APPENDIX 7

Muscoy Plume OU Preliminary Baseline Risk Assessment
# Table of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>FOREWORD</td>
<td>1</td>
</tr>
<tr>
<td>BACKGROUND OF THE BASELINE RISK ASSESSMENT</td>
<td>3</td>
</tr>
<tr>
<td>1.0 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN</td>
<td>4</td>
</tr>
<tr>
<td>2.0 EXPOSURE ASSESSMENT</td>
<td>5</td>
</tr>
<tr>
<td>2.1 POTENTIALLY EXPOSED POPULATIONS</td>
<td>5</td>
</tr>
<tr>
<td>2.2 POTENTIAL EXPOSURE PATHWAYS</td>
<td>5</td>
</tr>
<tr>
<td>2.3 QUANTIFICATION OF EXPOSURE</td>
<td>6</td>
</tr>
<tr>
<td>2.3.1 Exposure Estimation for Noncarcinogenic Effects</td>
<td>6</td>
</tr>
<tr>
<td>2.3.2 Exposure Estimations for Carcinogenic Effects</td>
<td>6</td>
</tr>
<tr>
<td>2.3.3 Average and Reasonable Maximum Exposure Scenarios</td>
<td>6</td>
</tr>
<tr>
<td>2.4 CHEMICAL CONCENTRATIONS</td>
<td>7</td>
</tr>
<tr>
<td>2.5 EXPOSURE TO CHEMICALS IN HOUSEHOLD TAP WATER</td>
<td>9</td>
</tr>
<tr>
<td>3.0 TOXICITY ASSESSMENT</td>
<td>12</td>
</tr>
<tr>
<td>3.1 NONCARCINOGENIC EFFECTS</td>
<td>12</td>
</tr>
<tr>
<td>3.2 CARCINOGENIC EFFECTS</td>
<td>13</td>
</tr>
<tr>
<td>3.3 TOXICITY VALUES</td>
<td>13</td>
</tr>
<tr>
<td>3.3.1 Reference Dose</td>
<td>15</td>
</tr>
<tr>
<td>3.3.2 Cancer Slope Factor</td>
<td>15</td>
</tr>
<tr>
<td>4.0 HEALTH RISK CHARACTERIZATION</td>
<td>17</td>
</tr>
<tr>
<td>4.1 NONCARCINOGENIC RISKS</td>
<td>17</td>
</tr>
<tr>
<td>4.2 CARCINOGENIC RISKS</td>
<td>17</td>
</tr>
<tr>
<td>4.3 ESTIMATED RISKS-QUANTITATIVE ASSESSMENT</td>
<td>18</td>
</tr>
<tr>
<td>5.0 UNCERTAINTIES</td>
<td>23</td>
</tr>
<tr>
<td>5.1 UNCERTAINTY IN EXPOSURE ASSESSMENT</td>
<td>23</td>
</tr>
<tr>
<td>5.2 UNCERTAINTY IN TOXICITY INFORMATION</td>
<td>24</td>
</tr>
<tr>
<td>5.3 UNCERTAINTY IN THE CHARACTERIZATION OF RISKS</td>
<td>25</td>
</tr>
<tr>
<td>6.0 ECOLOGICAL ASSESSMENT</td>
<td>25</td>
</tr>
<tr>
<td>6.1 POTENTIAL ECOLOGICAL RECEIVERS</td>
<td>25</td>
</tr>
<tr>
<td>6.1.1 Site Description</td>
<td>25</td>
</tr>
<tr>
<td>6.1.2 Ecological Setting</td>
<td>26</td>
</tr>
<tr>
<td>6.2 CHARACTERIZATION OF ECOLOGICAL EXPOSURE AND EFFECTS</td>
<td>27</td>
</tr>
<tr>
<td>6.3 RISK CHARACTERIZATION</td>
<td>28</td>
</tr>
<tr>
<td>BIBLIOGRAPHY</td>
<td>29</td>
</tr>
</tbody>
</table>

(62380-D/mp-rifs.a-7)
LIST OF TABLES

Table 1  Chemicals of Potential Concern .................................................. 8
Table 2  Pathway-Specific Exposure Values ............................................... 10
Table 3  EPA Weight-of-Evidence Categories for Potential Carcinogens .......... 14
Table 4  Dose-Response Variables for Chemicals of Concern ........................ 16
Table 5  Systemic Toxicity Summary: Chronic Hazard Index Estimates, Reasonable
         Maximum Exposure ........................................................................... 19
Table 6  Systemic Toxicity Summary: Chronic Hazard Index Estimates,
         Average Exposure ............................................................................ 20
Table 7  Carcinogenic Risk Estimates: Reasonable Maximum Exposure .......... 21
Table 8  Carcinogenic Risk Estimates: Average Exposure ............................. 22

LIST OF FIGURES

Figure 1  Muscoy Plume OU Area Surface Features ................................. 2
This document presents the results of the preliminary baseline risk assessment for the Muscoy Plume Operable Unit (OU) of the Newmark Groundwater Contamination Superfund Site project. The purpose of this preliminary risk assessment is to support the selection of an interim remedial action. Interim remedial actions do not require a completed baseline risk assessment, although enough information must be available to demonstrate that action is necessary to stabilize the site, prevent further degradation, or achieve significant risk reduction quickly (Preamble to the NCP Final Rule, Federal Register 55:8704, 1990).

The preliminary risk assessment estimates potential, not actual, risk. The risk estimates are based on the unlikely assumption that the federal Safe Drinking Water Act (SDWA) (42 US Code 300(f) et seq.) and the more stringent California Safe Drinking Water Act (Health & Safety Code section 4010 et seq.) drinking water standards (e.g., Maximum Contaminant Levels or MCLs) are not enforced, in which case residents of the San Bernardino area could be supplied with contaminated groundwater extracted from within or near the OU area of contamination. This is only an assumption; drinking water delivered by a public water system must comply with all enforceable drinking water standards. The SDWA and California regulations (22 CCR 64401 et seq.) defines a public water system as one which provides piped water for human consumption to at least fifteen service connections, or regularly serves an average of at least twenty-five individuals at least sixty days per year.

It should be noted that remedial actions must comply with applicable or relevant and appropriate requirements (ARAR). For actions involving contaminated drinking water supplies, the ARARs for treated water to be served as drinking water are federal or state MCLs, whichever are more stringent. An MCL is the maximum permissible level of a contaminant in water which is permitted in drinking water delivered by a public water system. California MCLs, adopted by the California Department of Health Services' (DHS) Office of Drinking Water (22 CCR Division 4, Chapter 15) are ARARs if they are more stringent than MCLs set by EPA under SDWA. State MCLs are set at levels as close to the recommended public health levels as possible but not at levels less stringent than the MCLs set by the EPA under the SDWA.

For remedial actions impacting surface waters regulated under the federal Clean Water Act (CWA), water quality criteria could include National Ambient Water Quality Criteria and standards set pursuant to the state’s Porter-Cologne Water Quality Control Act (Porter-Cologne Act), and the Santa Ana Region Basin Plan.

The preliminary risk assessment examines exposure to volatile organic chemicals (VOCs) detected in groundwater that has impacted an aquifer two miles long by 1.5 miles wide on the west side of the Shandin Hills in San Bernardino and an unincorporated community known as Muscoy. This plume, the Muscoy plume, resulted in the temporary closure of several municipal water supply wells and may threaten other wells in the Muscoy Plume OU area. Figure 1 depicts the location of the Muscoy plume and OU, and possible site of the treatment plant. Vadose zone contamination or remediation is not a goal of the interim action given the absence of a known source or sources in the Muscoy Plume OU and the depth to groundwater. Therefore, exposure to contaminated soil or soil gas will not be addressed in this
preliminary risk assessment. The possibility of exposure to contaminants in the unsaturated zone was explored by sampling for contaminated soils at a suspected source area in the Newmark OU, where groundwater contamination was greater, and by a screening study of the indoor air in nearby residences (see URS 1992).

BACKGROUND OF THE BASELINE RISK ASSESSMENT

Baseline risk assessments are conducted at Superfund sites to fulfill one of the requirements of the National Oil and Hazardous Substances Contingency Plan (NCP). The NCP (40 CFR Part 300) sets forth the manner in which Superfund remediations are planned and conducted. The NCP (Section 300.430[d]) requires that information collected as part of a remedial investigation (RI) be used to conduct a site-specific baseline risk assessment at sites listed on the National Priorities List (NPL) under the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA), as amended by the Superfund Amendments and Reauthorization Act of 1986 (SARA). The baseline risk assessment characterizes the current and potential threats to human health and the environment that may be posed by contaminants in the groundwater. The NCP also specifies that the results of the baseline risk assessment be used to assist in developing "acceptable exposure levels" for use in the feasibility study (FS). Due to the highly urbanized setting of the Muscoy Plume OU, the focus of the baseline risk assessment is public health.

Nevertheless, an ecological risk assessment is provided in Section 6. The assessment qualitatively addresses the possible ecological risks associated with the groundwater contamination within the Muscoy Plume OU. It should be noted that there is no known hydrogeologic groundwater/surface water connection.

In general, the objective of the baseline risk assessment is the qualitative or quantitative characterization of potential human health effects of specific chemical contaminants on individuals or populations if no action is taken to eliminate or reduce the extent of the contamination. The information is then used by a risk manager or decision maker to determine remedial action objectives. The baseline risk assessment process comprises four basic steps: (1) hazard identification (data collection and evaluation), (2) exposure assessment, (3) toxicity assessment, and (4) risk characterization. A summary of the purpose of each element and the organization of this baseline risk assessment is as follows:

- **Hazard Identification:** Data are evaluated and the chemicals of potential concern (COPC) are identified. The COPCs are the site-related contaminants to be included in the risk assessment. Not all contaminants detected in the groundwater are used, such as those infrequently detected and at concentrations not of concern to human health or the environment based on MCLs, risk-based screening concentrations, or preliminary remediation goals (PRGs).

- **Exposure Assessment:** Evaluates the potential for human contact with the contaminants. Identifies potential pathways by which exposures could occur; characterizes the site and potentially exposed populations; and estimates the magnitude, frequency, and duration of exposure (i.e., quantification of exposure).
Toxicity Assessment: Evaluates the potential adverse health effects of COPCs; and the
dose-response relationship between the dose of a particular COPC and the possibility of
an adverse health effect.

Health Risk Characterization: Integrates information from the toxicity and exposure
assessments to estimate potential risks to public health from exposure to site chemicals.
Also defines the cancer risks/adverse noncancerous health effects from multiple
exposure pathways and multiple COPCs.

Uncertainties: Summarizes uncertainties associated with the data, assumptions, and
methodology used in the risk assessment. The general sources of uncertainty include:
environmental sampling, chemical analyses, selection of COPCs, fate and transport
assumptions, exposure assumptions, toxicological data, assumptions for characterizing
multi-chemical and multi-exposure pathway risks, and chemical interactions of the
COPCs.

Ecological Assessment: Qualitatively evaluates areas where potential ecological effects
or impacts may occur.

1.0 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

The preliminary risk assessment is based on groundwater data collected from six wells during EPA
sampling activities in the Muscoy Plume OU area during 1993. Detection of tetrachloroethene (PCE) in
a well was used to determine whether that well was included within the contaminant plume of the Muscoy
Plume OU. Only those wells included or assumed to be within and representative of the contaminants of
the Muscoy plume were used for this baseline risk assessment. These wells consisted of six existing non-
EPA municipal water supply wells (MUNI-104, MUNI-105, MUNI-106, MUNI-107, MUNI-108, MUNI-
109).

Groundwater sampling results from this effort are presented in RI/FS Section 5. The analytical data were
analyzed and validated in accordance with EPA Contract Laboratory Program (CLP) procedures (EPA
1991a). Results (noted by a "J" qualifier) which fell between the instrument detection limit (IDL) and
the contract required quantitation limit (CRQL) are considered estimated quantities. For the purposes of
this baseline risk assessment, the J-qualified data were used in the same way as positive unqualified data.

Eleven VOCs were identified in groundwater from the six Muscoy Plume OU wells. Two of these VOCs
(carbon tetrachloride and chloroform) were detected at or below CRQL in only one sample from two of
the wells (MUNI-106, MUNI-108). These two VOCs were eliminated from further consideration as
possible representative contaminants in the Muscoy plume. The nine remaining chemicals detected in
groundwater are considered COPCs for this preliminary baseline risk assessment.

Five VOCs (PCE, trichloroethene [TCE], cis-1,2-dichloroethene [cis-1,2-DCE], 1,1-dichloroethane [1,1-
DCA], Freon 12, Freon 11) were the most commonly detected contaminants. PCE and Freon 12
(dichlorodifluoromethane) were detected in each of the wells including J-qualified data for PCE from
MUNI-108; Freon 11 (trichlorofluoromethane), cis-1,2-DCE, and TCE were detected in five wells; and
1,1-DCA in four wells. Trans-1,2-dichloroethene and 1,2-dichloropropane were detected in three wells
and one well, respectively. Vinyl chloride was detected in only one well (MUNI-106) and the data was
1 J-qualified. However, vinyl chloride was included as a COPC since it is a known human carcinogen
2 (EPA Category A) and a possible PCE or TCE biodegradation product (see RI/FS Subsection 6.3.1).

3 Inorganic chemicals have been detected in the Muscoy Plume OU municipal supply and monitoring wells
4 at levels consistent with newly constructed wells. In operating public water supply wells within the
5 Muscoy Plume OU, long-term monitoring required by health officials has detected no inorganic chemicals
6 exceeding background levels for the area and none of public health concern. (Analytical results from
7 URS’ (1993) interim groundwater sampling activities detected only one exceedance of inorganic MCLs:
8 the aluminum level in MUNI-108 was reported as 1,350 µg/l, exceeding the state MCL of 1,000 µg/l.)
9 Inorganics are not included as COPCs in this baseline risk assessment.

2.0 EXPOSURE ASSESSMENT

10 Exposure assessment is the determination or estimation of the magnitude, frequency, duration, and route
11 of exposure to contaminant(s). This section identifies the potentially exposed human populations and the
12 means by which these individuals can come into contact with contaminants detected in groundwater in
13 the Muscoy Plume OU area.

2.1 POTENTIALLY EXPOSED POPULATIONS

16 Most of the land in the Muscoy Plume OU area has been developed for residential, commercial, and
17 industrial land use. Water supply wells in the highly contaminated portions of the Muscoy and Newmark
18 OU areas have been shut down, abandoned, or had treatment systems installed. For the purpose of this
19 preliminary risk assessment, it is assumed that future uses of the groundwater in the Muscoy Plume
20 and Newmark OU areas would include use of untreated groundwater for domestic purposes. The
21 potentially exposed populations include residents and workers in the OU area.

2.2 POTENTIAL EXPOSURE PATHWAYS

23 An exposure pathway is the route by which a receptor makes contact with a contaminant source. This
24 preliminary risk assessment is limited to the groundwater exposure pathway. Exposure to contaminants
25 in groundwater could occur through the use of groundwater for domestic purposes. In residences, people
26 can be exposed to contaminants through ingestion of water used for drinking and cooking. They can also
27 be exposed through dermal absorption of contaminants, primarily during bathing and showering, and
28 inhalation of VOCs released from the water into the household air during showering, bathing, cooking
29 or by the use of household appliances such as washing machines. Exposure to contaminants in
30 groundwater can also occur through the use of groundwater for industrial purposes. Workers could be
31 exposed through dermal absorption of contaminants or inhalation of VOCs.

32 Residents and workers could also be exposed to contaminants in groundwater through the transport of
33 VOCs from groundwater through soil and into ambient air or into building through the foundation. This
34 is unlikely to be a significant exposure route in the Muscoy Plume OU area because the depth to
35 groundwater throughout the area is greater than 100 feet. Results of an EPA screening study of the air
36 quality of residences in the Newmark OU, where groundwater was more highly contaminated but the
depth to groundwater and geology are similar to the Muscoy Plume OU, support this conclusion (see URS 1992).

This preliminary risk assessment will quantitatively address risk due to exposure to contaminants in groundwater used for domestic purposes. Based on potential exposure frequency, duration, and estimated intake exposures, residents exposed to contaminated groundwater used for domestic purposes are expected to be the maximally exposed population.

2.3 QUANTIFICATION OF EXPOSURE

Exposure is defined as the contact of an organism with a chemical or physical agent. The measure of exposure (or intake) is expressed as milligrams of chemical per kilogram of body weight per day (mg/kg-day). Six basic factors are used to estimate intake: (1) exposure frequency; (2) exposure duration; (3) ingestion, inhalation, or contact rate; (4) chemical concentration; (5) body weight; and (6) averaging time.

Intake can be calculated by the following general equation:

\[
\text{Intake} = \frac{\text{Concentration} \times \text{Ingestion or Inhalation Rate} \times \text{Exposure Frequency} \times \text{Exposure Duration}}{\text{Body Weight} \times \text{Averaging Time}}
\]

2.3.1 Exposure Estimation for Noncarcinogenic Effects

The intake of chemicals evaluated for noncarcinogenic health effects is estimated over an averaging time dependent on the assessed toxic effect (i.e., health effect). This assessment evaluates chronic exposure to chemicals on the basis of systemic toxic effects and the estimated period of exposure. The averaging time for noncarcinogenic effects is equal to the exposure duration (i.e., 9 or 30 years) for this assessment.

2.3.2 Exposure Estimations for Carcinogenic Effects

The intake of a chemical evaluated for carcinogenic health effects is referred to as the lifetime average chemical intake. The lifetime average chemical intake is calculated by prorating the total cumulative dose of the chemical over an averaging time of an entire life span (assumed to be 70 years) (EPA 1989a, 1989b).

2.3.3 Average and Reasonable Maximum Exposure Scenarios

The need for remedial action at Superfund sites is based on an estimate of the RME or "reasonable maximum exposure." The RME is defined as the "highest exposure that is reasonably expected to occur at a site" (EPA 1989a). The intent of the RME is to estimate a conservative exposure scenario (i.e., well above average) that is still within the range of possible exposures. Each exposure factor has a range of possible values.

Exposure, or contact with a chemical or physical agent, is normalized for time and body weight in terms of intake, expressed in units of mg/kg-day. Intake variable values for a given pathway are selected so that all intake variables result in an estimate of the average and RME scenarios for a given pathway. The
RME reflects the maximum exposure that is reasonably expected to occur at a site and is based on the reasonable maximum exposure concentration(s), contact rate (90%-95% value), frequency and duration of exposure (30 years), body weight (70 kg), and averaging time. The averaging time for noncarcinogenic effects is equal to the exposure duration (9 or 30 years x 365 days/yr), and 70 years x 365 days/yr for carcinogenic effects.

The following sections provide estimates of exposure factors for both the average and the RME scenarios.

2.4 CHEMICAL CONCENTRATIONS

Table 1 provides a list of the COPCs, exposure concentrations (means, standard deviations, 95% UCL, maximum detected concentrations), and drinking water standards. The exposure concentration is the arithmetic average of the concentration to which a population could be potentially exposed during the exposure period. The concentration does not necessarily represent the maximum concentration of potential exposure, because long-term exposure to groundwater at this concentration is not considered reasonable. Because there is uncertainty associated with estimating a true average concentration or level of exposure, the 95 percent upper confidence limit (UCL) of the arithmetic average is commonly used. The 95 percent UCL is a conservative estimate of the average concentration providing reasonable assurance that the true average is not underestimated.

However, due to the limited number of samples there was considerable variability in the measured concentrations of groundwater contaminants. Consequently, the 95 percent UCL on the arithmetic average was high, and in many instances several times greater than the maximum detected value. When this was the case, the maximum detected concentration was used to estimate potential exposure concentrations in accordance with EPA guidance (EPA 1989a). This approach is conservative, but is considered reasonable given the limited Muscoy Plume OU groundwater data. The 95 percent UCL of the data set is used only when this value does not exceed the maximum detected concentration. The 95 percent UCL or maximum detected concentration, as appropriate, is used to evaluate groundwater exposure for both the average and RME exposure scenarios.

If a chemical is not detected in a particular sample, but is detected in other groundwater samples in the Muscoy Plume OU area wells, a value equal to one-half the detection limit is used to calculate the mean, standard deviation, and 95 percent UCL of the data set. The CRQL for all the COPCs was 0.2 µg/L. The 95 percent UCL (lognormal or normal distribution) is calculated in accordance with EPA guidance (EPA 1992) and values presented in Gilbert (1987). The Shapiro-Wilk W Test (see Gilbert 1987) was applied to determine whether the data set was consistent with a normal or lognormal distribution. In cases where duplicate samples were collected, the sample and duplicate results are averaged before summary statistics are calculated. The arithmetic mean, standard deviation, and 95 percent UCL on the arithmetic mean groundwater concentrations are summarized in Table 1.

For the purposes of this baseline risk assessment, it is assumed that contaminant concentrations remain constant for the duration of the exposure period. Unvalidated historical data from municipal wells in the Muscoy Plume OU area have shown some fluctuation in contaminant concentrations in the different wells during past years. Maximum PCE concentrations have ranged from 48 µg/L in MUNI-106 to 0.6 µg/L in MUNI-108; TCE concentrations ranged from 10 µg/L in MUNI-106 to 0.2 µg/L in MUNI-108.
Table 1

CHEMICALS OF POTENTIAL CONCERN

<table>
<thead>
<tr>
<th>Chemical</th>
<th>#Detect/ #Samples</th>
<th>Arithmetic Mean (µg/L)</th>
<th>Standard Deviation (µg/L)</th>
<th>95 Percent Upper Confidence Limit (µg/L)¹</th>
<th>Maximum Detected Concentration (µg/L)</th>
<th>Drinking Water Standards Maximum Contaminant Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tetrachloroethene (PCE)</td>
<td>6/6</td>
<td>9.20</td>
<td>9.20</td>
<td>NA</td>
<td>27</td>
<td>5</td>
</tr>
<tr>
<td>Trichloroethene (TCE)</td>
<td>5/6</td>
<td>1.89</td>
<td>2.25</td>
<td>120²</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>cis-1,2-Dichloroethene</td>
<td>5/6</td>
<td>1.93</td>
<td>2.13</td>
<td>102²</td>
<td>6</td>
<td>70</td>
</tr>
<tr>
<td>trans-1,2-Dichloroethene</td>
<td>3/6</td>
<td>0.17</td>
<td>0.12</td>
<td>NA</td>
<td>0.4</td>
<td>100</td>
</tr>
<tr>
<td>1,1-Dichloroethene</td>
<td>4/6</td>
<td>0.32</td>
<td>0.26</td>
<td>1.28¹</td>
<td>0.8</td>
<td>NA</td>
</tr>
<tr>
<td>1,2-Dichloropropane</td>
<td>1/6</td>
<td>0.13</td>
<td>0.08</td>
<td>NA</td>
<td>0.3</td>
<td>5</td>
</tr>
<tr>
<td>Dichlorodifluoromethane (Freon 12)</td>
<td>6/6</td>
<td>8.15</td>
<td>10.53</td>
<td>16.5¹</td>
<td>28</td>
<td>NA</td>
</tr>
<tr>
<td>Trichlorofluoromethane (Freon 11)</td>
<td>5/6</td>
<td>1.17</td>
<td>1.43</td>
<td>24.8¹</td>
<td>4</td>
<td>NA</td>
</tr>
<tr>
<td>Vinyl Chloride</td>
<td>1/6</td>
<td>0.10²</td>
<td>0</td>
<td>NA</td>
<td>0.1²</td>
<td>2</td>
</tr>
</tbody>
</table>

¹ Lognormal Distribution, " Normal Distribution; determined by Shapiro-Wilk W Test for normal or lognormal density function; quantile value = 0.788, for 6 samples (see Gilbert 1987).
² Concentration estimated, data are qualitatively acceptable (J-qualified).
NA = Not available, or distribution (normal or lognormal) not demonstrated by W Test.
2.5 **EXPOSURE TO CHEMICALS IN HOUSEHOLD TAP WATER**

Human exposure to contaminants in water used for domestic water supply can occur through three routes: ingestion, inhalation, and dermal absorption.

**Ingestion.** People can be directly exposed to contaminants in groundwater through the ingestion of tap water. The degree of exposure to contaminants through ingestion depends on the amount of water ingested on a daily basis, the frequency and duration of exposure, the body weight of an exposed population, and the averaging time for the health effects to occur. The following equations are used for calculating daily chemical intake from ingestion and inhalation of contaminants (COPCs) in drinking water:

\[
I_{\text{ingestion}} = \frac{(C_w \times IR_w \times EF \times ED)}{(BW \times AT)}
\]

\[
I_{\text{inhalation}} = \frac{(C_w \times IR_a \times EF \times ED)}{(BW \times AT)}
\]

where:

- \( I \) = Chemical intake (mg/kg body weight-day)
- \( C_w \) = Chemical concentration in water (mg/L)
- \( IR_w \) = Ingestion rate (liters/day)
- \( IR_a \) = Inhalation rate (m$^3$/day)
- \( K \) = Volatilization factor for VOCs (unitless)
- \( EF \) = Exposure frequency (days/year)
- \( ED \) = Exposure duration (years)
- \( BW \) = Body weight (kg)
- \( AT \) = Averaging time (days)

The average and RME intake variable values for both ingestion and inhalation pathways are presented in Table 2. The volatilization factor, \( K \), is discussed further below.

**Inhalation.** Individuals can also be exposed to VOCs transferred from tap water to the air from showers, baths, toilets, dishwashers, washing machines, and cooking. Chemicals are considered VOCs when they have a molecular weight less than 200 g/mole, vapor pressure greater than 0.001 mm Hg, and a Henry’s Law constant greater than 10E-05 atm-m$^3$/mole. All of the COPCs are VOCs. Studies have suggested that exposure to volatile chemicals from inhalation can be as great as or greater than from ingestion alone. This assessment uses a volatilization constant (\( K \)) with a value of 0.0005 x 1000 t/m$^3$ (1000 t/m$^3$ is a conversion factor used to convert the air concentration to mg/m$^3$). The \( K \) constant is an upper-bound value based on assumed water usage (720 L/day), residence volume (150,000 L), air exchange rate (0.25 m$^3$/hr.), and a 50 percent transfer efficiency (i.e., percentage of the chemical in water that would be transferred into the air). Daily indoor inhalation rates (\( IR_a \)) shown in Table 2 are based on 16 hours of daily indoor exposure at average and RME hourly indoor \( IR_a \) of 0.63 m$^3$/hr and 0.89 m$^3$/hr, respectively (EPA 1989c).
## Table 2

### PATHWAY-SPECIFIC EXPOSURE VALUES

<table>
<thead>
<tr>
<th>Intake Parameter</th>
<th>Exposure Pathway: Ingestion of Chemicals in Drinking Water</th>
<th>Exposure Pathway: Inhalation of Airborne Chemicals (VOCs) in Drinking Water</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average</td>
<td>RME</td>
</tr>
<tr>
<td>Chemical Concentration in Water (C&lt;sub&gt;w&lt;/sub&gt;) (mg/l)</td>
<td>Chemical Specific</td>
<td>Chemical Specific</td>
</tr>
<tr>
<td>Volatilization Factor (K)&lt;sup&gt;1&lt;/sup&gt; (unitless)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Ingestion Rate (IR&lt;sub&gt;I&lt;/sub&gt;) (l/day)</td>
<td>1.4</td>
<td>2</td>
</tr>
<tr>
<td>Inhalation Rate (IR&lt;sub&gt;I&lt;/sub&gt;) (l/m&lt;sup&gt;2&lt;/sup&gt;/day)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Exposure Frequency (EF) (days/year)</td>
<td>350</td>
<td>365</td>
</tr>
<tr>
<td>Exposure Duration (ED)&lt;sup&gt;3&lt;/sup&gt; (years)</td>
<td>9</td>
<td>30</td>
</tr>
<tr>
<td>Body Weight (BW) (kilograms)</td>
<td>70</td>
<td>70</td>
</tr>
<tr>
<td>Averaging Time (AT) for Noncarcinogenic Effects (years x 365 days/yr)</td>
<td>9</td>
<td>30</td>
</tr>
<tr>
<td>Averaging Time (AT) for Carcinogenic Effects (years x 365 days/yr)</td>
<td>70</td>
<td>70</td>
</tr>
<tr>
<td>Intake&lt;sup&gt;4&lt;/sup&gt; Noncarcinogenic Effects (mg/kg-day)</td>
<td>0.0192 x C&lt;sub&gt;w&lt;/sub&gt;</td>
<td>0.0286 x C&lt;sub&gt;w&lt;/sub&gt;</td>
</tr>
</tbody>
</table>

(62380-D/mp-rifs.a-7)
### Table 2 (Cont’d.)

**PATHWAY-SPECIFIC EXPOSURE VALUES**

<table>
<thead>
<tr>
<th>Intake Parameter</th>
<th>Exposure Pathway: Ingestion of Chemicals in Drinking Water</th>
<th>Exposure Pathway: Inhalation of Airborne Chemicals (VOCs) in Drinking Water</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average</td>
<td>RME</td>
</tr>
<tr>
<td>Intake(^4) Carcinogenic Effects (mg/kg-day)</td>
<td>0.00247 x (C_w)</td>
<td>0.0122 x (C_w)</td>
</tr>
</tbody>
</table>

1. Volatilization factor is a default upper-bound value for VOCs based on Andelman (1990) of 0.0005 x 1000 \(t/m^3\) for an assumed water usage of 720 \(t/day\), residence volume of 150,000 \(t\), air exchange rate of 0.25 \(m^3/hr\), and a 50% transfer efficiency.

2. Average and RME inhalation rates are EPA Exposure Factors based on 0.63 \(m^3/hr\) and 0.89 \(m^3/hr\), respectively for 16 hours/day (see EPA 1989c, pp. II-1-24).

3. Exposure durations for average and RME exposures based on national median (50th percentile) of 9 years at one residence, and national upper bound (90th percentile) at one residence of 30 years, respectively (EPA 1989c).

4. Intake via ingestion or inhalation is the product of the computed exposure values (in units of \(t/kg-day\)) and the chemical-specific water concentration or \(C_w\) (mg/l).

NA Not applicable.
Dermal. Another potential route of exposure associated with water use is dermal absorption of contaminants. Dermal absorption could occur during bathing, showering, food preparation, and washing dishes. Skin is not very permeable and, therefore, is a relatively good lipid barrier separating humans from their environment; however, some chemicals can be absorbed by the skin in sufficient quantities to produce systemic effects. Absorption of a chemical requires passage through the outer skin layer. Passage through the primary barrier (stratum corneum) is the rate limiting step in dermal absorption. It appears that, in general, toxicants move across the stratum corneum by passive diffusion following Fick's Law.

Dermal absorption of volatile compounds in pure form or dilute solution has been observed and documented by several studies (Dutkiewitz and Tyros 1967, 1968; Scheuplein and Blank 1971; Scheuplein and Ross, 1974). There have been varying estimates on the amount of chemical intake that can result from dermal absorption of chemicals in water (Brown et al. 1984; Levin et al. 1984). Cothern et al. (1985) suggest that intake through dermal absorption would normally be much less (by several orders of magnitude) than either the ingestion or inhalation routes in a household setting where exposure comes from the water supply. Estimation of household exposures by Foster and Chrostowski (1986) yielded similar results.

This risk assessment assumes that dermal absorption in the residential use setting is not a significant route of exposure.

Other. A lifetime average body weight of 70 kg is used to estimate both the average and RME scenarios (EPA 1989b). Exposure frequencies of 350 and 365 days/year are assumed for the average and RME scenarios, respectively. For the average exposure scenario the national median (50th percentile) residence time of 9 years at once residence is used; for the RME scenario the national upper-bound (90th percentile) of 30 years is used (EPA 1989a).

3.0 TOXICITY ASSESSMENT

In this preliminary risk assessment, human health risks are evaluated in terms of noncarcinogenic and carcinogenic effects. The noncarcinogenic health effects of chemicals with carcinogenic risks must also be considered. The toxicological information presented in this section includes the dose-response data, that describe or predict the relationship of a quantified dose to a possible human health effect. The dose-response data are derived values that estimate the risk to exposed individuals for (a) given exposure route(s) (e.g., ingestion, inhalation, and dermal contact).

3.1 NONCARCINOGENIC EFFECTS

Chemicals causing noncarcinogenic effects are believed to exhibit a safe threshold level of exposure (from above zero to some finite level) that can be tolerated by the organism without causing an adverse health effect. Noncarcinogenic health effects include a variety of toxic effects on body systems, ranging from renal toxicity (toxicity to the kidney) to central nervous system disorders. It is believed that organisms might have protective mechanisms that must be overcome before a toxic endpoint (effect) is manifest.
Three types of toxicological data are normally used to derive estimates of toxicity: laboratory animal experimental data (e.g., short-term [acute] and/or long-term [chronic] animal studies) data adapted or derived from structurally similar chemicals, and actual human experience (i.e., epidemiological studies).

### 3.2 CARCINOGENIC EFFECTS

Carcinogenesis is generally thought to be a phenomenon for which risk evaluation based on presumption of a threshold is inappropriate (i.e., mutagenic and carcinogenic effects have no dose threshold). It is assumed that a smaller number of molecular events can evoke changes in a single cell that can eventually lead to cancer. This hypothesized mechanism for carcinogenesis is referred to as "nonthreshold" because there is assumed to be essentially no level of exposure to such a chemical that does not pose a finite probability, however small, of generating a carcinogenic response (i.e., no dose is considered risk free).

EPA has developed a carcinogen classification system that uses a weight-of-evidence approach to classify the likelihood of a chemical being a human carcinogen. The current classification, as shown in Table 3, is adjusted upward or downward by the EPA based on tumor data from human and animal studies as well as supporting carcinogenic data from physiological, biochemical, toxicological, metabolic and pharmacokinetic studies, and microbiological studies and cell cultures.

### 3.3 TOXICITY VALUES

Toxic response depends on the dose or concentration of the toxicant. Toxicity values are a quantitative expression of the dose-response relationship, and take the form of reference doses (RfDs) for noncarcinogenic effects and cancer slope factors (CSFs) for carcinogenic effects. Both RfDs and CSFs are specific to the exposure route.

The primary source of toxicity values used in this risk assessment is EPA’s Integrated Risk Information System (IRIS) data base (EPA 1994a). The IRIS data base, which is updated monthly, contains up-to-date health risk and EPA regulatory information. IRIS contains only those RfDs and CSFs that have been verified by EPA work groups and are considered by EPA to be the preferred source of toxicity information.

If a toxicity value is not available through IRIS, the next data source consulted is the most recently available Health Effects Assessment Summary Tables (HEAST) issued by EPA’s Office of Research and Development. HEAST, which is updated quarterly, summarizes interim (and some verified) RfDs and CSFs.

The California Environmental Protection Agency (Cal EPA) has developed CSFs, called cancer potency factors, for use in CERCLA risk assessments as well as regulatory actions or standards. The updated Cal EPA (1994) list of criteria for carcinogens, which is expected to be released by the end of 1994, was consulted as a source of CSFs when EPA-derived values were not available and to determine whether the state values were more stringent, and possibly more protective of human health.
### Table 3

**EPA WEIGHT-OF-EVIDENCE CATEGORIES FOR POTENTIAL CARCINOGENS**

<table>
<thead>
<tr>
<th>EPA Category</th>
<th>Description of Group</th>
<th>Description of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>Human Carcinogen</td>
<td>Sufficient evidence from epidemiology studies to support causal association between exposure and human cancer.</td>
</tr>
<tr>
<td>Group B1</td>
<td>Probable Human Carcinogen</td>
<td>Limited evidence of carcinogenicity in humans from epidemiology studies.</td>
</tr>
<tr>
<td>Group B2</td>
<td>Probable Human Carcinogen</td>
<td>Sufficient evidence of carcinogenicity in animals; inadequate evidence of carcinogenicity in humans.</td>
</tr>
<tr>
<td>Group C</td>
<td>Possible Human Carcinogen</td>
<td>Limited evidence of carcinogenicity in animals; no data for humans.</td>
</tr>
<tr>
<td>Group D</td>
<td>Not Classifiable as to Human Carcinogenicity</td>
<td>Inadequate evidence of carcinogenicity in animals.</td>
</tr>
<tr>
<td>Group E</td>
<td>No Evidence of Carcinogenicity in Humans</td>
<td>No evidence of carcinogenicity in at least two adequate animal tests or in both epidemiology and animal studies.</td>
</tr>
</tbody>
</table>
Cal EPA’s inhalation CSFs, or cancer potency factors, for PCE and TCE, and the oral CSF for TCE, which are more stringent than the provisional EPA (ECAO) values (2.0E-03 inhalation CSF for PCE; 6.0E-03 and 1.1E-02 inhalation and oral CSFs, respectively, for TCE), are used for this risk assessment. The Cal EPA cancer potency factor for both inhalation and oral exposure to 1,2-dichloropropane, the only other probable or possible human carcinogen among the COPCs, is 6.3E-02.

The EPA’s Environmental Criteria and Assessment Office (ECAO) derives provisional toxicity values. Although these values are subject to change ECAO is another source of EPA toxicity values when they are not available through IRIS, HEAST, or Cal EPA. ECAO also provides technical guidance for the evaluation of chemicals without toxicity values as well as other toxicity information.

3.3.1 Reference Dose

The toxicity value describing the dose-response relationship for noncarcinogenic effects is the RfD, generally expressed in units of mg/kg-day. Inhalation RfDs may be expressed as either mg/kg-day or milligrams per cubic meter (mg/m³) of air, commonly referred to as reference concentrations (RfCs). Chronic RfDs are an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure to the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA 1989a).

The RfDs used in this assessment are presented in Table 4. Whenever route-specific RfDs are not available for some of the chemicals detected in groundwater in the Muscoy Plume OU area, RfDs are extrapolated from the other route of exposure. In most instances, this involved the use of oral RfDs for inhalation RfDs.

In the absence of IRIS or HEAST values, EPA’s ECAO provisional oral RfD value of 0.006 (mg/kg-day) for trichloroethene (EPA 1994b) has been used for both the oral and inhalation exposure pathway in this preliminary risk assessment.

3.3.2 Cancer Slope Factor

The dose-response relationship for carcinogens is expressed as a cancer slope factor (CSF). The CSFs used in this risk assessment are presented in Table 4. Generally, the CSF is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical averaged over a 70-year lifetime.

The approach used to estimate the slope factor from animal studies or human data assumes a dose-response relationship with no threshold. Cancer risks estimated by this method produce an estimate that provides a rough but plausible upper limit of risk (i.e., it is not likely that the true risk would be much more than the estimated risk, but it could be considerably lower [EPA 1989a]).
### Table 4

**DOSE-RESPONSE VARIABLES FOR CHEMICALS OF CONCERN**

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Reference Doses (RfDs) for Noncarcinogen Effects</th>
<th>Cancer Slope Factors (CSFs) for Carcinogenic Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Oral RfD (oRfD) (mg/kg-day)</td>
<td>Source</td>
</tr>
<tr>
<td>Tetrachloroethene (PCE)</td>
<td>0.01</td>
<td>IRIS</td>
</tr>
<tr>
<td>Trichloroethene (TCE)</td>
<td>0.006</td>
<td>ECAO</td>
</tr>
<tr>
<td>cis-1,2-Dichloroethene</td>
<td>0.01</td>
<td>HEAST</td>
</tr>
<tr>
<td>trans-1,2-Dichloroethene</td>
<td>0.02</td>
<td>IRIS</td>
</tr>
<tr>
<td>1,1-Dichloroethane</td>
<td>0.10</td>
<td>HEAST</td>
</tr>
<tr>
<td>1,2-Dichloropropane</td>
<td>0.001</td>
<td>X</td>
</tr>
<tr>
<td>Dichlorodifluoromethane (Freon 12)</td>
<td>0.021</td>
<td>IRIS</td>
</tr>
<tr>
<td>Trichlorofluoromethane (Freon 11)</td>
<td>0.30</td>
<td>IRIS</td>
</tr>
<tr>
<td>Vinyl Chloride</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

1. IRIS = Integrated Risk Information System (EPA 1994a)
   HEAST = Health Effects Assessment Tables (EPA 1994b)
   ECAO = Environmental Criteria and Assessment Office (EPA 1994b)
   Cal EPA = California Environmental Protection Agency (Cal EPA 1994)
   X = Extrapolated from other exposure route
   NA = Not Available or Not Applicable. There is no toxicity value (RfD or CSF) listed since it is either not an appropriate route of exposure or because a value has not been derived.

2. Weight-of-Evidence Groups are listed in Table 3.