

**HUMAN HEALTH RISK ASSESSMENT  
FOR THE OLIVER RUBBER  
COMPANY PLANT 1  
1200 65<sup>th</sup> STREET OAKLAND, CA**

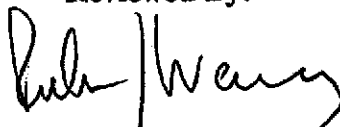
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ENVIRONMENTAL  
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## 1.0 INTRODUCTION

The following human health risk assessment (HHRA) was prepared for the redevelopment site of the Oliver Rubber Company property located at 1200 65<sup>th</sup> Street, Oakland, CA (the Site). The ChemRisk Group (ChemRisk) within McLaren/Hart, Inc. prepared this HHRA for the Site to determine the nature and extent of the potential health risks associated with on-site exposure to chemicals in subsurface soil and groundwater. The Oliver Rubber Company contracted ChemRisk to perform HHRA to facilitate closure of the site and subsequent redevelopment of the site for residential use.

### 1.1 PURPOSE AND OBJECTIVES

The purpose of this HHRA is to provide information regarding the potential for human exposure and potential incremental cancer risks and non-cancer health effects associated with direct and indirect contact with chemicals in subsurface soil and groundwater at the Site. At present, the Site is completely paved with six inches of concrete and/or asphalt. Consequently, the scope of this HHRA accounts for exposure to chemical vapors diffusing to the surface through soil and asphalt/concrete from contaminated shallow groundwater. In anticipation of proposed residential development of the property, this HHRA specifically evaluates inhalation of vapors emanating through cracks in the foundation of hypothetical future residences built on the Site. By accounting for this route of exposure, the scope of this risk assessment is more relevant than the California Environmental Protection Agency, Department of Toxic Substances Control (DTSC) Preliminary Endangerment Assessment (PEA) methodology that does not specifically evaluate groundwater vapor emissions (DTSC, 1994). The objectives of the risk assessment are to:

- Identify chemicals in subsurface soil and groundwater that, based on existing site conditions, may pose an adverse health risk to potential receptors under future land use scenarios;
- Identify direct and/or indirect exposure pathways by which individuals frequenting the Site under future land use scenarios might contact chemicals in subsurface soil and groundwater; and,
- Quantify potential incremental cancer risks and potential non-cancer health effects associated with upper-bound (high) and mid-range (typical) levels of exposure to chemicals based on RME and MLE scenarios.

Estimated health risks will be compared to values considered acceptable by regulatory agencies to determine whether existing conditions are protective of human health and the environment.

## 1.2 CONCEPTUAL APPROACH

This HHRA follows the general protocols described in *the Risk Assessment Work Plan: Oliver Rubber Company Plant 1, 1200 65<sup>th</sup> Street, Oakland, California* (ChemRisk, 1998) approved by representatives of the Alameda County Department of Environmental Health and the California State Water Quality Control Board. The technical approaches used in this document are consistent with risk assessment guidance and methodologies provided in the following documents:

- California Environmental Protection Agency (Cal-EPA) Department of Toxic Substances Control (DTSC), 1992. *Supplemental Guidance for Human Health Multimedia Risk Assessment of Hazardous Waste Sites and Permitted Facilities*, October 7, 1992.
- DTSC, 1994. *Preliminary Endangerment Assessment Guidance Manual*, January. Sacramento, California.
- United States Environmental Protection Agency (USEPA), 1989. *Human Health Evaluation Manual-Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual, Part A (RAGS)*, December. Washington, DC.
- USEPA, 1992a. *Supplemental Guidance to RAGS: Calculating the Concentration Term*, May. Washington, DC.
- USEPA, 1996a. *Exposure Factors Handbook*. August. Washington, DC.

## 1.3 REPORT ORGANIZATION

This report consists of seven sections, identified as Sections 1.0 through 7.0. Figures and tables cited within these sections are included after Section 7.0. The remainder of this report is organized as follows:

**Section 2.0 Data Analysis** - This section presents the statistical evaluation of the subsurface soil and groundwater data collected at the Site, and presents the methods used to select chemicals of interest (COIs) for purposes of evaluating potential human health risks under future land use scenarios.

**Section 3.0 Dose-Response Assessment** - This section presents the Cal-EPA and/or USEPA toxicity criteria selected to evaluate potential inhalation exposures to the identified COIs. Toxicity criteria include reference doses (RfDs) for evaluating potential adverse non-cancer health effects and cancer slope factors (SFs) for estimating incremental cancer risks.

**Section 4.0 Exposure Assessment** - This section describes the methods used to quantify exposure to the COIs. In particular, this section describes the exposure scenarios, potentially exposed populations, and potentially complete exposure pathways evaluated in this HHRA. Assumptions, models, parameters, and parameter values used to calculate exposure point concentrations and quantify potential exposures are presented.

**Section 5.0 Risk Characterization and Uncertainty Analysis** - In this section, the results of the dose-response assessment and exposure assessment are combined to characterize the potential adverse non-cancer health effects and incremental cancer risks for the populations of interest. This section also provides a qualitative description of the uncertainties associated with the exposure and risk estimates.

**Section 6.0 Conclusions** - This section presents the results of the HHRA and an analysis of risk management implications.

**Section 7.0 References** - This section provides full bibliographic citations for references used in the development of this HHRA and cited within the text.

## 2.0 HAZARD IDENTIFICATION

Hazard identification is the process of evaluating site conditions and environmental data to identify chemicals that, under certain conditions, could potentially have an adverse effect on human health. The purpose of this process is to focus attention on chemicals of interest (COIs) that may potentially present a health risk, and to eliminate those that would not contribute significantly to the overall risk estimate or that are not representative of site conditions (e.g., lab contamination).

### 2.1 SUMMARY OF SITE DATA

Subsurface soil and groundwater samples were collected at the Site from soil borings drilled September 2 and 3, and September 25, 1998 by Aqua Science Engineers (ASE), as described in *Reports of Additional Soil and Groundwater Assessment* (ASE, 1998a,b,c). Samples were measured for concentrations of volatile organic compounds (VOCs) and semi-volatile organic compounds (SVOCs).

During the September 2 and 3, 1998 Site assessment, four soil borings were drilled to a depth of 20 feet below ground surface (bgs) at the Site (ASE, 1998b). Three of these borings were located inside the building (borings BH-18, BH-19, and BH-21) and one was located near the former RAFFEX tank vault (boring BH-20). Soil samples were collected at depths ranging from 3.5 to 9.5 feet bgs. Groundwater grab samples were collected at the top of the groundwater table (ranging from 6 to 8 feet bgs) from each boring.

During the September 25, 1998 Site assessment, six additional soil borings were drilled at the Site (ASE, 1998c). Three of these borings were drilled along the upgradient edges of the property (borings BH-22, BH-23, and BH-24), two were drilled in central portions of the rear yard area (borings BH-25 and BH-26), and one was drilled adjacent to boring BH-20 to confirm previous results for that boring (boring BH-27). Soil samples were collected at a depth of 4 feet bgs, and groundwater grab samples were collected at the top of the water table (6 to 8 feet bgs).

Soil samples were analyzed by Chromalab for VOCs and SVOCs by EPA Methods 8240 and 8270, respectively. Groundwater samples were analyzed for VOCs using either EPA Method 8240 or 8260, and analyzed for SVOCs using EPA Method 8270. VOCs and SVOCs were detected in three of the soil samples at very low concentrations: ethylbenzene in sample BH-18 at 0.0065 mg/kg, chloroform in sample BH-25 at 0.0054 mg/kg, and phenol in sample BH-21 at 0.29 mg/kg (Table 1). VOCs and SVOCs were detected in five of the groundwater samples (BH-20, BH-24, BH-25, BH-26, and BH-27) (Table 2). Detected VOCs in groundwater included the following:

- 1,1-Dichloroethane in sample BH-20, BH-24, and BH-27 (range of detects 2.1 - 11 µg/L)

- 1,1-Dichloroethene in sample BH-20, BH-25, and BH-27 (range of detects 1.4 - 260  $\mu\text{g/L}$ )
- Toluene in sample BH-26 (0.56  $\mu\text{g/L}$ )
- 1,1,1-Trichloroethane in sample BH-20, BH-25, and BH-27 (range of detects 1.4 - 99  $\mu\text{g/L}$ )
- Trichloroethene in sample BH-26 (0.54  $\mu\text{g/L}$ )

In addition, butyl benzyl phthalate, an SVOC, was detected in sample BH-20 at a concentration of 9.4  $\mu\text{g/L}$ .

## 2.2 SELECTION OF CHEMICALS OF INTEREST

The detection of a chemical in the environment (e.g., soil, groundwater, and air) does not necessarily indicate that the chemical is potentially harmful to human health. The selection of chemicals identified during a site investigation for further evaluation has been termed "selection of chemicals of interest." The purpose of this process is to focus attention on those chemicals that are most likely to pose a potential health risk and to eliminate those chemicals that would not contribute significantly to the overall risk estimate or are not representative of site conditions (e.g., lab contaminants). Guidance provided in USEPA's *Risk Assessment Guidance for Superfund* (USEPA, 1989) and *Guidance for Data Useability in Risk Assessment* (USEPA, 1992b) was used to select COIs.

### 2.2.1 Selection of COIs for Soil

For purposes of identifying potential COIs for soils, a screening approach was used in which detected concentrations were compared to chemical-specific USEPA Region IX residential soil Preliminary Remediation Goals (PRGs) (USEPA, 1998a). PRGs are chemical concentrations in environmental media that correspond to fixed levels of risk (either a one-in-one-million cancer risk or a noncarcinogenic hazard quotient of one, whichever occurs at a lower concentration), assuming lifetime exposure (USEPA, 1998a). PRGs are derived using "standard" USEPA exposure parameters and dose equations, and assume lifetime exposure through common exposure pathways, such as ingestion, dermal absorption, or inhalation (USEPA, 1998a). For example, residential soil PRGs for carcinogenic chemicals assume that an individual is exposed to contaminated soil 350 days per year for 30 years (6 years as a child and 24 years as an adult). As a child, an individual is assumed to consume 200 milligrams [mg] of soil per day and as an adult, an individual is assumed to consume 100 mg/day of soil.

Per USEPA (1998), "exceeding a PRG suggests that further evaluation of the potential risks that may be posed by site contaminants is appropriate" although "chemical concentrations above these levels would not automatically designate a site as 'dirty' or trigger a response action." As presented in Table 3, all detected soil concentrations are below 10% of the chemical-specific residential soil PRGs. As a result, exposure to



chemicals in subsurface soils is not anticipated to contribute significantly to the overall risk estimate and these chemicals were not selected as COIs for this HHRA (i.e., exposure to this medium was not evaluated).

### 2.2.2 Selection of COIs for Groundwater

Chemicals detected in Site groundwater include toluene, 1,1-dichloroethane, 1,1-dichloroethene, 1,1,1-trichloroethane, trichloroethene, and butylbenzylphthalate (Table 2). Groundwater at the Site is not used as a drinking water source and there is not expected to be any potential for direct contact with groundwater. However, exposure to chemical vapors emanating from contaminated shallow groundwater is a potentially complete exposure pathway. As a result, volatile chemicals detected in Site groundwater were selected as COIs for this HHRA.

Based on this evaluation, the following chemicals were included as COIs for purposes of evaluating potential human health risks from inhalation of airborne chemical vapors from groundwater:

- 1,1-Dichloroethane
- 1,1-Dichloroethene
- Toluene
- 1,1,1-Trichloroethane
- Trichloroethene

Butyl benzyl phthalate was not selected as a COI for this HHRA because, as an SVOC with a Henry's Law constant less than  $10^{-5}$  atm-m<sup>3</sup>/mole and a molecular weight greater than 200 g/mole, it is not anticipated to diffuse significantly from the water column into indoor air (USEPA, 1998a).

### 3.0 DOSE-RESPONSE ASSESSMENT

The purpose of the dose-response assessment is to weigh the available evidence regarding the potential for a chemical to cause an adverse effect in the exposed population and to determine the quantitative relationship between the dose of that chemical and the incidence and severity of its assumed effect. Toxicity criteria will be used for evaluating potential noncarcinogenic and carcinogenic health effects from assumed exposures to COIs. Specifically, in this HHRA, USEPA reference doses (RfDs) were used to evaluate noncarcinogenic effects and Cal-EPA and/or USEPA cancer slope factors (SFs) were used to calculate potential incremental cancer risks.

The sources of the toxicity criteria and the hierarchy among the sources that were used in this HHRA are:

- California Office of Health Hazard Assessment (OEHHA), 1994. *Memorandum: California Cancer Potency Factors: Update*. November. (Used only for cancer slope factors);
- USEPA, 1998b. *Integrated Risk Information System (IRIS)*, on-line service available through the National Library of Medicine;
- USEPA, 1995. *Health Effects Assessment Summary Table (HEAST) FY-1995 Annual*. May; and,
- USEPA, 1998a. *USEPA Region IX Preliminary Remediation Goals (PRGs) 1998*. Available from USEPA, Region IX, San Francisco, CA.

The toxicity criteria used in this assessment to evaluate potential noncarcinogenic and carcinogenic health effects are described in the following sections.

### 3.1 NONCARCINOGENIC REFERENCE DOSES

For evaluating exposure to airborne chemicals via inhalation for potential noncarcinogenic effects, USEPA noncarcinogenic inhalation Reference Doses (RfDs) are used. RfDs (expressed in units of milligrams per kilogram of body weight per day, mg/kg-day) represent estimates of the level of daily human exposure to a chemical that is likely to be without appreciable risk of deleterious health effects to anyone (including sensitive subgroups) during either a portion of a lifetime (subchronic RfD) or a complete lifetime (chronic RfD) (USEPA, 1989).

USEPA RfDs are generally derived from threshold doses or concentrations (for chemicals in air) below which adverse health effects have not been observed, in either animal studies or studies of human populations. The first adverse effect that is observed in these studies as the dose or concentration increases beyond the threshold is

called the "critical effect" (Dourson *et al.*, 1996). Development of RfDs from threshold doses is based on the assumption that if the critical effect is prevented, than all toxic effects are prevented. Typically, the threshold dose or concentration is divided by safety factors to account for limitations in the quality or quantity of available data--the range of safety factors may vary between 100 to 10,000 (Dourson *et al.*, 1996). These safety factors establish an additional margin of safety between the dose or concentration associated with an adverse response in the animal studies or studies of workplace exposures, and the acceptable level of human exposure and ensure that the RfD is protective of human health even for the most sensitive individuals within a population (USEPA, 1989).

The USEPA RfDs used in this HHRA to evaluate potential noncarcinogenic health effects from inhalation exposure to the COIs in air are presented in Table 4.

### 3.2 CANCER SLOPE FACTORS

The USEPA assesses the carcinogenic potential of chemicals and assigns them to one of the following groups, according to the weight-of-evidence (WOE) from epidemiologic and animal studies (USEPA, 1986):

- Group A - Human Carcinogen (sufficient evidence of carcinogenicity in humans).
- Group B - Probably Human Carcinogen (B1 - limited evidence of carcinogenicity in humans; B2 - sufficient evidence of carcinogenicity in animals with inadequate or lack of human data).
- Group C - Possible Human Carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data).
- Group D - Not classifiable as to human carcinogenicity (inadequate or no evidence).
- Group E - Evidence of noncarcinogenicity for humans (no evidence of carcinogenicity in adequate studies).

In health risk assessment, potential incremental cancer risks are calculated for chemicals assigned to Groups A, B, or C. Health risks for exposures to carcinogens are defined in terms of probabilities. The probabilities identify the likelihood of a carcinogenic response in an individual who receives a given dose of a particular compound. These probabilities are based on the predicted rate of uptake and the chemical-specific cancer slope factor (SF). The SF is expressed in units of (mg/kg-day)<sup>-1</sup> and represents the 95% upper confidence limit (UCL) of the probability of a

carcinogenic response per unit daily intake of the chemical over a 70-year average human lifetime (USEPA, 1989). Use of the 95% UCL yields a conservative estimate of carcinogenic response, and it is considered unlikely that the actual risk posed by a chemical can be underestimated using this approach. The estimated daily intake of a chemical multiplied by the SF yields an estimate of the potential lifetime incremental cancer risk due to exposure to that chemical.

SFs are derived from mathematical models that extrapolate from the results of epidemiology and/or animal studies conducted at high levels of chemical exposure to the low exposure levels that may occur in humans (USEPA, 1989). The currently accepted regulatory policy specifies that carcinogenic chemicals be treated as if they do not have a threshold (USEPA, 1986). This approach assumes that the dose-response curve for carcinogens shows zero response only at zero dose (i.e., for all non-zero doses, some chronic carcinogenic response is assumed to be possible), with no differentiation between the various mechanisms of carcinogenesis, including those that may involve exposure thresholds. To estimate the theoretical response at low doses, various mathematical models are used. The accuracy of the projected risk depends on how well the model predicts the true relationship between dose and risk at dose levels where the relationship cannot be actually measured. This approach assumes that some level of risk is possible at all levels of exposure to chemicals that are known (Group A) or suspected carcinogens (Groups B and C).

While the USEPA's guidelines for carcinogen risk assessment are currently being revised (USEPA, 1995b), the above approach still represents regulatory policy. The no-threshold approach applies to all potential human carcinogens, even though it may overestimate the potential incremental cancer risks for some chemicals (Munro and Krewski, 1981; NAS, 1983).

Two of the COIs evaluated in this assessment, 1,1-dichloroethene and trichloroethene, have been identified as carcinogens by the USEPA (1998a). Of these COIs, Cal-EPA has established an inhalation SF for trichloroethene (OEHHA, 1994). In addition, Cal-EPA recognizes another COI evaluated in this assessment as a carcinogen, 1,1-dichloroethane (OEHHA, 1994). For purposes of this HHRA, the Cal-EPA inhalation SFs for trichloroethene and 1,1-dichloroethane were used to calculate potential incremental cancer risks from exposure to these chemicals. In the case of 1,1-dichloroethene, the USEPA inhalation SF was employed. The SFs used in this HHRA to evaluate potential carcinogenic health effects from inhalation exposure to these COIs in air are presented in Table 4.

## 4.0 EXPOSURE ASSESSMENT

Exposure assessment is the process of measuring or estimating the magnitude, frequency, and duration of potential human exposure to a chemical, the size and nature of potentially exposed populations, and the uncertainties inherent in these estimates. The principle elements of an exposure assessment are:

- Identification of potential receptor populations and exposure pathways;
- Determination of potential exposure point concentrations; and,
- Estimation of pathway and chemical-specific doses.

The exposure assessment for the Site was conducted consistent with USEPA and DTSC published guidance, as identified in Section 1.2.

The following sections present the components of the exposure assessment performed for the Site, including the assumptions and calculations used to quantitatively estimate potential doses to future on-site residents resulting from exposure to volatile organic COIs emanating from groundwater into building air.

### 4.1 IDENTIFICATION OF RECEPTOR POPULATIONS AND EXPOSURE PATHWAYS

The Site is currently being considered for redevelopment as residential property. Consequently, for purposes of this HHRA, the potential for COI exposures to adults and children residing in homes at the Site was included in the analysis.

The presence of a chemical in an environmental medium (e.g., groundwater) does not mean that exposure to the chemical will occur. In order for exposure to occur, a pathway to the substance must exist. In accordance with USEPA (1989) guidance, for an exposure pathway to be complete, it must have the following four elements: (1) a demonstrable source of release to the environment; (2) a transport medium (e.g., air); (3) a point of potential human contact with the medium; and, (4) a human exposure route at the contact point (e.g., dermal contact, ingestion, or inhalation). If a pathway is not complete, there is no potential for exposure

Groundwater at the Site is not a source of drinking water. At present, the Site is completely paved with six inches of concrete and/or asphalt. It is possible, but unlikely, that chemicals in groundwater will migrate through the soil and asphalt/concrete to the surface and volatilize to air. Exposure to chemical vapors emanating from contaminated shallow groundwater through cracks in the pavement is, theoretically, a potentially complete exposure pathway. Therefore, the only exposure pathways that have been identified as complete or potentially complete are:

#### 4.2.1 Calculation of Representative Groundwater Concentrations

The vapor flux at the soil surface resulting from VOCs in groundwater was modeled using Jury's Behavior Assessment Model (BAM; Jury *et al.*, 1983), described in detail in Section 4.2.2. To model chemical- and site-specific flux rates, the BAM requires representative chemical-specific COI concentrations in groundwater and site-specific information regarding the depth of the groundwater table.

Representative groundwater concentrations were determined in accordance with USEPA's *Supplemental Guidance to RAGs; Calculating the Concentration Term* (USEPA, 1992b). USEPA guidance suggests that the media concentration term used in the risk assessment be "an estimate of the arithmetic average concentration for a contaminant based on a set of site sampling results. Because of the uncertainty associated with estimating the true average concentration at a site, the 95% UCL of the arithmetic mean should be used for the variable" (USEPA, 1992b). The choice of the arithmetic mean concentration as an appropriate measure for estimating exposure derives from the need to estimate an individual's long-term average exposure. Therefore, the arithmetic mean is appropriate regardless of the pattern of daily exposures over time or the type of statistical distribution that might best describe the sampling data (USEPA, 1992b).

To calculate 95% UCLs, the groundwater data were statistically tested for normality and lognormality using the skewness test method of D'Agostino *et al.* (1990) at a 5% percent significance level ( $p=0.05$ ). All data sets were found to be neither normal nor lognormal. Following USEPA guidance (USEPA, 1992a), the data sets were assumed to be lognormally distributed and 95% UCLs were then calculated following the equations presented in Gilbert (1987) and USEPA's *Supplemental Guidance to RAGs; Calculating the Concentration Term* (USEPA, 1992b). The process involves the following steps:

1. Calculate the arithmetic mean and standard deviation of the log-transformed data (natural log of the data),
2. Determine the H-statistic, and
3. Calculate the 95% UCL using the following equation:

##### Equation 4-1. Calculation of 95% UCLs

Where:

$$UCL = e^{\left( x + 0.5 s^2 + \frac{s * H}{\sqrt{n-1}} \right)}$$

UCL            =    upper confidence limit  
e                =    constant (base of natural log, approximately 2.718)  
x                =    arithmetic mean of the log-transformed data

s	=	standard deviation of the log-transformed data
H	=	H-statistic (Gilbert, 1987)
n	=	number of samples

Per USEPA (1989), when the 95% UCL exceeds the maximum detected concentration, the maximum detected concentration should be used to characterize potential exposures. In this HHRA, the calculated 95% UCL concentrations for each of the groundwater COIs exceeded the maximum detected concentration. Consequently, the maximum detected concentrations were used to evaluate exposures for the RME scenario. The arithmetic mean groundwater concentration was used to evaluate the MLE scenario.

Table 5 presents representative groundwater concentrations for each of the COIs for the RME and MLE scenarios. As further discussed in Section 4.2.2, these concentrations are used to model vapor flux of VOCs at the soil surface, due to emissions from subsurface groundwater.

#### 4.2.2 Vapor Flux Modeling

Representative groundwater concentrations of COIs were used in the BAM to model the vapor flux of VOCs at the soil surface. The infinite source version of BAM is used by the USEPA (1996b) to develop inhalation Soil Screening Levels (SSLs) for VOCs in soil, and by the American Society for Testing Materials (ASTM) in Guide ES 38-94, *Emergency Standard Guide for Risk-Based Corrective Action Applied to Petroleum Release Sites* (ASTM, 1994). In addition, the finite source version is one of the refined methods for vapor emission recommended by the USEPA (1996b). Limited validation of the finite source version of BAM for VOCs in soil indicated good correlation between measured emission rates and model predictions under controlled conditions (USEPA, 1996b). Because of its use by the USEPA and the results of the limited validation study, the BAM was selected for use in estimating vapor emission rates for this HHRA. In this HHRA, a modified version of the BAM was used that includes emissions from groundwater.

The modified version of BAM that includes groundwater emissions assumes that the groundwater is uniformly contaminated at an initial groundwater concentration. In this HHRA, chemical-specific flux rates from groundwater used to characterize exposures for the RME scenario were calculated by setting the initial groundwater concentration equal to the maximum detected groundwater concentration. For the MLE scenario, flux rates were calculated using the arithmetic mean groundwater concentration. The initial chemical concentration in soil was set to zero since a major portion of the unsaturated zone soils were found to possess negligible concentrations of VOCs, as described in Section 2.0. Vapor emissions from groundwater were assumed to only occur at the surface of the water table.

BAM calculates a time-averaged vapor flux (in units of  $\text{mg}/\text{m}^2\text{-sec}$ ) at the soil surface based on the following assumptions (Jury *et al.*, 1983):

- A second-order partial differential equation that describes the fate and transport of a single organic species in a one-dimensional, homogeneous porous medium. Chemical transport is subject to gaseous diffusion, liquid diffusion, liquid advection, evapotranspiration, volatilization, degradation, liquid-solid sorption, and liquid-gaseous partitioning.
- Soil, chemical, and environmental properties are assumed to be uniform and constant throughout the soil column and water-bearing zone. These include the soil bulk density, moisture content, air content, porosity, liquid water flux (e.g., evaporation), partition coefficients, diffusion coefficients, organic carbon content, and temperature.
- The liquid-solid phase partitioning in the unsaturated zone is described by a linear, equilibrium sorption isotherm, similar to the one used to describe soil porewater concentrations in unsaturated zone soils.
- The liquid-vapor phase partitioning in the unsaturated zone is described by Henry's Law.
- There is a uniform initial concentration of chemical in the soil and groundwater at an initial time zero. In the present case, the soil was assumed to be initially uncontaminated between the surface and the top of the water table. The chemical concentration of the VOC in groundwater is uniform throughout the time-averaging period. However, with the passage of time, the soil in the vadose zone slowly becomes contaminated as the VOCs move vertically through the soil column. This occurs because the VOCs in the gaseous phase due to groundwater vapor emissions will partition back to the aqueous and sorbed phases as the emissions travel through the soil column.
- Chemical volatilization and water evaporation to the atmosphere are limited by gaseous diffusion through a stagnant air boundary layer above which the chemical has zero vapor concentration and the water vapor is at 50 percent relative humidity.
- Both the soil-gas diffusion coefficient and the soil-liquid diffusion coefficient include the Millington-Quirk model (Shearer *et al.*, 1973) of tortuosity. This tortuosity factor takes into account the reduced flow area and increased path length of diffusing gas or liquid molecules in partially saturated soil.



Besides the initial concentration in soil and groundwater, BAM utilizes three calculated parameters to describe vapor emissions through soil from groundwater. These included the effective solute advective velocity ( $V_E$ ), the effective diffusion coefficient ( $D_E$ ), and the effective stagnant air boundary coefficient ( $H_E$ ). The equations describing these variables are given by Jury *et al.* (1983). In order to determine the aforementioned parameters, site-specific soil properties as well chemical properties of the VOCs are needed. The site-specific soil properties, chemical properties, and environmental data that are required as input parameters to the model include soil dry bulk density, soil porosity, gravimetric and volumetric moisture contents, volumetric air content, fraction of organic carbon in soil, soil temperature, net water flux through the soil column, air-gas diffusion coefficient, water-liquid diffusion coefficient, the Henry's Law constant, and the organic carbon partitioning coefficient. For purposes of this HHRA, these values were based on default geophysical parameters from USEPA's *Soil Screening Guidance* (USEPA, 1996b) and chemical-specific physical-chemical properties.

Table 5 presents the chemical-specific vapor flux values for the RME and MLE scenarios.

#### 4.2.3 Calculation of Indoor Air Concentrations

A modified box model was used to estimate indoor air concentrations resulting from the chemical-specific flux rates estimated using BAM. For indoor air, the modified box model assumes that vapors enter a theoretical enclosed space or box above the area of interest via emissions through the cracks in the floor and that emissions are diluted by the continual flow of outside air through the box (i.e., ventilation). The box model used in this assessment, taken from the USEPA's *Air/Superfund National Technical Guidance Study Series Assessing Potential Indoor Air Impacts at Superfund Sites* (USEPA, 1992c), is as follows:

##### *Equation 4-2. Modified Box Model for Calculation of Indoor Air Concentrations*

$$C_{in} = \frac{E_i \times A \times F}{ACH / CF \times V}$$

Where:

- $C_{in}$  = Indoor air concentration (mg/m<sup>3</sup>)
- $E_i$  = Chemical-specific vapor flux rate from groundwater, at the soil surface (mg/m<sup>2</sup>-sec)
- $A$  = Emission area or area of the building (m<sup>2</sup>)
- $F$  = Fraction of floor area that is cracked (unitless)
- $ACH$  = Air exchanges per hour (hr<sup>-1</sup>)
- $CF$  = Conversion factor (sec/hr)

$V$  = Volume of air in building ( $m^3$ )

The input parameters, including their source and rationale, were obtained from site-specific information or literature sources where available. Of note, the "fraction of floor that is cracked" ( $F$ ) is highly dependent on the type of foundation (e.g., raised versus slab) and the age of the structure. Typical leakage ratios have been found to range between approximately 1 and 10  $cm^2$  per square meter of floor (Grimsrud et al., 1983). Site-specific information suggests the fraction of floor that is cracked at the Site is at the low end of this range, due to the integrity of the slab foundation; however, a conservative value of 10  $cm^2/m^2$  (i.e., 0.001) was used in the model. The emission area ( $A$ ) and volume of air in building ( $V$ ) parameters were based on information on the proposed dimensions for the on-site live/work studios. The input parameters used in this HHRA to calculate indoor air concentrations of volatile COIs due to emission of vapors from groundwater are presented in Table 6. Table 5 presents the chemical-specific indoor air concentrations calculated for the RME and MLE scenarios.

#### 4.3 ESTIMATION OF PATHWAY- AND CHEMICAL-SPECIFIC DOSES

The potential for occurrence of an adverse health effect associated with a specific exposure scenario depends on the degree of exposure and the degree of local or systemic uptake (amount absorbed into the blood and tissues). For any route of exposure, the dose ( $D$ ) is the product of exposure ( $E$ ) and the absorption efficiency ( $A$ ):

$$D = E \times A$$

Although a number of different factors are used to quantify exposure, this mathematical relationship holds true for all exposure routes, and is expressed as mass of chemical per mass of body weight per day (mg/kg-day).

Potential chemical-specific doses through inhalation of vapor in indoor air were estimated using the dose equation for this pathway provided by USEPA (1989), and were estimated in conjunction with receptor-specific parameter values. Values for exposure parameters, e.g., body weight, were taken from DTSC (1992), USEPA (1996a), or literature sources as appropriate. Different exposure assumptions were used to estimate doses for the RME and MLE scenarios. For example, for the RME scenario, the duration of exposure was assumed to be 30-years—this value is an upper bound estimate of the number of years that an individual remains at the same residence. For the MLE scenario, the exposure duration was assumed to be 15 years, based on the 50<sup>th</sup> percentile value for time at the same residence (USEPA, 1996a).

The following equation was used to estimate the chemical dose received by a receptor as a result of the inhalation of airborne (vapor phase) COIs:

*Equation 4-3. Inhalation of Airborne (Vapor Phase) Chemicals*

$$Dose_{IV} = \frac{C_i \times BR \times B_i \times ET \times EF \times ED}{BW \times AT}$$

Where:

<i>Dose<sub>IV</sub></i>	=	Dose received through inhalation of indoor air vapor (mg/kg-day);
<i>C<sub>i</sub></i>	=	COI concentration in indoor air (mg/m <sup>3</sup> );
<i>BR</i>	=	Breathing rate (m <sup>3</sup> /hr);
<i>B<sub>i</sub></i>	=	Bioavailability through inhalation (percent);
<i>ET</i>	=	Exposure time (indoors) (hrs/day);
<i>EF</i>	=	Exposure frequency (days/year);
<i>ED</i>	=	Exposure duration (years);
<i>BW</i>	=	Body weight (kg); and
<i>AT</i>	=	Averaging time (days).

Exposure parameters used to calculate doses received through inhalation of indoor air vapor for the RME and MLE scenarios are presented in Table 7.

Results of dose calculations for the RME and MLE scenarios are chemical-specific point estimates for average daily dose (*ADD*) calculated for noncarcinogenic chemicals, and lifetime average daily dose (*LADD*) calculated for carcinogenic chemicals. For the *ADD*, the exposure is averaged over the exposure duration (i.e., *AT* is assumed to be equal to *ED*). For the *LADD*, the exposure is averaged over the entire lifetime regardless of the length of exposure, based upon the toxicological nature of carcinogenic agents (i.e., *AT* is assumed to be 70 years; USEPA, 1989).

## 5.0 RISK CHARACTERIZATION

Risk characterization integrates the exposure and dose-response assessments to characterize the likelihood of adverse health effects in potentially exposed receptor populations. In particular, this section provides a qualitative and quantitative summary of the potential health risks posed to future on-site residents from exposure to vapors in indoor air, including both potential adverse non-cancer health effects and potential incremental cancer risks. Potential adverse non-cancer health effects are characterized using hazard quotients (HQs) and hazard indices (HIs). Cancer risks are evaluated separately as probabilities of an individual developing cancer during a 70-year average human lifetime.

The risk characterization methods used in this HHRA are consistent with those developed by USEPA (1989, 1998b) and DTSC (1992, 1994).

### 5.1 POTENTIAL ADVERSE NON-CANCER HEALTH EFFECTS

As discussed in Section 3.1, each of the COIs is evaluated for potential non-cancer effects. The potential for adverse non-cancer health effects is evaluated by comparing the estimated ADD to an appropriate USEPA reference criterion (e.g., RfD). If the estimated ADD is at or below the reference criterion, then the ADD is not expected to pose a significant non-cancer health hazard under the conditions evaluated (USEPA, 1989).

For each chemical and exposure scenario, a "Hazard Quotient" (HQ) is calculated. The HQ is defined as the ratio of the chemical-specific ADD calculated for a given scenario to the chemical-specific reference criterion:

*Equation 5-1. Calculation of Hazard Quotient (HQ)*

$$HQ = \frac{ADD_a}{RfD_a}$$

A HQ of less than one (<1) indicates that the estimated exposure is acceptable and is not expected to pose an adverse health hazard. The smaller the HQ, the greater the degree of protection.

Under USEPA policy where individual chemicals potentially act on the same target organs or result in the same health endpoint (e.g., respiratory irritants), the cumulative effect of exposures to multiple chemicals should be addressed (USEPA, 1989). For this evaluation, multiple chemical exposures were evaluated by summing the HQs for each chemical. HQs were then summed, regardless of the target organ affected, to calculate a Hazard Index (HI):

*Equation 5-2. Calculation of Hazard Index (HI)*

$$HI = \frac{ADD_a}{RfD_a} + \frac{ADD_b}{RfD_b} + \text{etc.}$$

An HI less than or equal to one indicates that exposure to multiple chemicals is unlikely to result in adverse non-cancer health effects to the populations of interest.

The HQs calculated for each COI and the total HI calculated for each receptor for the RME and MLE scenarios are presented in Table 8. Appendix A presents the detailed dose calculations for each COI. Figure 2 illustrates the HIs calculated for both adult and child residents under the RME and MLE scenarios.

As shown in Table 8, HI values under the RME scenario are less than one and range from 0.004 to 0.009. The MLE HI values are lower and range from 0.0004 to 0.0009. Based on this evaluation, there is no expectation that adverse health effects will occur as a result of potential exposure to COIs detected in groundwater at the Site.

## 5.2 POTENTIAL INCREMENTAL CANCER RISKS

Cancer risk is defined as the potential incremental probability of an individual developing cancer as a result of chemical exposure under a given set of conditions during a 70-year average human lifetime. The incremental probability of developing cancer is that risk attributed to potential exposure to the Site chemicals and is independent of exposure to chemicals not evaluated in this assessment (e.g., chemicals present in the environment that are not related to the Site). For example, National Cancer Statistics indicate that each person has a three-in ten chance, or 300,000 chances in a million, of developing cancer during his or her lifetime. Therefore, an individual with an incremental cancer risk of one in a million (denoted as either  $1 \times 10^{-6}$  or 1E-06) has a risk of 300,001 in a million of developing cancer. The incremental risks associated with exposures to COIs in indoor air were calculated using USEPA-recommended methods (USEPA, 1989).

Potential incremental cancer risk is a function of the LADD, which is defined as the total incremental dose of the chemical received as a result of exposure averaged over a lifetime, and the chemical-specific SF. For each COI, the potential incremental cancer risk was calculated as follows:

*Equation 5-3. Calculation of Potential Cancer Risks*

$$\text{Potential Incremental Cancer Risk} = \text{LADD} \times \text{SF}$$

Where:

$LADD$  = Lifetime Average Daily Dose (mg/kg-day); and,  
 $SF$  = Cancer Potency Slope Factor (mg/kg-day)<sup>-1</sup>.

As described in Section 3.2, three COIs (1,1-dichloroethane, 1,1-dichloroethene, and trichloroethene) were evaluated for carcinogenic risks in this HHRA. The potential incremental cancer risks calculated for each COI are presented in Table 9. Appendix A presents the detailed calculations performed to estimate the potential incremental cancer risk for each COI that was identified as a carcinogen.

As shown in Table 9, the cancer risk estimates for the adult and child residents under the RME scenario are below  $1 \times 10^{-5}$  with the highest estimated risk of  $2 \times 10^{-6}$  associated with the adult resident. The potential incremental cancer risk for the child resident under the RME scenario was  $1 \times 10^{-6}$ . The estimated cancer risks for the MLE incorporate more realistic exposure assumptions. The estimated cancer risks for the MLE scenario ranged from  $8 \times 10^{-8}$  to  $1 \times 10^{-7}$ .

There is a range of cancer risk criterion that are used by risk managers to evaluate whether estimated incremental cancer risks associated with site exposures may be of potential significance. For example, regulatory agencies have historically considered an increased lifetime cancer risk of one in a million ( $10^{-6}$ ) to be of negligible interest. The National Contingency Plan (40 CFR 300) defines an excess cancer risk range of  $10^{-6}$  to  $10^{-4}$  (one in a million to one in ten thousand), depending on site-specific considerations, as being the range of risk considered *de minimus*. A carcinogenic risk of less than  $10^{-5}$  (10 in a million) is considered insignificant and of no regulatory interest according to the State of California Safe Drinking Water and Toxic Enforcement Act of 1985 (Proposition 65) and the California AB2588 Air Toxic Hot Spots Program (CAPCOA, 1992).

Under all residential receptor conditions, all total cancer risk estimates are less than  $1 \times 10^{-5}$  and are within acceptable cancer risk levels.

### 5.3 UNCERTAINTIES

There are multiple sources of uncertainty that may be identified in any risk assessment. Although some exposure factors have a strong scientific basis, others have much less and therefore introduce some uncertainty into the results. The assumptions that introduce the greatest uncertainty, and the effects these uncertainties have on the estimates of risk, are qualitatively discussed below. The conservative assumptions used in this HHRA result in the estimation of health risks that are highly unlikely to be exceeded by actual conditions at the site.

Conservative Exposure Point Concentrations: For the RME scenario, the exposure point concentrations of COI vapors in indoor air were based on the maximum detected concentrations of each COI in groundwater. Because the

available groundwater data are limited, the maximum detected concentration was used for the RME scenario rather than the 95% UCL, as prescribed by USEPA guidance (USEPA, 1992a). Use of maximum concentrations provides a very conservative approximation of potential long-term average exposures, and likely overestimates actual site-wide conditions. Further, by assuming that there is no degradation of the substances or source depletion, the groundwater concentrations likely overestimate the actual long-term concentrations.

Incomplete Pathway: For this risk assessment, the potential health consequences associated with exposures to COIs in groundwater have been assumed to occur through an indirect exposure pathway involving diffusion of vapors through a six-inch concrete/asphalt slab into indoor air. It was assumed that vapors emanated through cracks in the floor surface, which were assumed to comprise 1% of the total surface. This is likely a highly conservative assumption, because of the integrity of the concrete/asphalt slab.

Multiple Conservative Exposure Assumptions: Exposure assumptions used in this analysis likely overestimate the health risk. In this assessment, the residential exposure duration period is assumed to be continuous for 15 years for the MLE scenario and 30 years for the RME scenario. The use of an assumed 30-year exposure duration is very conservative.

For the RME scenario, the frequency of exposure is assumed to be 24 hours per day for 350 days per year (DTSC, 1992). In practice, it is known that only a fraction of the day and year will be spent at home. A review of age-specific time-use studies performed at the University of Michigan and analyzed by the USEPA in the *Exposure Factors Handbook* indicates that the percentage of a day that an average adult is likely to spend at his or her residence (including sleeping time) is about 64% (USEPA, 1996a).

Animal to Human Extrapolation: For many chemicals, animal studies provide the only reliable information on which to predict the possible adverse health effects in humans. The procedures used to extrapolate from animals to humans include conservative assumptions so that the predicted adverse effects in humans will be overestimated not underestimated.

High to Low Dose Extrapolation: The concentrations of substances to which people are exposed in the environment are usually much lower (sometimes several orders of magnitude) than the doses to which animals are exposed in laboratory studies. Predicting effects at very low doses, therefore, requires the use of models that contain assumptions that introduce some degree of uncertainty.

Usually, the level of uncertainty in risk estimates at low levels of exposure is larger for carcinogens than noncarcinogens. Estimates of carcinogenic potency

are derived from experimental data in animals or from epidemiological studies in exposed workers or other populations. Typically, potency values will have a best and an upper bound estimate. There may be significant uncertainty leading to a large difference between these two values. For some substances, the upper bound may be 10 times higher than the best estimate. It is common practice by regulatory agencies to use the upper bound of this range in performing risk estimates so that the risks are far more likely overestimated than underestimated.

#### Evaluation of Possible Additive Effects

Potential noncarcinogenic health effects were conservatively assumed to be additive for all COIs without regard to the target organ affected. Predicting the total potential hazard by summing hazard quotients irrespective of the mechanism of action, pharmacokinetics, and target organ, tends to significantly overestimate the potential for occurrence of the health effect. Total cancer risk also is calculated by summing the predicted carcinogenic risk across all routes and media. This is a conservative approach since different chemicals generally have different mechanisms of action and different target organs relative to carcinogenesis. Further, the presence of certain carcinogens can also reduce the carcinogenic hazard posed by others.

#### 5.4 CONCLUSIONS

Based upon the HQs and risks presented in Tables 8 and 9, representative concentrations of COIs in groundwater at the Site do not appear to pose either adverse non-cancer health effects or cancer risks to potential future on-site residential receptors.



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**Table 1**  
**Chemical Concentrations in On-Site Subsurface Soils**  
**Oliver Rubber Company Plant 1**

Sample Id.	Depth (ft)	Ethylbenzene (ppm)	Chloroform (ppm)	Phenol (ppm)
BH-18	3.5	<b>0.0065</b>	< 0.005	< 0.1
BH-19	3.5	< 0.005	< 0.005	< 0.1
BH-20	7.5	< 0.005	< 0.005	< 0.1
BH-21	9.5	< 0.005	< 0.005	<b>0.29</b>
BH-25	4	< 0.005	<b>0.0054</b>	NA
BH-26	4	< 0.005	< 0.005	NA
BH-27	4	< 0.005	< 0.005	NA

*Notes:*

Detected concentrations in bold.

"<": Chemical not detected for this sample. Detection limit provided.

NA: Chemical not analyzed for this sample.

**Table 2**  
**Chemical Concentrations in On-Site Groundwater**  
**Oliver Rubber Company Plant 1**

Sample Id.	Toluene (ppb)	1,1-Dichloroethane (ppb)	1,1-Dichloroethene (ppb)	1,1,1-Trichloroethane (ppb)	Trichloroethene (ppb)	Butylbenzylphthalate (ppb)
BH-18 WATER	<2	<2	<2	<2	<2	<14
BH-19 WATER	<2	<2	<2	<2	<2	<12
BH-20 WATER	<2	<b>11</b>	<b>260</b>	<b>99</b>	<2	<b>9.4</b>
BH-21 WATER	<2	<2	<2	<2	<2	<5.8
BH-22 WATER	<0.5	<0.5	<0.5	<0.5	<0.5	NA
BH-23 WATER	<0.5	<0.5	<0.5	<0.5	<0.5	NA
BH-24 WATER	<0.5	<b>2.1</b>	<0.5	<0.5	<0.5	NA
BH-25 WATER	<0.5	<0.5	<b>1.4</b>	<b>1.4</b>	<0.5	NA
BH-26 WATER	<b>0.56</b>	<0.5	<0.5	<0.1	<b>0.54</b>	NA
BH-27 WATER	<0.5	<b>8.4</b>	<b>120</b>	<b>52</b>	<0.5	NA

*Notes:*

Detected concentrations in bold.

"<": Chemical not detected for this sample. Detection limit provided.

NA: Chemical not analyzed for this sample.

**Table 3**  
**On-Site Subsurface Soil Concentrations**  
**vs. 10% USEPA Residential Soil PRGs<sup>a</sup>**  
**Oliver Rubber Company Plant 1**

Chemical	Sample Id.	Detected Concentration (ppm)	10% Residential Soil PRG (ppm)
Ethylbenzene	BH-18	0.0065	23
Chloroform	BH-25	0.0054	0.024
Phenol	BH-21	0.29	3,300

Notes:

a. *Region IX Preliminary Remediation Goals (PRGs) 1998* (USEPA, 1998)

**Table 4**  
**Toxicity Criteria for Chemicals of Interest (COIs)**

Chemical	Inhalation Reference Dose (RfDi) <sup>a</sup> (mg/Kg-day)	Inhalation Slope Factor (SFi) <sup>a</sup> (mg/Kg-day) <sup>-1</sup>
Toluene	1.1E-01	NA
1,1-Dichloroethane	1.4E-01	5.7E-03 <sup>b</sup>
1,1-Dichloroethene	9.0E-03	1.8E-01
1,1,1-Trichloroethane	2.9E-01	NA
Trichloroethene	6.0E-03	1.0E-02 <sup>b</sup>

*Notes:*

NA: Indicates corresponding toxicity criteria were not available.

a: Inhalation slope factor and reference dose were used to evaluate inhalation pathway.

Inhalation slope factors obtained from *Region IX Preliminary Remediation Goals (PRGs) 1998* (USEPA, 1998) unless otherwise noted.

b. Slope Factor obtained from *California Cancer Potency Factors: Update* (OEHHA, 1994).

**Table 5**  
**Representative Concentrations for COIs**

Chemical	RME Scenario	MLE Scenario
<b>Representative Groundwater Concentration (µg/L)</b>		
Toluene	1.0	0.58
1,1-Dichloroethane	11	2.6
1,1-Dichloroethene	260	27
1,1,1-Trichloroethane	99	16
Trichloroethene	1.0	0.58
<b>Vapor Flux Rate (mg/sec-m<sup>2</sup>)</b>		
Toluene	9.3E-08	5.4E-08
1,1-Dichloroethane	2.8E-06	6.5E-07
1,1-Dichloroethene	1.9E-04	2.0E-05
1,1,1-Trichloroethane	3.0E-05	4.7E-06
Trichloroethene	1.4E-07	8.0E-08
<b>Exposure Point Concentration in Indoor Air (mg/m<sup>3</sup>)</b>		
Toluene	6.1E-08	3.5E-08
1,1-Dichloroethane	1.9E-06	4.3E-07
1,1-Dichloroethene	1.2E-04	1.3E-05
1,1,1-Trichloroethane	2.0E-05	3.1E-06
Trichloroethene	9.2E-08	5.3E-08



**Table 6**  
**Parameters for Indoor Air Box Model**

Parameter	Reasonable Maximum Exposure (RME)		Most-Likely Exposure (MLE)	
	Value	Chemical Specific	Value	Same
Emission Rate (E)	Value	Chemical Specific	Value	Same
	Rationale	Emission rate modeled from maximum groundwater concentration using Jury <i>et al.</i> (1983.)	Rationale	Emission rate modeled from average groundwater concentration using Jury <i>et al.</i> (1983)
Emission Area (A)	Value	92.4 (m <sup>2</sup> )	Value	Same
	Rationale	Assumed floor dimensions of residence (1000 ft <sup>2</sup> )	Rationale	Same
Fraction of floor that is cracked (F)	Value	0.001	Value	Same
	Rationale	Grimsrud <i>et al.</i> , 1983	Rationale	Same
Air Exchanges per Hour (ACH)	Value	1.5 (hr <sup>-1</sup> )	Value	Same
	Rationale	ASHRAE, 1989	Rationale	Same
Volume of Air in Building (V)	Value	337.1(m <sup>3</sup> )	Value	Same
	Rationale	Calculated using assumed ceiling height of 12 feet and floor dimensions	Rationale	Same

**Table 7**  
**Exposure Parameters for the RME and MLE Scenarios**

Parameter	Reasonable Maximum Exposure (RME)		Most-Likely Exposure (MLE)	
	Value	Chemical-specific (mg/m <sup>3</sup> ) Modeled based on RME vapor emission rate	Value	Chemical-specific (mg/m <sup>3</sup> ) Modeled based on MLE vapor emission rate
Air Concentration Based on Box Model (C <sub>a</sub> )	Rationale		Rationale	
Breathing Rate (BR)	Value	0.83 m <sup>3</sup> /hr - adult 0.415 m <sup>3</sup> /hr - child	Value	Same
	Rationale	DTSC 1994 PEA Guidance	Rationale	Same
Bioavailability (B <sub>i</sub> )	Value	100 (percent)	Value	Same
	Rationale	Maximum assumed	Rationale	Same
Exposure Time (ET)	Value	24 (hr/day)	Value	Same
	Rationale	DTSC 1994 PEA Guidance	Rationale	Same
Exposure Frequency (EF)	Value	350 (days/year)	Value	Same
	Rationale	USEPA, 1989a	Rationale	Same
Exposure Duration (ED)	Value	24 (years) - adult 6 (years) - child	Value	9 (years) -adult Same
	Rationale	Default values (DTSC, 1992)	Rationale	Adult value based on 50th %ile for time at same residence (USEPA, 1996a)
Body Weight (BW)	Value	70 (Kg) - adult 15 (Kg) - child	Value	Same Same
	Rationale	Default values (DTSC, 1992)	Rationale	Same
Averaging Time (AT)	Value	25,550 (days) (carcinogens) 8760 (days) (noncarcinogens) - adult 2190 (days) (noncarcinogens) - child	Value	Same (carcinogens) 3285 (days) (noncarcinogens) - adult Same (noncarcinogens) - child
	Rationale	Default values (DTSC, 1992)	Rationale	Same

**Table 8**  
**Potential Chemical-Specific Adverse Non-Cancer Health Effects (Hazard Quotients)**  
**Inhalation of Chemical Vapors (Indoor)**

Chemical	RME <sup>a</sup> Scenario		MLE <sup>b</sup> Scenario	
	Adult Resident	Child Resident	Adult Resident	Child Resident
1, 1 Dichloroethane	4.E-06	9.E-06	8.E-07	2.E-06
1,1 Dichloroethene	4.E-03	9.E-03	4.E-04	9.E-04
Toluene	2.E-07	4.E-07	9.E-08	2.E-07
1,1,1-Trichloroethane	2.E-05	4.E-05	3.E-06	7.E-06
Trichloroethylene	4.E-06	1.E-05	2.E-06	6.E-06
<b>Hazard Index</b>	<b>4.E-03</b>	<b>9.E-03</b>	<b>4.E-04</b>	<b>9.E-04</b>

*Notes:*

- a. Reasonable Maximum Exposure
- b. Most-Likely Exposure

**Table 9**  
**Potential Incremental Chemical-Specific Cancer Risks**  
**Inhalation of Chemical Vapors (Indoor)**

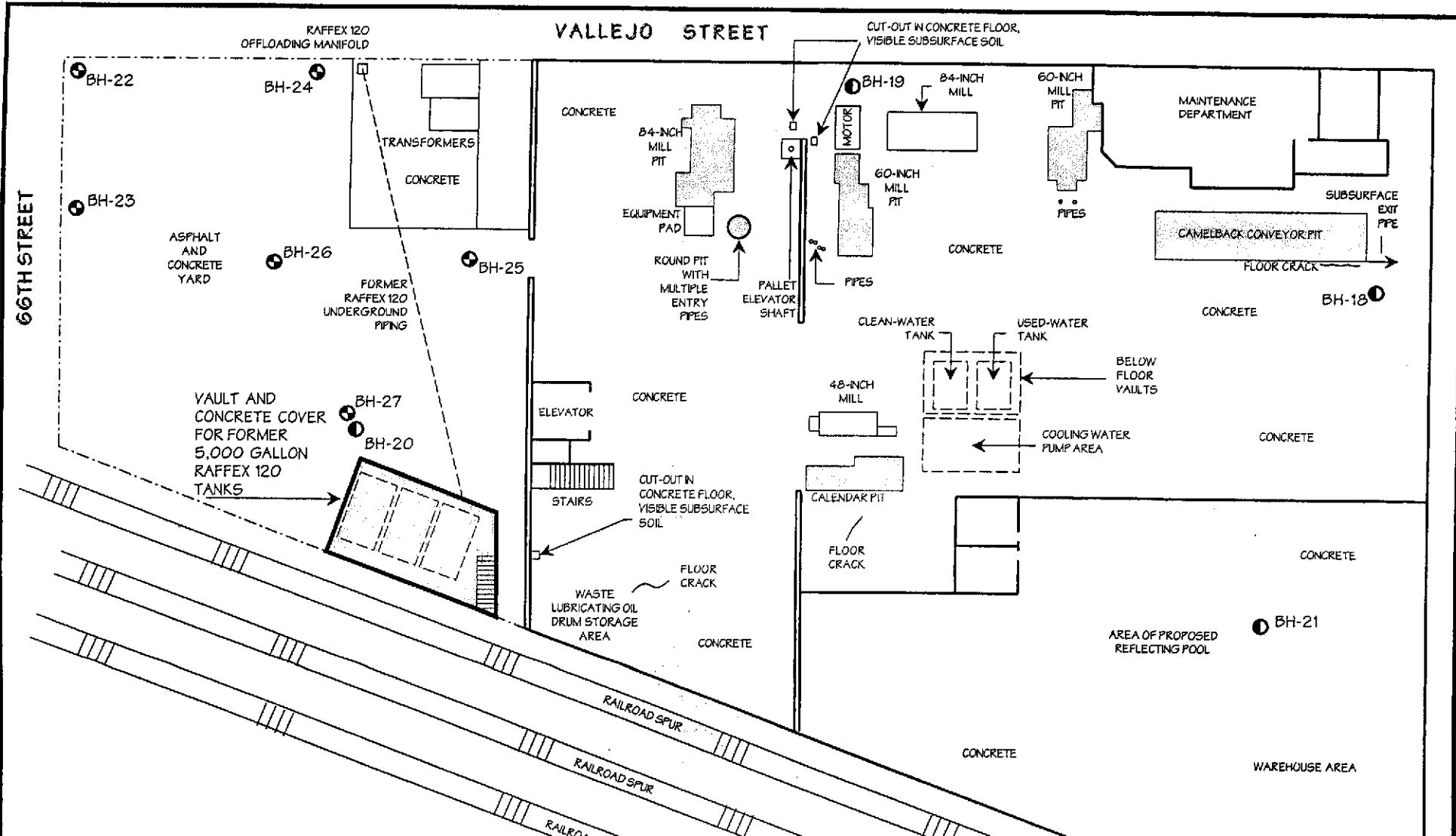
Chemical	RME <sup>a</sup> Scenario		MLE <sup>b</sup> Scenario	
	Adult Resident	Child Resident	Adult Resident	Child Resident
1, 1 Dichloroethane	1E-09	6E-10	9E-11	1E-10
1,1 Dichloroethene	2E-06	1E-06	8E-08	1E-07
Toluene	No SF	No SF	No SF	No SF
1,1,1-Trichloroethane	No SF	No SF	No SF	No SF
Trichloroethylene	9E-11	5E-11	2E-11	3E-11
<b>Receptor Total</b>	<b>2E-06</b>	<b>1E-06</b>	<b>8E-08</b>	<b>1E-07</b>

*Notes:*

No SF - USEPA Cancer Slope Factor not available

a. Reasonable Maximum Exposure

b. Most-Likely Exposure



**SOIL BORING LOCATION MAP**

OLIVER RUBBER COMPANY PLANT I  
1200 65TH STREET  
OAKLAND, CALIFORNIA

AQUA SCIENCE ENGINEERS, INC.    FIGURE 1



NORTH

SCALE  
1" = 30'

**LEGEND**

BH-21	●	SOIL BORING DRILLED SEPTEMBER 2-3, 1998
BH-27	⊙	SOIL BORING DRILLED SEPTEMBER 25, 1998

**NOTE:**  
ALL EQUIPMENT WITHIN BUILDING HAS BEEN REMOVED, ALL SUBSURFACE PITS AND PIPING HAVE BEEN FILLED WITH CONCRETE.

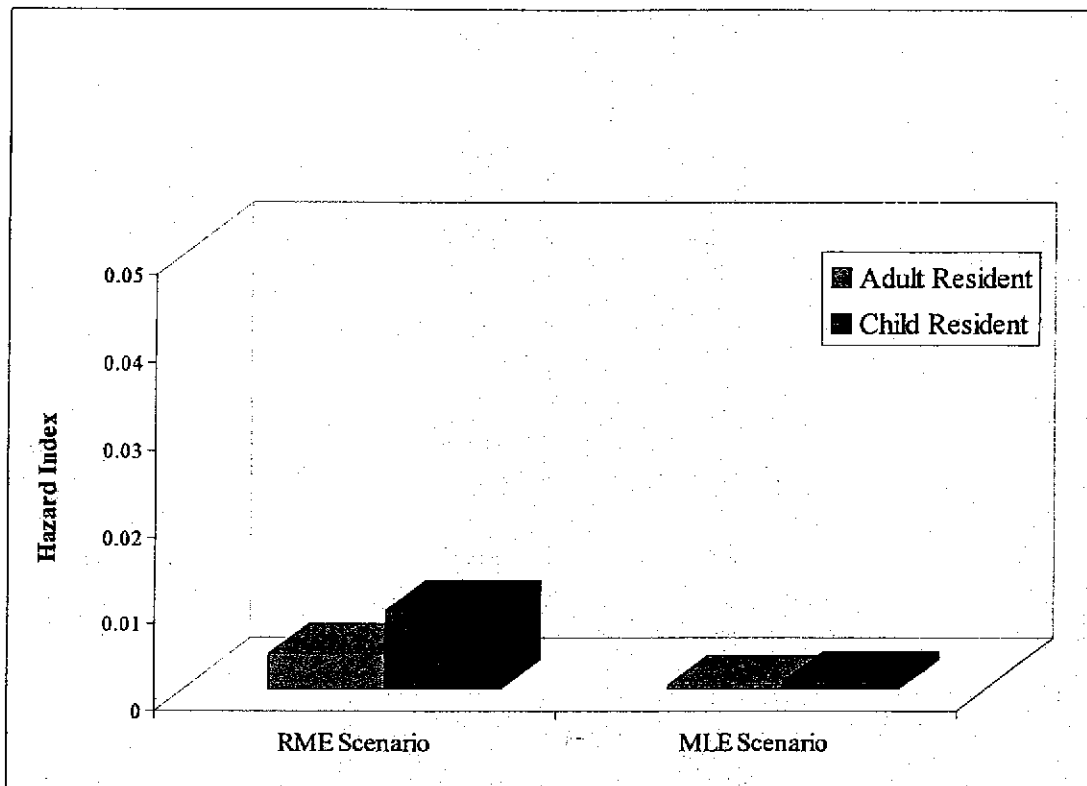
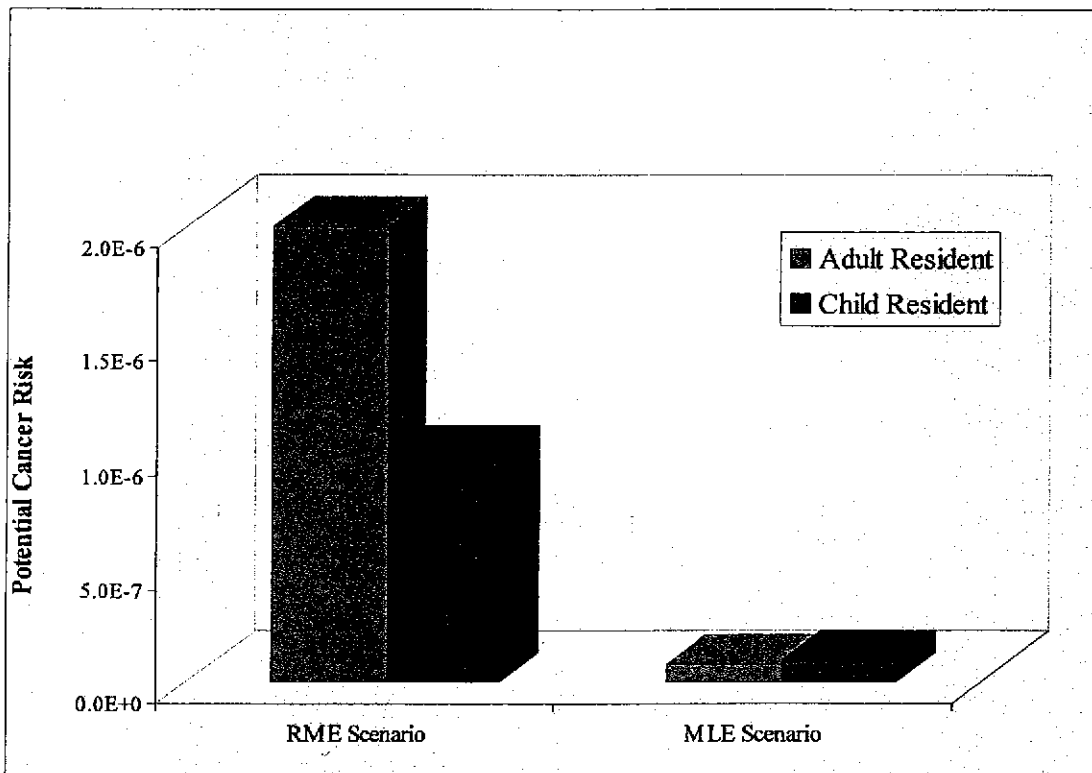


FIGURE 2  
 Cumulative Adverse Non-Cancer Health Effects  
 (Hazard Indices)



**Appendix A**  
**Indoor Air Box Model Calculations and Hazard and Risk Calculation Results**

**RME Indoor Air Concentration Calculation**  
 $C_{in} = (E_i \cdot A \cdot F) / [(ACH/CF) \cdot V]$

Chemical	Emission Rate ( $E_i$ ) (mg/sec-m <sup>2</sup> )	Emission Area (A) (m <sup>2</sup> )	Frctn of Flr that is Crkd (F) (cm <sup>2</sup> /cm <sup>2</sup> )	Air Exchange Rate (ACH) (hr) <sup>-1</sup>	Volume of Air in Bldg. (V) (m <sup>3</sup> )	Conversion Factor (sec/hr)	RME Indoor Air Conc. ( $C_{in}$ ) (mg/m <sup>3</sup> )
1,1-Dichloroethane	2.8E-06	92.4	0.001	1.5	337.1	3600	1.9E-06
1,1-Dichloroethene	1.9E-04						1.2E-04
Toluene	9.3E-08						6.1E-08
1,1,1-Trichloroethane	3.0E-05						2.0E-05
Trichloroethylene	1.4E-07						9.2E-08

**RME Cancer Risk Calculations**  
 $CR = [(C_{in} \cdot BR \cdot B_{IV} \cdot ED \cdot EF \cdot ET) / (BW \cdot AT)] \cdot SF$

Chemical	RME Indoor Air Conc. ( $C_{in}$ ) (mg/m <sup>3</sup> )	Breathing rate (BR) (m <sup>3</sup> /hr)	Bioavailability ( $B_{IV}$ ) (unitless)	Exposure Duration (ED) (years)	Exposure Frequency (EF) (days/year)	Exposure Time (ET) (hr/day)	Body Weight (BW) (kg)	Averaging Time (AT) (days)	Slope Factor (SF) (mg/kg-day) <sup>-1</sup>	Potential Incremental Cancer Risk (CR)
<b>Adult</b>										
1,1-Dichloroethane	1.9E-06	0.83	1	24	350	24	70	25550	0.0057	1.0E-09
1,1-Dichloroethene	1.2E-04								0.18	2.1E-06
Toluene	6.1E-08								No SF	0
1,1,1-Trichloroethane	2.0E-05								No SF	0
Trichloroethylene	9.2E-08								0.01	8.6E-11
<b>Total Cancer Risk</b>										<b>2.1E-06</b>
<b>Child</b>										
1,1-Dichloroethane	1.9E-06	0.415	1	6	350	24	15	25550	0.0057	5.8E-10
1,1-Dichloroethene	1.2E-04								0.18	1.2E-06
Toluene	6.1E-08								No SF	0
1,1,1-Trichloroethane	2.0E-05								No SF	0
Trichloroethylene	9.2E-08								0.01	5.0E-11
<b>Total Cancer Risk</b>										<b>1.2E-06</b>

**RME Hazard Quotient Calculations**  
 $HQ = [(C_{in} \cdot BR \cdot B_{IV} \cdot ED \cdot EF \cdot ET) / (BW \cdot AT)] / RfD$

Chemical	RME Indoor Air Conc. ( $C_{in}$ ) (mg/m <sup>3</sup> )	Breathing rate (m <sup>3</sup> /hr)	Bioavailability (unitless)	Exposure Duration (years)	Exposure Frequency (days)	Exposure Time (hr)	Body Weight (kg)	Averaging Time (days)	Reference Dose (RfD) (mg/kg-day)	Hazard Quotient (HQ)
<b>Adult</b>										
1,1-Dichloroethane	1.9E-06	0.83	1	24	350	24	70	8760	0.14	3.6E-06
1,1-Dichloroethene	1.2E-04								0.009	3.7E-03
Toluene	6.1E-08								0.11	1.5E-07
1,1,1-Trichloroethane	2.0E-05								0.29	1.8E-05
Trichloroethylene	9.2E-08								0.006	4.2E-06
<b>HI</b>										<b>0.004</b>
<b>Child</b>										
1,1-Dichloroethane	1.9E-06	0.415	1	6	350	24	15	2190	0.14	8.5E-06
1,1-Dichloroethene	1.2E-04								0.009	8.7E-03
Toluene	6.1E-08								0.11	3.5E-07
1,1,1-Trichloroethane	2.0E-05								0.29	4.3E-05
Trichloroethylene	9.2E-08								0.006	9.8E-06
<b>HI</b>										<b>0.009</b>



**Appendix A**  
**Indoor Air Box Model Calculations and Hazard and Risk Calculation Results**

**MLE Indoor Air Concentration Calculation**  
 $C_{in} = (E_i \cdot A \cdot F) / ((ACH/CF) \cdot V)$

Chemical	Emission Rate (E <sub>i</sub> ) (mg/sec-m <sup>2</sup> )	Emission Area (A) (m <sup>2</sup> )	Frcn of Flr that is Crkd (F) (cm <sup>2</sup> /cm <sup>2</sup> )	Air Exchange Rate (ACH) (hr) <sup>-1</sup>	Volume of Air in Bldg. (V) (m <sup>3</sup> )	Conversion Factor (sec/hr)	MLE Indoor Air Conc. (C <sub>in</sub> ) (mg/m <sup>3</sup> )
1,1-Dichloroethane	6.5E-07	92.4	0.001	1.5	337.1	3600	4.3E-07
1,1-Dichloroethene	2.0E-05						1.3E-05
Toluene	5.4E-08						3.5E-08
1,1,1-Trichloroethane	4.7E-06						3.1E-06
Trichloroethylene	8.0E-08						5.3E-08

**MLE Cancer Risk Calculations**  
 $CR = [(C_{in} \cdot BR \cdot B_{iv} \cdot ED \cdot EF \cdot ET) / (BW \cdot AT)] \cdot SF$

Chemical	RME Indoor Air Conc. (C <sub>in</sub> ) (mg/m <sup>3</sup> )	Breathing rate (BR) (m <sup>3</sup> /hr)	Bioavailability (B <sub>iv</sub> ) (unitless)	Exposure Duration (ED) (years)	Exposure Frequency (EF) (days/year)	Exposure Time (ET) (hr/day)	Body Weight (BW) (kg)	Averaging Time (AT) (days)	Slope Factor (SF) (mg/kg-day) <sup>-1</sup>	Potential Incremental Cancer Risk (CR)
<b>Adult</b>										
1,1-Dichloroethane	4.3E-07	0.83	1	9	350	24	70	25550	0.0057	8.6E-11
1,1-Dichloroethene	1.3E-05								0.18	8.2E-08
Toluene	3.5E-08								No SF	0
1,1,1-Trichloroethane	3.1E-06								No SF	0
Trichloroethylene	5.3E-08								0.01	1.9E-11
<b>Total Cancer Risk</b>										<b>8.2E-08</b>
<b>Child</b>										
1,1-Dichloroethane	4.3E-07	0.415	1	6	350	24	15	25550	0.0057	1.3E-10
1,1-Dichloroethene	1.3E-05								0.18	1.3E-07
Toluene	3.5E-08								No SF	0
1,1,1-Trichloroethane	3.1E-06								No SF	0
Trichloroethylene	5.3E-08								0.01	2.9E-11
<b>Total Cancer Risk</b>										<b>1.3E-07</b>

**MLE Hazard Quotient Calculations**  
 $HQ = [(C_{in} \cdot BR \cdot B_{iv} \cdot ED \cdot EF \cdot ET) / (BW \cdot AT)] / RID$

Chemical	RME Indoor Air Conc. (C <sub>in</sub> ) (mg/m <sup>3</sup> )	Breathing rate (m <sup>3</sup> /hr)	Bioavailability (unitless)	Exposure Duration (years)	Exposure Frequency (days)	Exposure Time (hr)	Body Weight (kg)	Averaging Time (days)	Reference Dose (RID) (mg/kg-day)	Hazard Quotient (HQ)
<b>Adult</b>										
1,1-Dichloroethane	4.3E-07	0.83	1	9	350	24	70	3285	0.14	8.4E-07
1,1-Dichloroethene	1.3E-05								0.009	3.9E-04
Toluene	3.5E-08								0.11	8.8E-08
1,1,1-Trichloroethane	3.1E-06								0.29	2.9E-06
Trichloroethylene	5.3E-08								0.006	2.4E-06
<b>HI</b>										<b>0.0004</b>
<b>Child</b>										
1,1-Dichloroethane	4.3E-07	0.415	1	6	350	24	15	2190	0.14	2.0E-06
1,1-Dichloroethene	1.3E-05								0.009	9.1E-04
Toluene	3.5E-08								0.11	2.1E-07
1,1,1-Trichloroethane	3.1E-06								0.29	6.8E-06
Trichloroethylene	5.3E-08								0.006	5.6E-06
<b>HI</b>										<b>0.0009</b>