrations as low as 60 ppm.

Hepatotoxicity

Moderate fatty degeneration of the liver was reported in mice 1 day following a single 4-hour exposure to 200 ppm tetrachloroethylene, but not 3 days after exposure. Mice exposed similarly to 400, 800 or 1600 ppm tetrachloroethylene were observed to have moderate to massive fatty degeneration 1 and 3 days after exposure (Kylin et al., 1963). Mice exposed to tetrachloroethylene continuously for 30 days at concentrations of 9-150 ppm exhibited gross abnormal pathological appearances of the liver and significantly increased liver weights. At concentrations of 37-150 ppm, significantly increased butyrylcholinesterase activity was observed (Kjellstrand, et al., 1984). Burben and O'Flaherty (1985) reported increased liver weight and triglycerides at 100-2000 mg/kg tetrachloroethylene in corn oil, decreased G6P and increased SGPT at 500-2000 mg/kg, and histological changes in the liver at 200 and 1000 mg/kg doses.

Reproductive Toxicity

Schwartz et al. (1975) exposed pregnant rats to 300 ppm tetrachloroethylene for 7 hours/day on day 6 through 15 of gestation. Pregnant mice exhibited a 4% to 5% reduction in body weight, an increase in liver weight, and twice the number of resorptions per implantations as controls. In the mouse pups, significant subcutaneous edema, delayed skull ossification, and the presence of split sternebrae were observed.

Genotoxicity

Several genotoxicity studies have been performed for tetrachloroethylene employing a variety of in-vitro and in-vivo assays. These have revealed little or no evidence of genotoxic activity by tetrachloroethylene (EPA, 1987).

Carcinogenicity

The NCI (1977) found tetrachloroethylene that contained stabilizers to be a liver carcinogen in B6C3F mice administered 386 to 1,072 mg/kg tetrachloroethylene by gavage. The NTP (1986) exposed rats to 0, 200, 400 ppm and mice to 0, 100 and 200 ppm tetrachloroethylene. Male rats exhibited increased incidence in mononuclear cell leukemia, and an increased incidence of renal tubular adenoma/carcinoma (combined). Tetrachloroethylene induced hepatocellular carcinomas in male and female mice at both doses.

REGULATIONS AND STANDARDS

Tetrachloroethylene is considered a class B2 carcinogen by the EPA (1980). The carcinogenic potency factors for oral and inhalation exposures are 5.1×10^{-2} $(mg/kg/day)^{-1}$ and 5.2×10^{-7} $(ug/m^3)^{-1}$ or 0.0018 mg/kg/d, respectively (EPA, 1991). An oral RfD of 1×10^{-2} mg/kg/day has been established based upon a NOAEL of 14 mg/kg/day for liver effects in mice exposed via drinking water for 90 days (Hayes, et al., 1986). OSHA has developed a PEL of 100 ppm TWA, and a 200 ppm Ceiling. NIOSH recommends exposure to tetrachloroethylene be limited to the lowest feasible limit. ACGIH recommend a TLV-TWA of 25 ppm and a TLV-STEL of 100 ppm. The IDLH level is 150 ppm. An AWQC of 0.80 ug/l is associated with a cancer risk level of 10^{-6}. This assumes ingestion of 2 L water/day and 6.5 g fish/day. An MCLG of 0 has been proposed. Tetrachloroethylene is regulated under CERCLA with a RQ of 1 pound (ATSDR, 1987).

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Toxicology Brief

1,1,1-TRICHLOROETHANE

Synonyms:	Methyl Chloroform; Chlorothene; Aerothene TT; 1,1,1-TCA; Strobane; Inhibisol
CAS No:	71-55-6
Boiling point:	74°C
Color:	colorless
Conversion Factor:	1 ppm = 5.4 mg/m^3
Flammability:	
Flash Point (closed cup):	none (^0C)
Autoignition;	537 ^0C
LEL	8.0 % by vol.
UEL	10.5 % by vol.
Henry's Law Constant:	6.3 x 10^{-3} atm m^3/mol @ 20^0C
	17.2 x 10^{-3} atm m^3/mol @ 25°C
Log K_{oc}:	2.03
Log K_{ow}:	2.5
Melting Point:	-35/-36.7°C
Molecular Formula:	$C_2H_3Cl_3$
Molecular Weight:	133
Odor:	Ethereal; chloroform-like
Threshold:	120 ppm (air)
Specific gravity:	1.3249
Vapor Density:	4.63 (air = 1)
Vapor Pressure:	123 mm Hg @ 20°C
Viscosity:	0.903 cSt @ 20°C
Water Solubility:	1500 mg/l

(reference: ATSDR, 1990)

GENERAL DATA

1,1,1-trichloroethane (1,1,1-TCA) is a colorless solvent with a chloroform-like odor that is used extensively for industrial metal cleaning and in the manufacturing of adhesives. Other industrial applications include its use as a dry-cleaning agent, a vapor degreasing agent and a propellant. It is found in various consumer products in aerosol formulations. All of 1,1,1-TCA is eventually transmitted into the environment, primarily in the form of atmospheric emissions.

FATE AND TRANSPORT

In the **atmosphere** 1,1,1-TCA undergoes a slow photochemical decomposition to produce carbon monoxide, hydrogen chloride, phosgene, and various other halogenated products. In **water** the compound is hydrolyzed to acetic and hydrochloric acids (Dilling et al., 1975). The estimated half life of 1,1,1-TCA in water is greater than 6 months. When released to surface waters, 1,1,1-TCA migrates to

the atmosphere in a few days or weeks and has an estimated half life of 1 to 8 days in air (EPA, 1987). Evaporation is expected to be the predominant mechanism whereby 1,1,1-TCA is lost from the soil surface but in subsurface soil biodegradation is slow. Therefore, 1,1,1-TCA may remain significantly undegraded and leach into the groundwater (Tabak et al., 1981). Anaerobic biodegradation of TCA appears to be negligible (ATSDR, 1990). 1,1,1-TCA does not bioaccumulate to a significant extent in individual animals or in food chains. In the bluegill sunfish, the bioconcentration factor has been estimated to be 9 (EPA, 1987).

TOXICITY DATA

Human Toxicological Profile

Acute effects due to exposure to 1,1,1-TCA include mild eye irritation (1000-1100 ppm for 15 minutes), throat irritation (1900-2000 ppm for 15 minutes), and, in some subjects, light headedness and inability to stand at 2650 ppm for 15 minutes (Stewart et al., 1969). Exposure to 450 ppm of 1,1,1-TCA was observed to produce decreased perceptive capabilities in subjects upon inhalation for two periods of 4 hours with a 1.5 hour interval (Salvini et. al., 1971). Higher inhalation exposures to 1,1,1-TCA may result in death, apparently from a reversible depression of the CNS (EPA, 1984). 1,1,1-TCA affects the liver before it damages the kidney but respiratory tract irritation effects in humans appear to be only transitory (Weitbrecht, 1965). The odor threshold of 1,1,1-TCA ranges from 16-1700 ppm suggesting that individual sensitivity may play a part in determining irritation and sensitivity of various organs.

Pharmacokinetics

Stewart et. al. (1969) states that 1,1,1-TCA is absorbed rapidly and completely from the gastrointestinal tract although inhalation is the most common route of exposure. Retention of 1,1,1-TCA is reported to be 30 percent of the inspired air concentration at equilibrium after 4 hours of exposure to 70 or 140 ppm of 1,1,1-TCA (Monster et al., 1979 and Humbert and Fernandez, 1977). Unchanged 1,1,1-TCA is primarily excreted through the lungs; some is stored in the body fat and metabolites of trichloroacetic acid and trichloroethanol are excreted in the urine (EPA, 1987).

Significant Animal Studies

Like other halogenated hydrocarbons, 1,1,1-TCA influences the functions of the CNS, heart, lungs, liver and kidneys. The minimum fatal concentration is reportedly 65 mg/L in air. At a concentration of 45 mg/l, 1,1,1-TCA can produce complete narcosis (Lazarev, 1929). Krantz et al. (1959) estimated the dosage in dogs to be 450 mg/kg for induction of anesthesia and 800 mg/kg for respiratory failure. Following cessation of exposure, there is usually a rapid recovery of reflexes within 3 to 5 minutes. Impairment in motor control after exposure has been demonstrated in humans with concentrations as low as 250 ppm, but CNS effects appear to be dependent on concentration as well as exposure time. Inhalation of high levels of 1,1,1-TCA have been shown to produce decreased heart rate and blood pressure during the first few minutes of exposure (Herd et al., 1974). Cellular hypertrophy, a sign of cardiovascular toxicity, has also been reported.

Studies on the hepatic effects of 1,1,1-TCA have shown conflicting results due to variation in the animal species used, the dose, and the treatment schedule.

Genotoxicity

According to the EPA (1984), 1,1,1-TCA is weakly mutagenic to Salmonella TA1535 and TA100 although this may be due to the chemical stabilizers or manufacturing contaminants. In addition, EPA (1984) states that commercial samples of 1,1,1-TCA are genotoxic to mouse hepatocytes. Firm conclusions about the ability of 1,1,1-TCA to cause mutagenic effects are not possible due to the inadequacy of available data (EPA, 1984).

Carcinogenicity

The National Cancer Institute (NCI) conducted carcinogenesis bioassays of 1,1,1-TCA but due to the low survival rate and early lethality, the carcinogenicity assessment was precluded (NCI, 1977). A bioassay by the National Toxicology Program (NTP) in 1983 showed an increase in hepatocellular carcinoma occurrence in low and high dose males and high dose females, however, this study is being reviewed by the NTP for possible serious data discrepancies (EPA, 1984). IARC has not evaluated the risk to humans from exposure and the carcinogen assessment group of the EPA categorizes it as Group "D", which means it is not classified.

REGULATIONS AND STANDARDS

The American Conference of Governmental Industrial Hygienists (ACGIH, 1996) has recommended a threshold limit value of 350 ppm (1910 mg/m^3) and a STEL of 450 ppm (2460 mg/m^3). NIOSH has listed an IDLH of 700 ppm.

The EPA (1991) established RfD is 0.09 mg/kg/d and is based on the studies of Torkelson et al., (1958). The RfC is calculated as 3 mg/kg/d (EPA, 1991). These values are considerable lower than the values previously established as Acceptable Daily intakes (EPA, 1984). The Lowest Observable Adverse Effect Level (LOAEL) of slight growth retardation was 120 mg/kg/d. The uncertainty factor of 1000 is comprised of three factors of 10; for the use of a subchronic assay, for extrapolation from animals to man, and for the protection of sensitive human populations (IRIS, 1991). The MCL and MCLG are both 0.2 mg/l for 1,1,1-TCA (EPA, 1987).

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Toxicology Brief

TRICHLOROETHYLENE

Synonyms:	1,1,2-Trichloroethene, 1,1,2-Trichloroethylene, TCE, Ethinyl Trichloride, Ethylene Trichloride
CAS Number:	79-01-6
Boiling Point:	86.7 °C
Color:	clear; colorless
Conversion Factor:	1 ppm = 5.46 mg/m3
Flammability:	
Autoignition:	none
Flash Point:	none
LEL:	8.0 % by vol.
UEL:	10.5 % by vol.
Henry's Law Const:	2.0 x 10-2 atm m^3/mol.
Log Koc:	2.42
Log Kow:	2.29
Molecular formula:	C2HCl3
Molecular weight:	131.40
Odor:	ethereal; Chloroform-like; sweet
Specific Gravity:	1.465 (20/40°C)
Vapor density:	4.53 (air = 1)
Vapor pressure:	19.9 mm Hg at 0 °C 57.8 mm Hg at 20 °C
Viscosity:	0.566 cSt @ 20 °C
Water Solubility:	1.070 g/ l water@ 20 °C 1.366 g/ l water @ 25 °C

(references: ATSDR, 1989; Merck, 1983)

GENERAL DATA

Trichloroethylene (TCE) is a nonflammable, mobile liquid with a characteristic odor resembling that of chloroform. It is practically insoluble in water and reacts with alkali to form spontaneously flammable gas. When heated to decomposition, TCE emits toxic fumes of Cl_2-. Trichlorethylene has been widely used as a degreasing agent and as an intermediate in chemical synthesis. It is generally used as a solvent in such products as paints, typing correction fluids, and paint removers. In gas purification, it is used as a solvent of sulfur and phosphorous. It is used as a heat exchange fluid and refrigerant but such industrial uses are being phased out due to the carcinogenicity of the compound (HSDB, 1991).

FATE AND TRANSPORT

Because of its volatility, most of the TCE used in the United States is released into the atmosphere. The dominant TCE removal from air is its reaction with hydroxyl radicals with an estimated half life of approximately 6.8 days (Callahan et al., 1979; EPA, 1985; HSDB, 1991). The breakdown products include phosgene, dichloroacetyl chloride, and formyl chloride. TCE

spilled onto **soil** will also be dispersed to the atmosphere by volatilization or leach downward to the groundwater. In ground water, slow biodegradation of TCE does occur under anaerobic conditions (Barrio-Lage et al., 1987). TCE released into surface **water** will volatilize rapidly. The estimated volatilization half lives from typical bodies of water are: 11 days for a pond, 4 to 12 days for a lake, 1 to 12 days for a river (EPA, 1985).

TOXICITY DATA

Human Toxicology

Contact with liquid, relatively undiluted trichlorethylene can cause intense itching, subcorneal pustular eruptions, erythema and generalized dermatitis (Conde-Salazar et al., 1983). This syndrome is characterized by red blotches on the face neck, shoulders and backs. TCE is an irritant; volunteers exposed to 27, 31 or 201 ppm TCE for four hours suffered from irritation to the eyes and mucous membranes of the throat (Nomiyama and Nomiyama, 1977). A condition known as "Degreaser's Flush" has been described in workers who drink alçohol after exposure to TCE.

Because TCE was extensively used as both a general anesthetic and obstetrical analgesic, there are many reports of cardiac arrest, atrial and ventricular extrasystole, tachycardia and ventricular fibrillation (Defalque, 1961; EPA, 1985). The inhalation or ingestion of TCE by humans has resulted in a number of cases of sudden death due presumably to cardiac arrest. Not enough is known in these cases about the exposure conditions to establish a dose-response relationship (EPA, 1985).

The potent central nervous system depressant properties of TCE led to its use as an anesthetic. However, its popularity declined with reports of severe and occasionally fatal neurotoxic effects (Defalque, 1961). Acute exposure to high concentrations of TCE have been reported to cause irreversible nerve damage. Occupational exposure to TCE has caused nausea, headache, loss of appetite, weakness, dizziness, ataxia and tremors. There are reports of trigeminal nerve impairment in chronically exposed workers (Barret et al, 1982). Information on the duration and level of exposure are not available.

Both acute and chronic exposure to TCE can affect liver function. An individual who ingested an unknown amount of TCE developed jaundice and subsequently died. The autopsy revealed severe centrilobular necrosis (Kleinfeld and Tabershaw, 1954). Another case report describes a worker who was repeatedly exposed to TCE from heated degreasing tanks and repeatedly became inebriated. The worker switched to a job in which he was exposed to 1,1,1-trichloroethane. The worker then began to suffer from fatigue, weight loss, anorexia, icterus, and abdominal swelling. He was diagnosed as suffering from hepatic cirrhosis attributed to exposure to TCE and 1,1,1-trichloroethane (Thiele et al., 1982). Chronic exposure to TCE in some cases has caused adverse renal effects (Baerg and Kimberg, 1970).

Significant Animal Studies

Stott et al. (1982) investigated the effects of orally administered TCE on rats and mice. Mice were given 2,400 mg/kg by gavage daily for 3 days, or 5 days a week. The livers of the animals from both of these groups had altered hepatocellular morphology, centrilobular hepatocellular swelling, and necrosis. There was a statistically significant increase in liver weight and hepatic

DNA synthesis, and a slight reduction in DNA content per gram of liver. Rats treated with 1100 mg/kg TCE by gavage 5 days/week for 3 weeks had a statistically significant increase in liver weight and in hepatic DNA synthesis. Rats which received 1,100 mg/kg for three days did not have any liver changes.

Severe blood dyscrasias and myelotoxic anemia was observed in rabbits that were exposed to 2790 ppm TCE for 4 h/day, 6days/week for 45 days (Mazza and Brancaccio, 1967). Nomiyama et al., (1986) reported dose related changes in hemoglobin, hematocrit, reticulocyte count and erythroblast count in rats exposed to 50, 200 or 800 ppm TCE for 12 weeks.

Reproductive Toxicity

Manson et al., 1984 investigated the effect of trichloroethylene on the reproductive performance of females rats. The rats were treated with 10, 100, or 1000 mg/kg of trichloroethylene daily for 6 weeks. The trichloroethylene treatment started 2 weeks before mating, throughout mating (1 week), and through day 21 of gestation. The litters from the high dose females had a statistically significantly increased incidence of fetal death. The pups were only examined for major malformations and none were found.

Genotoxicity

TCE is primarily negative when tested in the Ames assay either with or without activation (Baden et al., 1979). Testing in *S. cereviseiae* has given mixed results (Callen et al., 1980). Cell transformation studies in RLV/Fischer embryo cells were positive (Price et al.,1978). *In vivo* genotoxicity testing of also produced mixed results. TCE was negative in a male mouse dominant lethal assay, but was positive at high concentrations for sperm abnormalities in another mouse strain. TCE was inconclusive in a mouse micronucleus test and negative in a rat DNA damage test but reportedly tested positive for single strand breaks in DNA in kidney and liver in other rat and mouse assays. TCE was "suggestive" for a causal relation for sister chromatid exchange in study on human occupational exposures (reviewed by ATSDR, 1989).

Carcinogenicity

TCE has been found to be carcinogenic in several animal studies. In a National Cancer Institute (NCI, 1976) long term cancer bioassay, TCE was administered to B6C3F1 mice and Osborne-Mendel rats of both sexes by gavage. Industrial grade TCE was used which contained 0.09% epichlorohydrin as a stabilizer. A large increase in incidence of hepatocellular carcinoma was seen in both the high and low dose male and female mice but no increase in tumors were detected in rats. Weaknesses of this study include the high doses of TCE used which resulted in a high mortality early in the study and the question of the purity of the TCE. An analysis of the number of cancers in the mice that could be due to the level of epichlorohydrin present has revealed that this could not account for the increased incidence of hepatocellular carcinoma (CARB, 1990).

The National Toxicology Program (NTP, 1982) essentially repeated the National Cancer Institute study with TCE which was stabilized with diisopropylamine. The study used B6C3F1 mice dosed by gavage with 1000 mg/kg, 5 days a week for 103 weeks. The F334/N rats were dosed with 500 or 1000 mg/kg with the same schedule. The mice had significantly higher incidences of hepatocellular carcinoma relative to the controls thus confirming the earlier NCI (1976) study.

In addition, the high dose male rats experienced a highly statistically significant increased incidence of renal tubular-cell adenocarcinoma.

Other studies not as definitive as the NTP (1982) study have been conducted which have found statistically increased incidences of pulmonary tumors in mice which were not dose dependent (Maltoni et al., 1986).

Epidemiological studies conducted to determine a link between occupational exposure to TCE and cancer have been inconclusive (CARB, 1990). IARC (1987) has determined that limited evidence of carcinogenicity in animals exists and inadequate evidence exists for carcinogenicity in humans. IARC (1987) therefore declared that TCE cannot be classified as to its carcinogenicity to humans. The EPA (1985) concluded that evidence for the carcinogenicity of TCE in animals was sufficient, and that the epidemiological data were inconclusive. TCE was classified as Group B2, a probable human carcinogen.

REGULATIONS AND STANDARDS

The American Conference of Governmental Industrial Hygienists (ACGIH, 1996) recommends a TWA-TLV: 50 ppm (269 mg/m3) and a Short Term Exposure Limit (STEL) of 100 ppm (mg/m3). The OSHA PEL is 100 ppm with a 200 ppm Ceiling and 1000 ppm IDLH (NIOSH, 1994). NIOSH considers TCE an occupational carcinogen and recommends that the levels of exposure be kept as low as feasible (NIOSH, 1994). The National Academy of Sciences (NAS) suggested a 1-day no-adverse-response level (SNARL for drinking water of 105 mg/L and a 7 day SNARL of 15 mg/L (ATSDR, 1989).

EPA has classified TCE as a B2 carcinogen with a cancer slope factor of 1.7 E-2 (mg/kg/day)-1 for inhalation and a cancer slope factor of 1.1 E-2 (mg/kg/day)-1 for ingestion. The inhalation slope factor is based upon two mouse inhalation studies showing lung tumors. The oral slope factor is based upon two mouse gavage studies showing liver tumors (EPA, 1991).

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Toxicology Brief

1,1-DICHLOROETHYLENE

Synonyms:	Vinylidene Chloride; 1-1-DCE; Vinylidene Dichloride
CAS No:	75-35-4
Boiling Point:	31.5°C
Conversion Factor:	1 ppm = 4.0 mg/m^3
Flammability:	Nonflammable
Henry's Law Const:	0.034 atm-m^3/mole
log K_{OC}:	65 ml/g
log K_{OW}:	1.84
Molecular Formula:	$C_2H_2Cl_2$
Molecular Weight:	96.95
Melting Point:	-122.2°C
Vapor Pressure:	591 mm Hg @ 25°C
Viscosity:	0.358 mN sec/m^2
Water Solubility:	2250 mg/l @ 20°C

GENERAL DATA

1,1-Dichloroethylene (1,1-DCE) is a highly reactive, flammable, clear, colorless liquid that can produce complex peroxides in the absence of chemical inhibitors. 1,1-DCE and its peroxides are violently explosive and produce formaldehyde, phosgene, and hydrochloric acid as decomposition products. 1,1-DCE can form explosive peroxides upon exposure to air and reacts vigorously with oxidizing materials (Sax and Lewis, 1989).

1,1-DCE is an intermediate in the synthesis of methylchloroform and in the production of barrier coating plastics such as polyvinylidene chloride copolymers (PVDCs). Polymers with high 1,1-DCE content, such as Saran Wrap, are commonly used for food packaging. The interior coatings for storage tanks and steel pipes and structures are often made of 1,1-DCE polymers (EPA, 1980). Such uses increase the likelihood that the substance will be found in the environment. Fishbein (1976) points out, however, that there are no data that document the extent to which untreated monomers of 1,1-DCE might migrate into food or water that comes in contact with the above-described plastics and coatings of discarded materials.

FATE AND TRANSPORT

Little information is available on the environmental fate and transport of 1,1-DCE (EPA, 1987). It is known, however, that because of 1,1-DCE's high vapor pressure (591 mm Hg 25°C), volatilization is a major transport mechanism. 1,1-DCE is expected to volatilize rapidly when released in surface waters and degrades in a matter of hours when released into the atmosphere (EPA, 1987). In water, 1,1-DCE is chemically stable. The half-life is estimated to be eight weeks (Pearson and McConnell, 1975). 1,1-DCE is mobile in soils and is expected to migrate with the groundwater (EPA, 1987). 1,1-DCE is very soluble in water (2250 mg/L @ 77°F) and has a correspondingly low octanol water partition coefficient (log K_{OW}) of 1.84. 1,1-DCE would not be expected to accumulate in animals because it is not very lipid-soluble.

TOXICITY DATA

Human Toxicological Profile

Relatively little information is available on human toxicity to 1,1-DCE exposure. One epidemiological study (Ott et al., 1976) examined an occupationally exposed cohort of 138 workers (who had no

concomitant exposures to other chemical solvents) for adverse health effects associated with exposure to 1,1-DCE. No significant findings were reported, however this study employed an insufficient sample size. NIOSH (1979) reports that liver dysfunction, headaches, vision problems, weakness, fatigue, and neurological sensory disturbances are some of the reported effects of occupational exposure to 1,1-DCE.

Pharmacokinetics

Pharmacokinetics data for absorption and excretion of 1,1-DCE are scarce. McKenna et al. (1978), report that 1,1-DCE given by gavage is completely absorbed, since virtually all of a single dose is excreted within 72 hours. Rat and mouse studies have revealed that rapid organ distribution to the kidney, spleen, liver, brain, and heart occurs (Jaeger et al., 1977). Subcellular distribution of 1,1-DCE includes compartmentalization in the microsomes, mitochondria, and cytosol.

It appears that the intermediate epoxide metabolites of 1,1-DCE are more reactive and more toxic than the compound itself. Pre-treatment of mice and rats with various epoxides (e.g., 2,3-epoxypropan-1-ol, styrene oxide) has been shown to interfere with the full metabolism of the intermediate epoxides and to exacerbate the toxic effects of 1,1-DCE (Anderson et al., 1977). In another study (Short et al., 1977), the hepatotoxic and nephrotoxic effects due to 1,1-DCE were mitigated when mice and rats were pretreated with disulfiram, an aldehyde dehydrogenase inhibitor.

Animal Toxicology and Significant Studies

The acute lethal oral dose for rats is 200 mg/kg (Short et al., 1977), while the acute lethal inhaled dose for mice is 98 ppm with a 22-hour exposure period (Anderson et al., 1977). Acute toxic effects include central nervous system depression at high concentrations and liver and kidney damage at lower concentrations (Proctor et al., 1988).

Reproductive Toxicity

Teratogenic effects have been reported in rats and rabbits exposed to 1,1-DCE via inhalation (Murray et al., 1979). Dosages of 20, 80 and 160 ppm were administered. Skeletal abnormalities were observed at the two higher concentrations in rats while only the higher dose caused "several minor skeletal variations in rabbits." The authors observed no adverse effects on embryonal or fetal development in the inhalation study at 20 ppm in the rat and 80 ppm in the rabbit. Nitschke et al. (1983) reported that reproduction in rats was not affected over three generations after exposure to 1,1-DCE at concentrations of 0, 50, 100, or 200 ppm in their drinking water.

Mutagenicity

Greim et al. (1975) reported that 1,1-DCE was mutagenic to E. coli at a concentration of 2.5 M in the presence of microsomal activation. Short et al. (1977) and Anderson et al. (1977) reported that, using the dominant lethal assay, mutagenic effects were not observed in mammalian assay systems after exposure to 1,1-DCE at 55 ppm for 6 hr/day for 11 weeks to 10 to 50 ppm for 6 hr/day for 5 days, respectively. Reitz et al. (1980) reported that in rats and mice exposed to 10 or 50 ppm of inhaled 1,1-DCE for 6 hours, 1,1-DCE binds slightly with the DNA in the liver and kidneys but causes massive tissue damage. There is sufficient evidence to state that 1,1-DCE is mutagenic (IARC, 1979).

Carcinogenicity

Eighteen animal studies have been reported concerning the carcinogenicity of 1,1-DCE (eleven inhalation, five oral, one dermal, and one subcutaneous exposure). These studies were not designed for maximum sensitivity to detect carcinogenic effects although 3 out of the five oral studies, were for lifetime exposures. Maltoni et al. (1985) was the only reported positive carcinogenic response (IRIS, 1991).

Maltoni et al. (1985) exposed both sexes of Swiss mice by inhalation to 10 and 25 ppm (maximum tolerated dose) for 4-5 days per week for 12 months and found a significant increase in kidney adenocarcinoma. Treatment was for only 50% of lifetime (12 months) and only two dose groups provided data suitable for modeling. Tumors were not observed in either the two control groups or the one 10 ppm treatment group, although both 25 ppm treatment groups developed tumors. According to IRIS (1991), within each dose pair (control and 25 ppm groups), there were no statistically significant differences between tumor incidences. These groups were combined for purposes of modeling. The 10 ppm group does not appear to have been included in the modeling, and its exclusion would cause the slope to be higher and the potency estimate to be more conservative. The data quality supporting the finding that 1,1-DCE is a carcinogen is weak, which resulted in U.S. EPA's classification of 1,1-DCE as a possible human carcinogen (Group C) as opposed to a probable human or known human carcinogen (Group B and A). From this study, an inhalation cancer slope factor of 1.2 $(mg/kg/day)^{-1}$ was derived.

The oral cancer slope factor of $6.0x10^{-1}$ $(mg/kg/day)^{-1}$ was derived from the highest of four slope factors derived in two studies. Neither the studies produced statistically significant increases in tumor incidences attributable to oral exposure to 1,1-DCE.

REGULATIONS AND STANDARDS

The American Conference of Governmental Industrial Hygienists (ACGIH, 1996) recommends a TWA-TLV: 5 ppm (20 mg/m^3) and a Short Term Exposure Limit (STEL) of 20 ppm (79 mg/m^3). NIOSH recognizes 1,1 DCE as an occpational crcinogen and recommends tht exposuresbe kept as low as feasible (NIOSH, 1994).

The noncarcinogenic risk estimates for 1,1-DCE have been summarized from the U.S. EPA Integrated Risk Information System (IRIS, 1991) chemical database. The oral reference dose (RfD) is based on the lowest observable adverse effect level (LOAEL) derived from a chronic oral rat bioassay (drinking water exposure) by Quast et al. (1983). An uncertainty factor of 1000 was employed to derive an oral RfD of 9.0×10^{-3} mg/kg/day. The endpoint effect in question was liver damage. CARB reports an oral RfD of 9.0E-3 x 10-1 mg/kg/day; from this, CAPCOA derived an inhalation RfD (RfC) of 3.2E+1 mg/m^3 (CAPCOA, 1991).

IRIS (1998) reports an oral cancer slope factor (CSF) of 6.0×10^{-1} $(mg/kg/day)^{-1}$ based upon gavage studies and an inhalation CSF of 1.2 $(mg/kg/day)^{-1}$ based upon an inhalation study. The U.S. EPA classified 1,1-DCE as a Group C carcinogen (possible human carcinogen) based upon the weak evidence supporting a causal relationship between 1,1-DCE and its ability to cause cancer in laboratory animals following lifetime exposure. Based upon this weak causal relationship, Class C carcinogens are not typically included in risk assessment activities. For purposes of establishing under RCRA and CERCLA, 1,1-DCE is not viewed as a carcinogen. The current Maximum Contaminant Level for drinking water for 1,1-DCE is set by U.S. EPA at 7.0 mg/L (IRIS, 1991). CAPCOA, 1991, does not list a slope factor for 1,1-DCE by either inhalation or ingestion routes of exposure.

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Toxicology Brief

VINYL CHLORIDE

Synonyms:	Choloroethene, Chloroethylene, ethylene monochloride, monochloroethylene.
CAS Number:	75-01-4
Boiling Point:	-13.37 °C
Color:	Colorless liquid or gas
Conversion Factor:	1 ppm = 2.6mg/m^3
Henry's Law Constant:	1.2 atm.m^3/mol.@10°C
Flammability Limits:	
Autoignition:	472°C
Flashpoint:	NA
LEL:	4%
UEL:	22%
Koc:	57 ml/g
Log Kow:	1.38
Melting Point:	-153.8°C
Molecular formula:	C2H3Cl
Molecular weight:	62.50
Specific gravity:	0.9106 (20/4 °C) (air = 1)
Vapor density:	2.15 (air=1)
Vapor pressure:	2530 mm Hg @ 20°C
Viscosity:	0.01702 cP @ 20°C (gas); 0.280 cP @ -20°C (liquid)
Solubility water:	1100-2763 mg/l @ 25°C

NA = Not available.
(Sax & Lewis, 1987; Cowfer & Magistro, 1983; Amoore & Hautula, 1983; HSDB, 1991; Verschueren, 1983)

GENERAL DATA

Vinyl chloride is a man-made volatile, colorless liquid or gas having a mild, sweet odor. It is considered to be a human carcinogen and hepatotoxin. Acute inhalation or ingestion of vinyl chloride may cause central nervous system effects and possibly death (Sax & Lewis, 1987).

Vinyl chloride is flammable and will react when exposed to heat, flames or oxidizers. Direct contact with nitrogen oxides may lead to explosions. Decomposition of vinyl chloride will release toxic chlorine gas (Sax & Lewis, 1987; HSDB, 1991).

The major use of vinyl chloride monomer is in making the vinyl chloride polymer which is used to make a variety of products, including food covering, pipe, wire coating, furniture, housewares, wallcovering, packaging materials, auto upholstery, and auto parts. Vinyl chloride is also used in very small amounts as a refrigerant gas and as a chemical intermediate. It was formerly used in small amounts as an aerosol propellant and as an ingredient in drugs and cosmetics (EPA, 1985a).

FATE AND TRANSPORT

Vinyl chloride exists as a gas at room temperature, therefore, nearly all vinyl chloride released into the environment reaches the atmosphere. Volatization is the chief transport process from soil and water. Once in the atmosphere photodegradation is the primary means of removing vinyl chloride from the air. The estimated the atmospheric half-life is 1.5-1.8 days. In areas of photochemical smog the half-life is substantially shorter, approximately 3-7 hours. The estimated half-life of vinyl chloride in a pond, lake and river is less than two days. Soil half-life depends on depth (EPA, 1985a; HSDB, 1991). Vinyl chloride is highly mobile in the soil (Lyman et al., 1982).

TOXICITY DATA

Human Toxicology

Vinyl chloride odor can be detected at 300 to 5000 ppm, and levels up to 8000 ppm may tolerated for 5 minutes without development of signs or symptoms of toxicity (ATSDR, 1991). Inhalation of vinyl chloride may cause respiratory irritation, bronchitis, dizziness, incoordination, headache, irritability, sensory polyneuritis, cardiac arrythmia, unconsciousness and death. Pulmonary effects include dyspnea, asthma, interstitial pneumonitis and pneumonoconiosis. Vinyl chloride is a severe irritant to the skin, eyes and mucous membranes. Occupational exposure to vinyl chloride vapor has resulted in deaths due to narcosis and development of a conditions known as "vinyl chloride disease" and "meat packer's asthma" (ATSDR, 1990). Symptoms of vinyl chloride disease include acroosteolysis, impaired peripheral circulation in the extremities, Raynaud's Syndrome, scleroderma, and hematologic, respiratory and hepatic effects (Mastromatteo et al., 1960; Lester et al., 1963; ATSDR, 1990).

Non-carcinogenic hepatotoxic effects may be reversible in occupationally exposed individuals (Ho et al., 1991). Scleroderma on the back of the hand at the metacarpal and phalangeal joints and inside of the forearms has been observed in some chronically exposed individuals having angioneurotic disorders (HSDB, 1991).

Metabolism

Absorption and distribution following inhalation or oral exposure is rapid. The liver is the primary site of distribution. Vinyl chloride is also distributed to the lungs, fat, muscle, skin and plasma (Watanabe et al., 1976; Withey, 1976). In the liver, vinyl chloride is metabolized in three alternative pathways. The most significant pathway is via the mixed function oxidase system into a reactive intermediate epoxide. The epoxide then undergoes further metabolism to chloroacetalaldehyde. Both the epoxide and aldehyde may bind to nucleic acids and proteins, impairing cell function (Hefner et al., 1975; Bolt et al., 1986). Vinyl chloride metabolites are excreted primarily in urine, while unchanged vinyl chloride is directly exhaled (Hefner et al., 1975)

Significant Animal Studies

The acute oral LD50 in rats is 500 mg/kg (Sax & Lewis, 1989). The acute LC50 levels for rodents range from 117 ppm in mice to 800 ppm in rabbits. The lowest reported lethal concentration in rats is 6000 ppm (EPA, 1985a). Anesthesia was observed in dogs at 70,000 ppm (Oster et al., 1947). Pulmonary edema, hemorrhaging, impaired blood clotting, and liver and kidney congestion also were observed in laboratory animals following acute exposure to vinyl chloride (Mastromatteo et al., 1960).

The liver is the primary target organ for vinyl chloride in animals and humans (Lee et al., 1977; Torkelson et al., 1961). Toxic effects in animals subject to chronic exposure to vinyl chloride include enlargement of the liver and spleen, reduced blood clotting time and development of angiosarcomas, adenosarcomas and heptacellular carcinomas (Feron and Kroes, 1979; Maltoni et al, 1980, 1981). The NOAEL and LOAEL in rats, based upon liver effects during a 15 week gavage study, was determined to be 30 mg/kg and 100 mg/kg. A lifetime feeding study in rats found the NOAEL and LOAEL to be 0.13 mg/kg/d and 1.3 mg/kg/d, respectively, based upon hepatotoxicity (Feron et al., 1975; Til et al., 1983). A one year rat inhalation study found the LOAEL and NOAEL to be 100 ppm and 10 ppm, respectively, based upon terminal body weights (Bi et al., 1985). The minimal risk level for "intermediate exposures" to vinyl chloride is estimated to be 50 ppm based on this data (ATSDR, 1988).

Proliferation of terminal bronchiolar cells, hyperplasia of alveolar epithelium, degeneration of alveolar septal cells and peribronchiolar or bronchiolar inflammation were observed in mice exposed to 2500 ppm or 6000 ppm during a six month period (Suzuki, 1978, 1980, 1983). Rats exposed to 5000 ppm for one year were observed to have hematologic effects, reduced growth, increased splenic hematopoiesis, degeneration of the myocardium, thickening of the arterial walls, hyperplasia of the olfactory epithelium, nephrotoxicity and mild alteration of the lungs and zymbal glands (Feron et al., 1979a, 1979b; Feron and Kroes, 1979).

Inhalation and ingestion of vinyl chloride has produced cancer in mice, rats and hamsters exposed to $\geq$ 50 ppm (Maltoni et al., 1980, 1981). Animal teratogenicity data is equivocal. No adverse maternal or teratogenic effects were observed in rats exposed to over 500 ppm for 7 to 12 day intervals during organogenesis (John et al., 1977; Ungvary et al., 1978). However, evidence of fetotoxicity and teratogenicity in rats subject to continuous exposure of 2.4 ppm through gestation has been observed (Mirkova et al., 1978).

Genotoxicity

Vinyl chloride has tested positive in in vitro and in vivo mutagenicity assays. The mutagenicity data implicates 2-chloroethylene oxide and 2-chloroacetalaldehyde as being the mutagenic agents associated with the positive results. Both metabolites have been shown to be more effective than vinyl chloride in inducing mutations in S. typhimurium (Bartsch et al., 1976). Vinyl chloride was positive in the recessive lethal assay, but negative in the dominant lethal assay in D. melanogaster (Verburght and Vogel, 1977). Vinyl chloride was negative in the mouse dominant lethal assay (Anderson et al., 1976). Vinyl chloride has been found to alkylate DNA in rats and mice (Green and Hathaway, 1978; Oster-Golker et al., 1977). Chromosomal aberrations in human peripheral lymphocytes of exposed workers have been detected in many studies and breaks have been reported to be localized in specific chromosomes (Sinues et al., 1991, Fucic et al., 1990).

Carcinogenicity

Vinyl chloride has been associated with cancer in humans and has been classified as an IARC Group 1 carcinogen and Group A carcinogen by the EPA (IARC, 1979; EPA, 1985a; 1987). Vinyl chloride was first associated with liver cancer in exposed workers in 1974 when rare liver angiosarcomas were detected in three workers in a single vinyl chloride polymerization plant (Creech and Johnson, 1974). A recent Scandinavian study detected a nearly three-fold increase in liver cancer among vinyl chloride workers which clearly correlated to time of initial exposure, duration of employment and estimated quantitative exposure (Simonato et al., 1991). In 1974 occupational exposure levels to vinyl chloride were reduced to 1 ppm and no new cases of angiosarcoma attributable soley to post-1974 exposure have

been reported (ATSDR, 1990).

Vinyl chloride has been implicated in other forms of cancer. Increased incidence of brain and CNS, lung and respiratory tract, digestive tract and hematopoietic/lymphopoietic cancers, and malignant skin melanomas have been associated with occupational vinyl chloride exposure (Fox and Collier, 1977; Heldass et al., 1984; Geryk and Zurdova, 1986).

REGULATIONS AND STANDARDS

The EPA (EPA, 1987) unit risk factor for vinyl chloride is 6.6×10^{-5} per part per billion. Vinyl chloride has been classified as a hazardous air pollutant by the EPA pursuant to section 112 of the Clean Air Act (Title 40 Code of Federal Regulations (CFR) section 401.15; 40 CFR 61.01; 7/1/88). Vinyl chloride concentration in exhaust from vinyl chloride manufacturing or purification plants is not to exceed 10 ppm (40 CFR 61.65 (a); 7/1/89). Vinyl chloride has been designated a toxic water pollutant by the EPA under section 307(a)(1) of the Clean Water Act (40 CFR 401.15; 7/1/88). Under the Safe Drinking Water Act the EPA has established the Maximum Concentration Limit (MCL) at 2 ppb (40 CFR 141.61; 7/1/88). The EPA has established a one day health advisory for vinyl chloride ingestion by a 10 kg child ingesting 1 liter of water per day to be 3 mg/day (EPA, 1991). A concentration of 0.15 ppb will give rise to a 10^{-6} risk for a 70 kg adult ingesting 2l/day for a lifetime (EPA, 1991). The California Department of Health Services Maximum Concentration Level is 0.5 ppb (EPA, 1991).

The ACGIH TLV is 5 ppm with non recommended STEL (ACVGIH, 1994). The OSHA TWA is 1 ppm and 15 PEL is 5 ppm (NIOSH, 1994;ATSDR, 1990). The use of vinyl chloride in all pesticide products, as inert or active ingredient, has been cancelled or suspended (HSDB, 1991). The FDA has also banned the use of vinyl chloride as an aerosol propellant and eliminated its use in drug products (NTP, 1985). The FDA has also alerted food manufacturers of the need to monitor packaging material containing vinyl chloride and has proposed limiting the vinyl chloride monomer content in packaging to 5-50 ppm (ATSDR, 1990).

The reportable quantity (RQ) for vinyl chloride release is 1 lb. (0.454 kg) or more (54 FR 33419; 8/14/89). Vinyl chloride is subject to management as a hazardous waste under RCRA regulations and corresponding state regulations once it becomes a waste (40 CFR 261.33). Any residue, or contaminated soil, water or debris containing the released vinyl chloride is also considered hazardous waste (40 CFR 261.3 (b)).

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