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RISK ASSESSMENT WORK PLAN

Former Montrose and Stauffer Facilities Henderson, Nevada

Prepared for

**Montrose Chemical Corporation of California
Stauffer Management Company LLC
Syngenta Crop Protection, Inc.
Olin Corporation**

Prepared by

Integral Consulting Inc.
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REVISED
June 2010



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To: William Knight	
From: Jim Lape	
Re: Revised Risk Assessment Work Plan	

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cc: BMI Compliance Coordinator, NDEP [CD enclosed]
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ACRONYMS AND ABBREVIATIONS

ABS _{GI}	fraction of contaminant absorbed in gastrointestinal tract
ACD	Agricultural Chemical Division
ACF	area correction factor
ADD	average daily dose
ALM	adult lead methodology
ARR	asbestos-related risk
AT	averaging time
ATSDR	Agency for Toxic Substances and Disease Registry
BaP	benzo(a)pyrene
BCL	basic comparison level
bgs	below ground surface
BHC	benzene hexachloride
BMI	Black Mountain Industrial
BRC	Basic Remediation Company
BSA	benzenesulfonic acid
BW	body weight
Cal/EPA	California Environmental Protection Agency
cm	centimeter
COPC	constituent of potential concern
CSF	cancer slope factor
CSM	conceptual site model
CTE	central tendency exposure
DAD	dermally absorbed dose
DCBP	dichlorobenzophenone
DEPT	diethyl phosphorodithoic acid
DMPT	dimethyl phosphorodithoic acid
DQA	data quality assessment
EC	exposure concentration

ED	exposure duration
EF	exposure frequency
EPA	U.S. Environmental Protection Agency
EPC	exposure point concentration
ET _{fi}	fraction of time spent indoors
ET _{fo}	fraction of time spent outdoors
f/cm ³	fiber per cubic centimeter
ft	foot
GI	gastrointestinal tract
GSF	gamma shielding factor
HCL	hydrochloric acid
HEAST	Health Effects Assessment Summary Tables
HHRA	human health risk assessment
HI	hazard index
HQ	hazard quotient
in.	inch
Integral	Integral Consulting Inc.
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
J & E	Johnson & Ettinger
kg	kilogram
LADD	lifetime average daily dose
lb	pound
LOAEL	lowest-observed-adverse-effects level
m ²	square meter
MDA	minimum detectable activity
MDL	method detection limit
mg/kg	milligram per kilogram
mg/kg-day	milligram per kilogram per day
mg/m ³	milligram per cubic meter

Montrose	Montrose Chemical Company of California
MRL	minimal risk level
NDEP	Nevada Division of Environmental Protection
NHANES	National Health and Nutrition Examination Survey
NOAEL	no-observed-adverse-effects level
Olin	Olin Corporation
ORNL	Oak Ridge National Laboratory
PAH	polycyclic aromatic hydrocarbon
PbB ₀	baseline blood lead level
PCB	polychlorinated biphenyl
pCBSA	4-chlorobenzene sulfonic acid
PCDD	polychlorinated dibenzo- <i>p</i> -dioxin
PCDF	polychlorinated dibenzofuran
pCi	picocurie
PEF	particulate emission factor
Pioneer	Pioneer Americas, LLC
POD	point of departure
ppb	part per billion
PPRTV	provisional peer reviewed toxicity values
PRG	preliminary remediation goal
QC	quality control
RAGS	Risk Assessment Guidance for Superfund
RAIS	Risk Assessment Information System
RAS	remedial alternatives study
RAWP	risk assessment work plan
RCRA	Resource Conservation and Recovery Act
RfC	reference concentration
RfD	reference dose
RME	reasonable maximum exposure
RPF	relative potency factor

SE	secular equilibrium
SMC	Stauffer Management Company LLC
SOP	standard operating procedure
SQL	sample quantitation limit
SRC	site-related chemical
Stauffer	Stauffer Chemical Company
SVOC	semivolatile organic compound
Syngenta	Syngenta Crop Protection, Inc.
TCDD	tetrachlorodibenzo- <i>p</i> -dioxin
TEF	toxic equivalency factor
TEQ	toxic equivalent
TEM	transmission electron microscopy
TIC	tentatively identified compound
Timet	Titanium Metals Corporation
TPH	total petroleum hydrocarbon
Tronox	Tronox, Inc.
UCL	upper confidence limit
UF	uncertainty factor
µg/dL	microgram per deciliter
µg/m ³	microgram per cubic meters
µm	micrometer
VOC	volatile organic compound
WHO	World Health Organization
WOE	weight-of-evidence

1 INTRODUCTION

This risk assessment work plan (RAWP) outlines the proposed approach for assessing potential human health risks at the former Montrose and Stauffer facilities in Henderson, Nevada (the Site). This RAWP has been prepared on behalf of Montrose Chemical Company of California (Montrose), Stauffer Management Company LLC/Syngenta Crop Protection, Inc. (SMC/Syngenta) and Olin Corporation (Olin) (the Companies) as part of the overall effort to characterize the nature and extent of contamination at this former facility and determine the need for, and effectiveness of, remedial actions to address overall risks.

1.1 SITE DESCRIPTION¹

The former Montrose and Stauffer facilities are located in the southwest portion of a heavily industrialized area currently referred to as the Black Mountain Industrial (BMI) Complex. The BMI Complex is located within an unincorporated portion of Clark County surrounded by the City of Henderson, NV. Under current operations, the BMI Complex includes property owned, leased, or administered by Olin (and formerly Pioneer Americas LLC [Pioneer]), Tronox, Inc. (Tronox), Titanium Metals Corporation (Timet), Chemstar Lime Company, and Basic Remediation Company (BRC) and its affiliates (Figure 1-1). The Site, as referred to in this work plan, comprises the portion of the BMI complex previously utilized by Montrose and Stauffer Chemical Company (Stauffer) and currently owned and operated by Olin for the production of liquid chlorine, caustic soda, hydrochloric acid (HCL), and bleach (Figure 1-2). The total acreage of the Site is approximately 315 acres.

1.1.1 Site Setting

The Site is located within the Las Vegas Valley and the southwestern part of the Basin and Range physiographic province. The climate is arid with precipitation averaging slightly less than 4.5 in. per year (NOAA 2009). Winters are mild and summers are hot with temperatures often above 100 degrees Fahrenheit (°F). The average annual daily temperatures range from a low of approximately 56°F to a high of approximately 80°F (NOAA 2009).

Land surface at the Site is a mixture of natural and non-native materials. Some portions of the Site are paved. Outside of these areas, most of the land surface is bare soil or sparsely vegetated. Surface and near-surface soils at the Site are generally coarse-grained, comprised of quaternary alluvium deposits consisting of sands and gravels, with varying amounts of silts and occasional cobbles (Hargis 2008). In some areas, caliche is present on the surface (PES 2006, 2007).

¹ The information summarized in this section is largely excerpted from previous reports prepared by PES (2006, 2007) and Hargis (2008).

Natural site drainage is to the north, but no perennially wet drainages or other natural water bodies exist on the Site. Wind direction is variable, but predominately from the northwest, south, southwest, and southeast (Figure 1-3).

The Site is currently used exclusively for industrial processes. Site access to Olin's current operating facility is controlled by gates and a guard house. A 6-ft high chain-linked razor wire-topped perimeter fence exits around portions of the Site.

Areas immediately adjacent to the Site are undeveloped or industrial/commercial. The nearest residences occur to the west-northwest, south, and southeast and are located more than 1/2- mile from the Site.

1.1.2 Site History

The Site was first developed as part of the original BMI Complex, which was constructed under a contract with the U.S. Defense Plant Corporation and operated by BMI to produce magnesium for the World War II effort from 1942 through 1944. Chlorine was essential to magnesium production and a chlorine and caustic soda plant was constructed at the Site (PES 2006).

From 1945 through 1984, the Site was operated by Stauffer for production of chlorine, sodium hydroxide, HCL, and agricultural chemical products (PES 2006). The most extensive operations included the manufacture of chlorine and caustic soda from 1945 through 1984, and the production of HCL from 1954 to 1984. Stauffer also manufactured the pesticides trithion® (carbophenothion) (1958 through 1984), imidan® (phosmet) (1964 through 1982), parachlorothiophenol (1960 through 1984), and thiophenol (1967 through 1982) at its Agricultural Chemical Division (ACD) Plant. Lindane (gamma-benzene hexachloride [BHC]) was produced at the former Lindane Plant from 1946 through 1958. The Stauffer manufacturing facilities were largely demolished in 1984.

Montrose constructed and operated a manufacturing plant to produce a variety of organic chemicals from 1947 through 1983 (Hargis 2008). Organic chemical products included chlorobenzene, polychlorinated benzenes (PCBs), chloral, and 4,4'-dichlorobenzil. Montrose ceased operations at the organic chemical plant in 1983 and demolished the plant in 1984. Montrose also constructed a manufacturing plant for the production of synthetic HCL in 1954 and at an expanded facility constructed in 1977 (Hargis 2008). Montrose produced HCL at these production facilities until 1985.

Olin currently operates chlor alkali production facilities at the Site and manufactures liquid chlorine, caustic soda, HCL, and bleach. Olin began operation in 2007 when they acquired Pioneer.

1.2 SCOPE AND FOCUS OF THE HUMAN HEALTH RISK ASSESSMENT

This document has been prepared to satisfy the Nevada Division of Environmental Protection (NDEP) requirements to provide a RAWP detailing the human health risk assessment (HHRA) methodology as part of the overall remedial alternatives studies (RAS) to be conducted at the Site (NDEP 2008a). As outlined by NDEP, RAS will be conducted at various source areas and potential source areas at the Site. This RAWP has been developed to detail the procedures to be used to evaluate human health risks at the areas where risk-based closure may be sought by the Companies, and/or where the evaluation of human health risks is appropriate to support the evaluation of remedial action alternatives.

The HHRA approach outlined here is consistent with basic procedures recommended by the U.S. Environmental Protection Agency (EPA) for conducting risk assessments at waste sites. Documents that will guide the risk assessment include, but are not limited to, the following:

- *Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual, Part A (USEPA 1989)*
- *Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual, Part E (USEPA 2004a)*
- *Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual, Part F (USEPA 2009a)*
- *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites (USEPA 2002a).*

In addition, the risk assessment will follow guidance developed by NDEP applicable to risk assessment, including data evaluation to support risk assessment, provided at the following website: <http://ndep.nv.gov/bmi/technical.htm>.

The focus of the HHRA will be to evaluate risks associated with conditions that exist, or are anticipated to exist, at the various source areas and potential source areas following implementation of the remedial decision (i.e., conditions at "closure"). Remedial decisions may include an active remedy and/or no further action. For purposes of the NDEP RAS process, these "post-remedy" or "closure" conditions constitute the baseline condition for each area that will be evaluated in the risk assessment.

The various source areas and potential source areas at the Site that may be evaluated via a HHRA are identified in the RAS document. The RAS document also identifies site assessment areas that were developed by grouping various source areas in order to simplify and organize future investigation and RAS activities. The HHRA will focus on potential exposures within ½-acre areas across the source/site assessment area. If sampling data for multiple ½-acre exposure areas exhibit similar concentration distributions they may be combined for evaluation in the HHRA. Sampling data may not be available within each of the ½-acre exposure areas of a

source/site assessment area; however, assumptions of similar concentration distributions across areas larger than ½-acre may allow the risk assessment to be applied to combined exposure areas. Aggregated ½-acre exposure areas, as supported by the data, would become decision units for the risk assessment. Use of the decision units would allow for risk management decisions to be made simultaneously for many ½-acre exposure areas within a source/site assessment area based on a similarity in the contaminant concentration distribution that allows for aggregation of individual exposure areas. Details on the manner in which data will be treated and risk will be characterized for source/site assessment areas is described in more detail in the remainder of this RAWP.

The HHRA will address potential exposures and risks assuming that the overall site will remain an industrial property after closure. As such, the assessment assumes that deed restrictions and institutional controls that limit the use of the site to industrial activity will be put in place as part of remedial actions. If such restrictions and controls are not implemented the conclusions of the risk assessment cannot be used to predict risks to receptors under alternate future use scenarios.

The HHRA will address potential exposures to onsite industrial/commercial workers, construction workers, and maintenance workers quantitatively. A qualitative assessment will be conducted to evaluate potential exposures to trespassers, and offsite residents. Potential exposures to constituents of potential concern (COPCs) detected in surface (i.e., 0-6 in. below ground surface [bgs]) and shallow soils (i.e., 0-10 ft bgs) will be evaluated for the direct contact pathways, as well as inhalation of vapor-phase and particulate-sorbed contaminants in indoor and outdoor air. For deeper vadose zone soils (i.e., >10 ft bgs) and groundwater, the potential for vapors to migrate from the subsurface to indoor and outdoor air also will be evaluated.

Groundwater is being addressed from a non-degradation standpoint and only factors into the HHRA via indirect exposures related to inhalation of volatiles released from groundwater beneath the Site. Direct exposures to groundwater via consumptive use will not be subject to a formal risk assessment. Instead, to support management decisions regarding remedial actions, groundwater quality data will be compared with chemical- and radionuclide-specific standards that define acceptable risk levels for consumptive use. Additionally, in order to characterize potential impacts of soils on groundwater quality, soil data (0-10 ft bgs) will be compared to NDEP basic comparison levels (BCLs) for leaching. Tables presenting the comparisons to leaching BCLs will be included in the risk assessment. However, evaluations related to protection of the overall quality of groundwater as a resource will be conducted separately as part of the remedial alternatives assessment.

1.3 RISK AND CHEMICAL-SPECIFIC GOALS

Remediation goals for a source/site assessment area will be developed on a case-by-case basis as part of the overall RAS process. The following conditions will be applied in development of the remediation goals.

1. Post-remediation chemical concentrations and radionuclide activities in Site soils will have a cumulative theoretical upper-bound incremental carcinogenic risk level point of departure (POD) of 10^{-6} . For cases where NDEP concurs that this goal is unfeasible, the goal may be re-evaluated in accordance with EPA guidance (USEPA 1991, 1995). The POD risk goal will be evaluated separately for chemicals, asbestos, and radionuclides.
2. Post-remediation chemical concentrations in Site soils are targeted to have an associated cumulative, noncancer hazard index (HI) of 1.0 or less. If the HI is determined to be greater than 1.0, target organ-specific HIs may be calculated for primary and secondary organs. The final risk goal will be to achieve target organ-specific noncancer HIs of 1.0 or less.
3. The risk-based target goal for lead in soil is 800 mg/kg for industrial/commercial land use. This is based on the EPA's Adult Lead Methodology (ALM) using default input factors for an industrial/commercial worker (USEPA 1996a, NDEP 2009a).
4. Where background levels exceed risk-based levels, Site soils are targeted to have risks no greater than those associated with background conditions.
5. Cancer risks from asbestos are based on the estimated additional deaths from lung cancer or mesothelioma due to constant lifetime exposure. The risk-based POD for asbestos is 10^{-6} . As mentioned above, risk from asbestos is evaluated separately from other chemicals and radionuclides. For cases where NDEP concurs that this goal is unfeasible, the goal may be re-evaluated in accordance with EPA guidance (USEPA 1991, 1995).
6. The target goal for dioxin/furan toxic equivalents (TEQ) for commercial and industrial land use is 1 ppb. This value is based on the 1998 USEPA OSWER Directive with a modification to address identified uncertainties (10-fold uncertainty factor) regarding cancer potency in humans that results in a screening range of 0.5-2 ppb. A single value of 1 ppb was selected (NDEP 2009a). Risks related to TEQs will only be quantified and presented if residual concentrations exceed the target goal. If risks are quantified the uncertainty analysis will

explain (at a minimum) the portion of the risks that are related to non-detected congeners as well as the risks associated with the NDEP 1 ppb TEQ target goal.

1.4 ORGANIZATION OF THIS REPORT

The remainder of this document provides a detailed overview of the approaches that will be used to address potential human health risks associated with constituents that are present in soils or groundwater at the Site. It is organized into the following sections:

- Section 2 – Exposure Scenarios for the Site
- Section 3 – Data Evaluation
- Section 4 – Exposure Assessment
- Section 5 – Toxicity Assessment
- Section 6 – Risk Characterization
- Section 7 – References.

2 EXPOSURE SCENARIOS FOR THE SITE

The exposure scenarios to be considered in the HHRA are dependent upon the exposure pathways relevant to the Site and receptor populations that use the Site. As discussed previously, the exposure scenarios to be evaluated assume that the Site will remain as an industrial facility at closure.

Figure 2-1 summarizes the exposure pathways and receptor populations that will be considered in any HHRA to be conducted at the Site. Importantly, this summary figure is meant to provide a comprehensive listing of the suite of potential exposure pathways and receptors at the Site as a whole. Not all exposure pathways and receptor groups will necessarily be applicable for every exposure area. The HHRA conducted for each exposure area or decision unit will discuss the selection of exposure pathways and receptor groups evaluated and provide the rationale for exclusion of any exposure pathways and receptor groups.

2.1 EXPOSURE PATHWAYS

EPA (1989) has developed the concept of an exposure pathway to define the ways in which receptors might be exposed to constituents. Exposure pathways combine information on the source and transport of a constituent to a point of contact with a receptor and the exposure routes at that point. To be considered complete, an exposure pathway must contain the following elements (USEPA 1989):

1. A source and mechanism of release
2. A retention or transport medium
3. A point of potential contact with the affected medium
4. An exposure route at the contact point.

If any of these elements is missing, exposure will not occur, and the exposure pathway is not complete. Only complete exposure pathways are selected for evaluation in risk assessments.

2.1.1 Sources, Transport, and Contaminated Media

A conceptual site model (CSM) is a tool used to describe the source, release, distribution, and transport of chemical constituents to potential receptor populations. As such, a CSM provides detail related to development of exposure pathways for the Site. A draft site-wide CSM has been developed to address contamination associated with the Site. As part of the overall RAS process (NDEP 2008a), this site-wide CSM is being supplemented by the development of area-specific CSMs. These focused CSMs are being used to guide data collection and remedy design

at the various source and/or site assessment areas, and also will be useful for determining the potentially complete exposure pathways that are relevant at such areas.

The area-specific CSMs will be updated as additional information is collected during site investigation and the evaluation of remedial actions. The draft site-wide CSM (Hargis 2008), however, provides sufficient background information to support a conceptualization of the range of sources, release, fate and transport mechanisms, chemicals, and contaminated media that could be considered in the subject risk assessments.

Briefly, past manufacturing and waste management activities resulted in the release of chemicals to soil and/or groundwater at the former Montrose and Stauffer facilities. These chemicals can be transported in the environment by a variety of mechanisms and reach potential human receptors who contact contaminated media.

Montrose manufactured organic chemicals including chlorobenzene, PCBs, choral and 4,4'-dichlorobenzil and HCL at the Site from 1947 to 1983. Stauffer manufactured chlorine, sodium hydroxide, hydrochloric acid, and agricultural chemical products including pesticides and herbicides at the Site from 1954 to 1984. Olin currently operates chlor alkali production facilities at the Site and manufactures liquid chlorine, caustic soda, HCL, and bleach. Historical perspective regarding the impact of these operations on environmental contamination will be provided within the area-specific CSMs.

In cooperation with NDEP a list of site-related chemicals (SRCs) was agreed upon based on a review of historic Site operations, practices, and analytical data (NDEP 2006a,b). SRCs have been detected in surface soils, subsurface soils, and groundwater at the Site. SRCs for the Site include:

- Volatile organic chemicals (VOCs)
- Semivolatile organic chemicals (SVOCs)
- Pesticides and related by-products
- PCBs
- Dioxins/furans
- Organic acids
- Metals
- Asbestos.

In addition to this list, a number of tentatively identified compounds (TICs) have been selected as SRCs for the Site. Hargis (2008) provided a list of SRCs that have been identified for the former Montrose and Stauffer operations. This list is included as Appendix B of this RAWP.

NDEP additionally has requested that radionuclides be addressed in the risk assessment (NDEP 2008b,c), and this RAWP therefore also includes procedures for evaluating radionuclide exposures and associated risks.

For ease of discussion, the previously identified SRCs along with radionuclides are collectively referred to as SRCs in this RAWP. The RAWP provides details on how all SRCs would be evaluated in a HHRA; however, it may be the case that only a subset of this full suite of SRCs will be addressed for a particular exposure area. Site-specific conditions that warrant deviation from the list of SRCs presented in this RAWP will be discussed with NDEP prior to generating the HHRA.

SRCs in soil can be directly contacted by persons using the Site. In addition, constituents that are sorbed to soils can be transported to air via wind erosion or due to other physical disturbances of the soil (e.g., vehicle traffic, excavation). Once in air, the soil-sorbed SRCs (i.e., particulates) can be transported to potential receptors both on and off the Site. In addition, vapors that are present in subsurface soils can be transported to the surface and subsequently be dispersed and reach receptors either on- or offsite. Volatile constituents in groundwater also can reach potential receptors as the result of vapor transport through vadose zone soils to surface environments. (As discussed earlier, direct consumptive uses of groundwater will not be evaluated in the risk assessment.) Radioactive elements in soil can additionally release gamma, beta, and alpha radiation to which receptors can be externally exposed.

2.1.2 Exposure Routes

Human receptors can be exposed to SRCs in contaminated media by the following exposure routes:

- Ingestion of contaminated media (e.g., soils)
- Dermal contact (e.g., with soils)
- Inhalation (i.e., vapor or particulate phase constituents).

In addition, human receptors can be exposed externally to certain radionuclides without direct contact or inhalation. These exposures are termed "external exposures".

2.2 POTENTIAL RECEPTOR POPULATIONS

As discussed earlier, the HHRA will address potential exposures and risks assuming that the overall site will remain an industrial property after remedial actions have been implemented

(i.e., at closure). As such, the primary receptor populations that could be exposed to SRCs at the Site are site workers. Other potential onsite receptors, such as trespassers, will not be evaluated quantitatively. As stated by EPA (2002a) evaluation of exposures to members of the public under a non-residential land use scenario is not warranted for two reasons; first, public access is generally restricted at industrial sites, and second, while the public may have access to commercial sites, onsite workers have a much higher exposure potential because they spend substantially more time at the site.

Some offsite receptors may exist under certain conditions. For example, SRCs that are transported from the Site in air (either as particulate or vapors) also could reach offsite receptors. The principal offsite receptors are nearby workers and residents. Exposures to offsite workers will be lower than those to onsite workers (due to fewer exposure routes and lower exposure levels). Based on a comparison of key exposure factors for the onsite and offsite receptors, exposure to offsite residents is additionally anticipated to be lower than for workers onsite. The conclusion is exemplified by the 100-fold difference in the default particulate emission factor (PEF) from construction for onsite receptors versus offsite receptors as recommended by EPA (2002a). Potential exposures to onsite workers will be higher because this parameter has a much larger influence on the inhalation pathway evaluation compared to other exposure factors that may be higher for the offsite resident. Therefore offsite receptors will not be evaluated quantitatively; a discussion of the rationale for the decision will be included in the risk assessments.

The principal receptor populations that will be quantitatively evaluated in the HHRA and the routes by which they might be exposed are discussed below. The particular receptors and exposure pathways to be evaluated will be discussed in the HHRA conducted for the relevant source/site assessment area.

2.2.1 Indoor Worker

The indoor worker is defined as a long-term, full-time employee who spends most of the day working indoors. Workers may be exposed to outdoor dusts that have infiltrated the building, outdoor soils that have been tracked in, and to contaminants present in indoor air as the result of vapor intrusion.

Potentially complete exposure pathways for the indoor worker are:

- Inhalation of indoor dust
- Inhalation of vapors and radon² in indoor air released from soil and groundwater

²An NDEP approved risk assessment methodology for radon is currently not available; risks that may occur via exposure to radon will be addressed in a future guidance document from NDEP; and will not be quantified in the risk assessments for the Sites completed prior to that guidance.

- Incidental ingestion of surface soil that has been tracked indoors
- External radiation exposure from surface soil that is outdoors, and surface soil that has been tracked indoors (radionuclides only).

Surface soils defined as the top 6 in. of the soil column, are used to define the potential concentrations of SRCs in dust/soils that reach indoors. The vapor inhalation pathway is based on volatile concentrations in the full soil column (i.e., from surface down to groundwater) and in the groundwater. External radiation exposure to radionuclides that are present in outdoor soil is limited to materials within the top 6 in. of soil; radionuclides found below this level are shielded by the top layer of soil and do not contribute to external radiation exposure (USEPA 2000).

Workers can additionally be exposed to radiation via physical immersion in airborne particulates containing radionuclides. This is a complete exposure pathway (as noted in Figure 2-1) but consistent with EPA guidance for developing preliminary remediation goals (PRGs) for radionuclides (USEPA 2009b), contributes negligibly to overall exposures and will not be evaluated in the risk assessments conducted for the Site.

2.2.2 Outdoor Worker

The outdoor worker is defined as a long-term, full-time employee who spends most of the day working outdoors. This receptor is assumed to participate in relatively low-intensity activities such as building maintenance, unloading and loading materials and supplies, or occasional digging. Soil exposure for this receptor group is limited to surface soils. Inhalation of vapors as well as dust generated by wind erosion and construction activities also may occur.

Potentially complete exposure pathways for the outdoor worker are:

- Inhalation of outdoor dust
- Inhalation of vapors and radon in ambient air released from soil and groundwater³
- Incidental ingestion of surface soil
- External radiation exposure from surface soil (radionuclides only)
- Dermal contact with soil.

Again, external radiation exposure via immersion is also a complete pathway, but contributes negligibly to exposure, and will not be evaluated.

³ Pathway will be evaluated quantitatively only if needed based on results of indoor air evaluation.

2.2.3 Construction Worker

Construction workers are expected to participate in shorter term, intermittent work at the Site. Work completed by this group might include demolition or construction activities completed as part of developing infrastructure for future onsite activities. The activities for this receptor may involve substantial onsite exposures to surface and subsurface soils. Workers are assumed to have potential for direct contact with soil from 0 to 10 ft bgs. Inhalation of dust and vapors also may occur.

The construction workers may contact exposure media via the following exposure pathways:

- Inhalation of outdoor dust
- Inhalation of vapors and radon in ambient air released from soil and groundwater⁴
- Incidental ingestion of surface and subsurface soil
- External radiation exposure from surface and subsurface soil (radionuclides only)
- Dermal contact with surface and subsurface soil.

Given that subsurface soils are hypothesized to be exposed during construction activities, radionuclides in subsurface soil could be a source for external radiation exposures for this receptor group. External radiation exposure via immersion is also a complete, but negligible, exposure pathway for this receptor group.

⁴ Pathway will be evaluated based on soil gas data and supplemented by perimeter air monitoring if appropriate.

3 DATA EVALUATION

Analytical data collected as part of past and future site investigations will be the source of the SRC data evaluated in the risk assessments. This section describes the types of data that may be used for the risk assessments as well as the proposed procedures to 1) evaluate and select data for use in each risk assessment, 2) process analytical sample results to support use in each risk assessment, and 3) select specific SRCs for quantitative evaluation in each risk assessment.

3.1 DATA TYPES

The following types of data may be evaluated in the risk assessments, as relevant and available:

- Soil data – all SRCs
- Groundwater data – volatile SRCs
- Soil vapor data - volatile SRCs.

Soil data would be used to evaluate direct contact exposures (i.e., ingestion, dermal contact, and inhalation of dust) and potential impacts to groundwater from leaching. Soil vapor data would be used to evaluate inhalation exposures to volatile chemicals that could migrate into indoor or ambient air. Groundwater and soil data could be used as a secondary line of evidence in support of the evaluation of the soil vapor data. The groundwater data that would be used in such an application modeling exercise would be that collected from the alluvial aquifer (i.e. Shallow Zone) and fine-grained Upper Muddy Creek Formation. This groundwater is closest to the surface and therefore best represents the potential source of groundwater chemicals available for vapor transport to the surface. As mentioned earlier, direct consumptive use of groundwater will not be evaluated in the risk assessment. The quality of all groundwater (shallow and deep) will, however, be evaluated separately as part of the remedial alternatives assessment.

3.2 DATA REVIEW AND SELECTION

Available analytical data will be reviewed to determine its suitability for use in each risk assessment. EPA guidance for data usability in risk assessment (USEPA 1992a,b) and NDEP procedures outlined in guidance issued for assessing data usability for environmental investigations at the BMI Complex and Common Areas (NDEP 2008d) will guide the data assessments. Data usability evaluations will be completed prior to the risk assessments, and will be documented in reports following specifications outlined by NDEP (2008d). The risk assessments will include a summary of the findings of the data usability evaluation. The implications of issues raised in the usability evaluation will be discussed in the uncertainty section of the risk assessment.

3.3 DATA PROCESSING

Following the data usability evaluation, data deemed of sufficient quality to support the risk assessment will be compiled in a database to support the exposure and risk calculations. Relevant sampling data for the Site may include detected and non-detected values, duplicate samples, and split samples. The treatment of these different data types will follow EPA (1989, 1992a,b) and NDEP guidance (2008d,e,f) and is discussed below.

3.3.1 Detected Analytes

Laboratory results can be broadly classified as detects or non-detects. Detected results reflect cases in which a measurable quantity of a constituent was determined and reported by the laboratory. Detected results may have a qualifier assigned by the laboratory, or during the data validation process. As part of the data usability evaluation, all qualifiers assigned to detected data will be reviewed and treated in accordance with EPA guidance (USEPA 1989, 1992a,b). Detected data that are deemed appropriate for use in the risk assessment by the data usability evaluation will be used at the full reported value.

3.3.2 Non-Detects

Cases where analytical parameters are not detected above some measurement threshold are defined as non-detections. Non-detected results are qualified as such by the laboratory and an associated quantitation limit is provided. Non-detected values can also carry other qualifiers assigned during the analysis or validation process. As part of the data usability evaluation, the qualifiers assigned to all non-detected values will be reviewed using EPA guidance (USEPA 1989, 1992a,b). All non-detected results that are considered appropriate for use in the risk assessment will be included in the database.

For non-detected results the sample quantitation limit (SQL)⁵ will be reported for all analytes with the exceptions of radionuclides and asbestos. For radionuclides the value reported by the laboratory, which may be less than the minimum detectable activity (MDA)⁶, will be reported. For asbestos, the reported analytical sensitivity for the non-detected sample will be presented.

Summary statistics characterizing both detected concentrations, and the quantitation limits specified above for non-detected results, will be provided in a form consistent with NDEP guidance (NDEP 2008e).

⁵ SQLs are sample-specific detection limits. They are usually an adjustment from the method detection limit (MDL) and reflect sample-specific actions, such as dilution or use of smaller aliquot sizes, and take into account sample characteristics, sample preparation, and analytical adjustments.

⁶ The MDA is the lowest level of activity in a given sample that is statistically distinguishable from a sample with no activity, at the 2-sigma confidence interval. MDAs for radionuclide analysis take into account sample volume, chemical recovery, instrument detection efficiency and background, and sample counting duration.

3.3.3 Duplicate and Split Samples

Duplicate samples and split samples are commonly included as part of data collection efforts for assessing environmental contamination. A field duplicate is a distinct sample collected from the same point in time and space as the first sample, or as near to the same time and place as possible. A field split sample is derived from a sample homogenized in the field; the homogenized sample is split into two samples, each of which is analyzed separately. The second sample is assigned the label of field split and is considered the quality control (QC) sample (NDEP 2008f).

Following NDEP recommendations (NDEP 2008f) the treatment of duplicate samples will depend on the variance of the QC sample and the site sample results. Variance is not a factor for consideration in the treatment of split samples. For the treatment of duplicates, sample results will be summarized to determine whether the variance between duplicate samples and site samples is similar. If appropriate to the data (e.g., sufficient sample size), statistical tests will be used to evaluate if variance in the duplicate samples are similar or different from the site samples. Following the assessment of variance, duplicate, and split samples will be treated for use in the risk assessment as follows:

Duplicates with variance similar to site samples –

- Samples will be treated independently. All results will be carried forward in the quantitative characterization of Site SRCs.

All splits, and duplicates with a variance that differs from site samples –

- The result of the first sample will be carried forth in the quantitative characterization of Site SRCs. The second QC sample will not be carried forward in a quantitative manner.

Uncertainties associated with the choice of the first sample will be tracked in the risk assessment and discussed in the uncertainty section as relevant.

3.4 SELECTION OF COPCS FOR EVALUATION IN THE RISK ASSESSMENT

More than 300 chemicals and analytical parameters have been identified as SRCs for the Site. To focus the risk assessment on those SRCs that are most important to defining potential human health risks at any given site assessment area, a series of screening steps will be applied to the data to select the particular SRCs to be considered in the risk evaluation. The SRCs selected for evaluation in the risk assessments are termed COPCs.

For purposes of the risk assessments, all analytes have been grouped initially by chemical class. The SRC group classifications presented in Hargis (2008), and presented in Appendix B, will be used to characterize Site SRCs with the addition of radionuclides.

The following SRCs/SRC Groups will not be selected as COPCs for the risk assessments.

- **General and indicator chemicals.** This group of general analytical parameters (e.g., alkalinity, chloride, pH, sodium, sulfate) was used at the Site primarily to characterize general site conditions (e.g., total inorganic and total organic carbon) or as indicators of the potential presence of other SRCs (e.g., pH as an indicator for acid SRCs, ions for several of the salts). The potential toxicity and risks from this group of SRCs will not be evaluated.
- **Inorganics.** This SRC group as defined in Hargis (2008) is comprised of fluoride, iodide, nitrate, and total carbon, and has been used at the Site primarily to understand general chemical conditions. These SRCs will be used to understand conditions at the Site but will not be separately evaluated for toxicity or risk.
- **Total petroleum hydrocarbons (TPH).** This is another type of a general indicator SRC group (in this case, for petroleum products). Because toxicity is dependent upon the individual constituents that comprise the TPH mixture, the potential toxicity or risks associated with TPH exposure will be evaluated for the constituent SRCs as reported in the database. The potential toxicity and risks from TPH as a whole will not be evaluated separately.
- **Tentatively Identified Compounds (TICs).** TICs will not be selected as COPCs for quantitative evaluation in the risk assessment because of the uncertainty associated with the identity of these compounds. These data will be evaluated qualitatively; however, and the potential risk implications discussed in the risk assessment.

The remaining SRCs will be further evaluated for selection as COPCs for inclusion in the risk assessments. The primary criteria to be used to select COPCs are a comparison to naturally occurring (background) levels and a comparison to risk-based levels. These steps are discussed in more detail below. For some site assessment areas, a frequency of detection screen may additionally be used to select COPCs, if SRCs are detected infrequently in any given area. This screen would not be used in any site assessment area without prior approval by NDEP, however.

3.4.1 Background Comparison

NDEP and EPA guidance allows for the elimination of constituents from further evaluation if detected levels are not elevated above naturally occurring levels (NDEP 2009b; USEPA 1989). Because metals and radionuclides occur naturally in the environment, concentrations of these

constituents will be compared to background concentrations. Metals and radionuclides that are present at the Site at concentrations that are similar to regional background concentrations will not be selected as COPCs.

The background dataset to be used for the background/onsite comparisons will be selected as part of the data usability evaluation. This selection will consider representativeness, comparability to onsite data, and statistical power/sample size of available background datasets. The selection and justification of the background data to be used for onsite comparisons will be included in the data usability evaluations and in the risk assessment reports.

As recommended by NDEP in past communications with the Companies, comparison of onsite and background data will be conducted via hypothesis testing using EnviroGiSdT Software developed by Neptune and Company, Inc. (Neptune 2008a). As outlined in the software's users' manual (Neptune 2008b), four two-sample hypothesis tests are conducted as part of background comparisons: the t-test, the Wilcoxon Rank Sum test using the Gehan ranking scheme, the Quantile test, and the Slippage test. Because considering the results of four tests in combination increases the overall false rejection rate for the entire procedure, an adjusted significance level aimed at producing an overall false rejection rate of 0.05 will be adopted for each test. Following NDEP guidance (2009c) a default adjusted rate of 0.025 will be used unless specific limitations on sample size or unusual data characteristics warrant that more specific values be developed. Such values would be developed following NDEP guidance (NDEP 2009c). Specific values used will be included in the HHRA documents. Results from statistical tests, consideration of their robustness and limitations, and graphical displays of the data will be used to determine whether onsite concentrations of metals and radionuclides exceed background concentrations.

In addition to direct comparisons with background data as described above, in cases in which a sufficiently robust data set is available, radionuclide data will additionally be evaluated by analysis of secular equilibrium (SE), following guidance prepared by NDEP (2009b). The presence or absence of SE for onsite data can be used to characterize the source of radionuclides. SE exists when the quantity of a radioactive isotope remains constant because its production rate is equal to its decay rate; under natural background conditions approximate SE is expected. In the case that onsite radionuclide data do not exhibit SE there is an indication of radionuclide-specific contamination (NDEP 2009d).

Natural chemical and physical processes may cause some deviations from SE, and only approximate or quasi-SE can be expected even under the best field and ideal testing conditions. In order to accommodate small differences, equivalence testing, which allows some flexibility in terms of the statistical hypothesis tested, will be employed. The equivalence testing approach will follow the protocols set forth in NDEP guidance (2009d). Standard background

comparisons, described above, and when employed the analysis of SE will be considered together in determining whether onsite radionuclides differ from background.

3.4.2 Risk-Based Screening

Soil SRCs that remain after the above screening step will be further screened by comparing to risk-based concentrations. No risk-based screening will be conducted for soil vapor data; instead, all volatile SRCs that are detected will be evaluated.

The risk-based concentrations to be used in the screening for SRCs in soil are BCLs developed by NDEP for chemicals and radionuclides (NDEP 2009a,e), BCLs for chemicals are based on direct contact exposure pathways (ingestion, inhalation, and dermal absorption). Radionuclide BCLs are based on ingestion, inhalation, and external radiation. The BCLs correspond to a target excess cancer risk of one in one million (1×10^{-6}), or a noncancer hazard quotient (HQ) of 1. BCLs developed for commercial/industrial settings will be used.

Soil SRCs that remain after the comparison to background levels will be evaluated by comparing the maximum detected concentration to one-tenth the value of the BCL for industrial/commercial land use scenarios (NDEP 2009a,e). Per NDEP guidance, the exceptions to this are lead, which will be compared directly to the commercial/industrial BCL of 800 mg/kg (NDEP 2008g), and titanium, which might be compared to a concentration limit that is lower than one-tenth of the BCL if it is present in substantial amounts in a form other than titanium metal or titanium oxide (NDEP 2008h).

Any organic SRC passing the initial screening steps that has a maximum detected concentration that exceeds the risk-based screening evaluation discussed above will be selected as a COPC for risk assessment. Similarly, any metal SRC or radionuclide that exceeds the risk-based screening and exceeds regional background levels and/or shows deviations from SE, will be selected as a COPC.

The detection of amphibole or chrysotile fibers will be used to screen asbestos for the quantitative risk evaluation.

4 EXPOSURE ASSESSMENT

The magnitude of exposure for any given receptor is a function of the amount of the constituent in the exposure medium and the frequency, intensity, and duration of contact with that medium. This section presents an overview of the equations and default assumptions that will be used to calculate potential exposures as part of the risk assessments to be conducted at the Site. In cases in which site-specific information on receptor populations or exposure patterns is available site-specific assumptions will be incorporated into the risk assessments in consultation with NDEP.

4.1 GENERAL APPROACHES TO EXPOSURE CALCULATIONS

For non-radiological constituents, oral and dermal exposures are expressed in terms of intake (i.e., mg chemical per kg body weight [BW] per day – mg/kg-day), whereas inhalation exposure is expressed in terms of an exposure concentration (EC) in air (i.e., $\mu\text{g}/\text{m}^3$, f/cm^3 [fibers/ cm^3]). These different expressions of exposure are used to match the toxicity criteria that are available to calculate risks for each type of exposure. For radionuclides, exposure is expressed as total dose in terms of picocuries (pCi).

The general approaches for quantifying exposures for chemicals and radionuclides are discussed below. Appendix C provides the pathway-specific equations and default parameter values. The approaches for quantifying exposures to asbestos are unique and discussed separately later in this section.

4.1.1 Chemical and Asbestos Exposure

Exposure to chemicals and asbestos⁷ for each scenario will be calculated using site-specific concentrations of constituents and receptor- and scenario-specific exposure assumptions.

The following equation is a general form of the equation used to estimate intake for oral and dermal exposures:

$$\text{Intake (mg/kg} \cdot \text{day)} = \frac{\text{CR} \times \text{C} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}} \quad \text{Eq. 4-1 (USEPA 1989)}$$

where,

CR = contact rate (e.g., mg/day)
C = contaminant exposure point concentration (e.g., mg/kg)

⁷ In line with NDEP guidance (2009f) only inhalation of asbestos following suspension of fibers from soil will be evaluated.

CF	=	conversion factor (e.g., 10 ⁻⁶ kg/mg)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
BW	=	body weight (kg)
AT ⁸	=	averaging time (days).

Intake will be expressed in various forms, depending on the risks that it will be used to assess. Average daily dose (ADD) and lifetime average daily dose (LADD) will be calculated and used as measures of exposure from oral and dermal routes, for characterizing noncarcinogenic and carcinogenic effects, respectively.

The EC is a function of a constituent's concentration in air measured at the exposure point and scenario-specific parameters, such as ED and EF. The following equation is a general form of the equation used to estimate the EC:

$$EC (\mu\text{g}/\text{m}^3, \text{f}/\text{cm}^3) = \frac{C_{\text{air}} \times ET \times EF \times ED}{AT} \quad \text{Eq. 4-2 (adapted from USEPA 2009a; NDEP 2009f)}$$

where,

C _{air}	=	contaminant concentration in air (μg/m ³ , f/cm ³)
ET	=	exposure time (hours/day)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
AT ⁸	=	averaging time (hours).

4.1.2 Radionuclide Exposure

Unlike chemicals and asbestos, radionuclide exposure is typically expressed in units of activity per unit of the exposure medium, rather than mass per unit. Exposure to radionuclides may result from internal and external exposure pathways.

Internal exposure is expressed for completed pathways using the following equation:

$$Dose (pCi) = C \times CR \times EF \times ED \quad \text{Eq. 4-3 (adapted from USEPA 2009b)}$$

where Dose is the dose due to internal exposure, and the remainder of the variables are the same as Equation 4-1 above, except that "C" is the concentration term for soil or air expressed in

⁸ When evaluating cancer risk, the averaging time (AT) is equal to a lifetime of 70 years. When evaluating noncancer hazard, the AT is equal to the total ED.

units of pCi/g, or pCi/m³, respectively, and "CR" is the contact rate expressed in the relevant units (i.e., g/day, m³/day) for that medium. The body mass and averaging time (AT) exposure factors are not relevant for radionuclides.

For some radionuclides, exposure via certain internal pathways (e.g., oral, dermal, or inhalation) may be insignificant (USEPA 2000). For instance, as reflected by their small dermal absorption and dermal permeability constants, dermal absorption of radionuclides is not an important pathway (USEPA 2000). The inhalation of particulates from dust represents significant exposure for only a few radionuclides (USEPA 2000). Quantitative exposure assessments will only be completed for significant pathways. The selection of pathways for quantitative evaluation will depend upon the radionuclide constituents that are present in and near each site assessment area and will be discussed in the individual risk assessment reports.

The external dose for radionuclide exposure will be calculated using the following equation:

$$\text{Dose (pCi - yr/g)} = C_{\text{soil}} \times [EF/CF_{\text{DY}}] \times ED \times ACF \times [ET_{\text{fo}} + (ET_{\text{fi}} \times GSF)] \quad \text{Eq. 4-4}$$

(adapted from
USEPA 2009b)

where,

C_{soil}	=	exposure concentration term for soil (pCi/g)
EF	=	exposure frequency (days/year)
CF_{DY}	=	conversion factor (days/year)
ED	=	exposure duration (years)
ACF	=	area correction factor (unitless)
ET_{fo}	=	fraction of time spent outdoors (unitless)
ET_{fi}	=	fraction of time spent indoors (unitless)
GSF	=	gamma shielding factor (unitless).

The EF and ED are the same as described above for calculating internal exposures to non-radiological and radiological constituents. As described in the context of internal exposures to radioactive constituents above, "C" is the concentration term for soil expressed in units of pCi/g.

The EPA model for external radiation assumes that an individual is continually exposed to a non-depleting radiological source that is effectively an infinite slab. The concept of an infinite slab means that the thickness of the contaminated zone and its aerial extent are so large that it behaves as if it were infinite in its physical dimensions. Source areas contaminated to a depth greater than 15 cm with an aerial extent greater than 1,000 m² will create a radiation field comparable to an infinite slab (USEPA 2000). The area correction factor (ACF) adjusts for smaller source areas. EPA has derived ACFs for various source area sizes, ranging from 10 to

10,000 m² (USEPA 2009b). These will be used to assess radiological risks at various site assessment areas at the Site.

The gamma shielding factor (GSF) is a factor that accounts for the shielding effect provided by buildings during times of indoor occupancy or by other site features. The fraction of time spent exposed in outdoor and indoor environments is described by ET_{fo} and ET_{fi} , respectively.

4.1.3 Range of Exposure Assumptions

The variables/exposure factors shown in the exposure algorithms above vary depending on the receptor population being evaluated. Each receptor population will be characterized by a number of assumptions regarding the frequency of contact with potentially contaminated media, duration of exposure, and other parameters unique to the receptor population.

EPA (1992c) guidance for Superfund sites discusses two types of exposure estimates that may be calculated in a HHRA; the reasonable maximum exposure (RME), and the central tendency exposure (CTE). The RME is defined as the highest exposure that could reasonably be expected to occur for a given exposure pathway at a site. The RME is intended to account for both uncertainty of the contaminant concentration and variability in exposure parameters. The CTE is designed to reflect an average estimate of exposure. RME estimates will be calculated for the risk assessments. The single exception is for lead. The ALM is sensitive to upper end values, and specifies the use of central tendency soil lead concentrations (USEPA 2003a); therefore, in the case that lead is brought forth as a COPC in the risk assessment, only CTE estimates will be calculated for the chemical. CTE estimates for other SRCs may additionally be calculated and presented in the uncertainty analysis as a means to provide context to the RME evaluation.

The specific equation and assumptions used to estimate exposure varies, depending on the exposure route being evaluated. Appendix C presents a complete set of exposure equations along with the specific exposure assumptions that will be used for contact rate; ET, EF, and ED; BW; and AT for each pathway and receptor group. It additionally presents exposure factors specific to radionuclide exposures including ET_{fo} , ET_{fi} , and GSF. In cases in which site-specific information on receptor populations or exposure patterns is available, site-specific exposure factors will be incorporated into the risk assessments.

General assumptions that are applicable to exposure estimates are discussed in Section 4.2 below. In addition to exposure assumptions, COPC concentration in the exposure medium at the point of contact are required for evaluating risks. Section 4.3 describes the approaches used to estimate exposure point concentrations (EPCs).

4.2 GENERAL INTAKE ASSUMPTIONS

Exposure assumptions for ED, EF, ET, BW, and AT are discussed below. EPA guidance was used as the basis of these values, if available.

4.2.1 Exposure Duration

The ED is the length of time during which someone may be exposed to a particular medium via a specific exposure pathway. The ED varies depending on the population being evaluated. Both chronic and subchronic exposures will be assessed at the Site, depending upon the receptors evaluated. EPA (2009a) defines chronic exposures as repeated exposures that occur over 7 years⁹ or more, and subchronic exposures as repeated exposures that occur over a period greater than a month and less than 7 years.

For a typical indoor or outdoor occupational worker, chronic exposures are evaluated. EPA (2002a) recommends a RME ED of 25 years. This value is based on U.S. Census data and represents an upper bound estimate for the length of time a person works at the same location. The average, or CTE, value for occupational ED is assumed to be 7 years, which is the median occupational tenure of the working population (USEPA 1997a).

Construction workers are expected to work on limited-term projects, such as building construction, and are assessed for subchronic exposures. If multiple construction projects occur on the Site, it is assumed that different workers will participate in each project. EPA recommends an ED of 1 year for construction workers (USEPA 2002a). For this risk assessment, based on best professional judgment, a value of 6 months is proposed as the CTE value. Site-specific values will be substituted for these defaults when available and in consultation with NDEP.

4.2.2 Exposure Frequency

EF describes how many days someone may have contact with the exposure media of interest in a typical 1-year period.

EPA recommends a RME EF of 250 days/year for indoor workers and 225 days/year for outdoor workers (USEPA 2002a). These values will be adopted for the default RME and CTE cases for these receptor groups. EPA recommends an EF of 250 days/year for construction workers (USEPA 2002a). This value will be used as the default RME and CTE value. Site-specific values will be used in lieu of defaults when available.

⁹ Seven years is one-tenth of an EPA-assumed standard lifetime of 70 years.

4.2.3 Exposure Time

The ET is the amount of time each day which someone may be exposed to a particular medium via a specific exposure pathway.

ET is assumed to be 8 hours/day for the indoor worker, outdoor worker, and construction worker. The entire 8 hour period is assumed to be spent indoors for the indoor worker; while the entire 8 hour period is assumed to be spent outdoors for the outdoor and construction workers.

4.2.4 Body Weight

A value of 70 kg (154 lbs) represents the BW for all adult receptors, based on average male and female adult BWs (USEPA 1991). This value will be used for all RME and CTE worker scenarios. This parameter is not included in dose estimation for radionuclides (USEPA 1989).

4.2.5 Averaging Time

The AT is the period over which an exposure is averaged. The ATs for evaluating carcinogenic and noncarcinogenic effects are different, and are expressed in different units dependent on the exposure route being evaluated. For evaluating carcinogenic effects, chemical intakes are averaged over a 70-year lifetime (25,550 days; 613,200 hours) to be consistent with the method by which cancer slope factors (CSF) and inhalation unit risks (IURs) are derived.

When evaluating noncarcinogenic effects, chemical intakes are averaged over the ED (USEPA 1989). Therefore, for noncarcinogenic effects, the ED is converted to days or hours and is used as the AT_{nc}. For example, the RME AT for the outdoor occupational and indoor worker is 25 years, or 9,125 days, or 219,000 hours. This parameter is not included in dose estimation for radionuclides (USEPA 1989).

4.3 EXPOSURE POINT CONCENTRATIONS

EPCs will be estimated using measured concentrations of chemicals and radionuclides in environmental media alone or in combination with fate and transport models. Methods for deriving EPCs in soil, airborne particulates, and ambient and indoor air vapors are described below.

4.3.1 Exposure Point Concentrations in Soil

Soil EPCs will be calculated to estimate direct contact exposure for onsite workers. The soil EPCs could also be used as inputs to emission models used for deriving airborne concentrations of SRCs released into the atmosphere as particles.

EPCs for soil in an exposure area will be derived using data results from soil samples taken within the source area. Representative EPCs will be based on the potential exposure depth interval for each receptor. For receptors exposed to surface soil (e.g., for indoor workers, outdoor workers), two EPCs will be calculated. For the first, data from the top 6 in. of soil will be used. For the second, a vertical average from the surface to 10 ft bgs will be used. The second EPC assumes a redevelopment scenario in which soil from the surface to 10 ft bgs is reworked and brought to the surface (i.e., 0-6 in. bgs). For receptors exposed to deeper soils (e.g., construction workers) data from the surface to 10 ft bgs will be used.

When developing the soil EPCs, the exposure areas will be combined to the greatest extent possible to make the largest decision units that can be justified for the source/site assessment area. Accordingly the modality of the data will be evaluated and areas of localized elevated concentrations will be evaluated as a separate decision unit, if necessary.

To estimate exposures that are representative of upper end exposures, EPA (1992c) recommends using the 95th upper confidence limit (UCL) of the arithmetic mean concentration. As recommended in past communications with NDEP, 95% UCLs will be estimated using EnviroGiSdT Software (Neptune 2008a). EnviroGiSdT provides three methods for computing the UCL; the Student's t- UCL and two bootstrap UCL methods. For each COPC the sample size, frequency of detection, and data distribution will be evaluated in order to select the appropriate method for computing a UCL. The EnviroGiSdT Software's default setting uses one-half the SQL, or reported value for radionuclides for non-detects when computing the 95% UCL. If the substitution of one-half of the SQL, or reported result for radionuclides for non-detects appears to be driving the risks, alternative substitution methods for non-detects may be explored within the uncertainty evaluation.

Further refinement of the EPCs will be considered based on the HHRA results estimated using the 95% UCL analysis. For example, more refined EPCs can be derived using area-weighted or spatial statistics using Thiessen polygons. Any refinement to the EPC calculation method will be discussed with NDEP prior to implementation in the HHRA.

In the cases that lead is brought forth as a COPC in the risk assessment, the arithmetic mean concentration of lead in soil will be adopted as the EPC for estimating risk. The ALM, which will be used to characterize risks from exposure to lead, is sensitive to upper end values, and specifies the use of central tendency soil lead concentrations (USEPA 2003a).

Results of statistical analyses conducted to characterize the distribution of the data and the recommended UCL will be provided in the risk assessment.

4.3.2 Airborne Particulates

Airborne particulate concentrations will be calculated for dust emissions sources within a given source area. For the purpose of this RAWP, airborne particulates will include nonvolatile chemicals, radionuclides, and asbestos. The emissions and dispersion modeling described in this section will be applied to all airborne particulates evaluated in the risk assessments. However, there are unique analytical data handling procedures used to develop the asbestos concentration to be used in the emissions models. These unique asbestos procedures are detailed at the end of this section on airborne particulates.

4.3.2.1 Emissions and Dispersion Modeling for Chemicals and Radionuclides

There are two primary sources of dust emissions at the Site: wind erosion, and soil disturbances associated with construction activities. For source areas where construction scenarios are not assumed to occur, wind erosion emissions are the only concern. For the purpose of this discussion such non-construction scenarios are defined as the non-construction emissions. The airborne particulate concentrations will be calculated separately for dust emissions from wind erosion and from construction-related activities. Dust emissions from construction activities are assumed to occur for a limited period (i.e., no more than 1 year) whereas emissions from wind erosion can occur throughout the assumed exposure period for a receptor. If construction activities are evaluated for a source area, a time-weighted airborne particulate concentration will be calculated for all receptors, except the construction worker, to reflect the combined emissions from short-term construction activities and long-term wind erosion. For construction workers, the airborne particulate concentration for the risk assessment will be based only on the dust emissions during construction.

For most SRCs, the incidental ingestion and dermal absorption exposure pathways to be quantified in the site risk assessment will result in higher potential health risks than the inhalation pathways. Therefore a tiered modeling approach that progresses from a simplified and upper-bound assessment to a refined and more accurate estimate of potential health risks is proposed for evaluating inhalation exposures related to airborne particulates released from the source area being addressed. The first tier for estimating airborne particulate concentrations will be based on the simplified site-specific PEF modeling provided in EPA's *Soil Screening Guidance* (USEPA 2002a). If the inhalation pathway risks based on the simple site-specific method drive the overall risks to the site assessment area, then more refined and less conservative tiers will be used. The proposed methodology for the more refined analysis will be provided to NDEP for approval as an interim deliverable for the relevant site assessment area. Procedures for estimating non-construction dust emissions and construction emissions for the first tier evaluation are described briefly below. Appendix D provides more complete details on the dust emission modeling proposed for the first tier.

Non-Construction Dust Emissions

The non-construction dust emission scenario for a site assessment area will be exposure to wind erosion. A PEF equation for wind erosion provided by EPA (2002a) will be used for estimating the chemical concentration in air associated with the surface soil concentration of the source. The soil concentrations for COPCs will be based on the 95% UCL for soils 0-6 in. bgs as described in Section 4.3.1. The area used in the PEF equation will reflect the size of the source area. In cases where modality is observed in the soil data, more than one wind erosion PEF may be required to address the entire source area. The fraction of vegetative cover will consider the land cover of the area being assessed; the value will be specified in the risk assessment for the given source area.

An integral part of the PEF equation is the dispersion factor which provides an estimate of the dilution that occurs during transport from the emission source to the point of exposure once dust is released into the atmosphere. The dispersion factor is linked to the PEF to calculate the airborne particulate concentration. The EPA (2002a) dispersion factor used in the wind erosion PEF equation assumes that the receptor is located either at the edge, or in the center, of the emission source. For the first tier screening analysis, all workers are assumed to be located at the center of the source area. The PEF model generates estimates of the ambient air concentrations. For receptors that spend all or some of their day indoors (i.e., indoor workers), an attenuation factor will be used to scale the ambient air dust concentrations to indoor air dust concentrations.

A sensitivity analysis will be conducted for the PEF model inputs to evaluate their impact on uncertainty in the risk estimates. Complete listings of the PEF equations and input values for the non-construction emissions are presented in Appendix D.

Construction Dust Emissions

If construction activities are anticipated to occur within a source area then relevant PEF equations from EPA (2002a) will be applied to estimate associated airborne concentrations for construction workers. Onsite workers not involved in construction activities are assumed to enter the exposure areas post-construction. EPA (2002a) has identified vehicle traffic as the most significant contributor to fugitive dust emissions during construction activities. Dust emissions for construction activities will be based in part on assumed vehicle traffic over unpaved surface soil. In addition, dust emissions from various construction activities (i.e., excavation, dozing, grading, and tilling) will also be calculated. The total outdoor ambient air dust concentration for construction activities will be estimated based on the combined contributions from wind erosion, vehicle traffic, and construction activities.

The soil disturbance area to be modeled in the PEF construction equations will be dependent on the size and characteristics of the assumed construction activities for the HHRA. A primary characteristic is the soil concentrations used in this modeling, which will be based on the 95%

UCL for the source area soil 0-10 ft bgs. As discussed in Section 4.3.1, the 95% UCL calculation will include an evaluation of modality to identify the largest justifiable decision unit within the source area. The soil disturbance area used in the construction activity PEFs will be reflective of the exposure area represented by the soil 95% UCL value.

The air dispersion factor used in the construction activity PEFs depends upon the location of the receptor relative to the dust emission sources. Construction workers will be assumed to be located at the center of the emission source for the duration of the exposure.

A sensitivity analysis will be conducted for the PEF model inputs to evaluate their impact on uncertainty in the risk estimates. A complete listing of the PEF equations and input values for the construction dust emissions is presented in Appendix D.

4.3.2.2 Asbestos Airborne Exposure Point Concentrations

Asbestos concentrations in site soils have been characterized using an elutriator method that reports the number of asbestos structures detected per mass of respirable dust (Berman and Kolk 2000). The intent of this method is to provide analytical asbestos measurements that can be directly combined with standard dust emission and dispersion models to predict airborne asbestos concentrations associated with soil disturbances that would release respirable dust. This asbestos measurement methodology was employed as part of an NDEP-approved sampling and analysis plan for site soils (PES 2006). This methodology has also been employed for NDEP approved risk assessment activities at adjacent sites (ERM 2007) and is specified for use in the recent NDEP technical guidance for calculating asbestos risks from soils (NDEP 2009f).

The asbestos sampling results from the elutriator method are reported as structures per gram of respirable dust¹⁰. The emissions and dispersion modeling discussed in Section 4.3.2.1 describes the approach for estimating the respirable dust concentration in air resulting from the various soils disturbing activities anticipated at the Site and is consistent with the NDEP (2009f) guidance for calculating asbestos risks from soils. The product of the airborne respirable dust concentration and the asbestos elutriator results yields an estimate of the airborne asbestos concentration that can be used in calculating potential human health risks as described in Sections 5 and 6 of this RAWP. Following NDEP guidance (2009f) EPCs will be calculated by multiplying the UCL for the number of fibers observed by the analytical sensitivity (in the case that only a single sample is available) or pooled analytical sensitivity (in the case that multiple samples are available). The uncertainty associated with assumptions used for deriving the EPC will be addressed by discussing the variability in the sample results and the risk implications of using other inputs to characterize asbestos concentrations in soil.

¹⁰ Concentrations are based on fibers observed in a sample multiplied by the analytical sensitivity of the measurement. In the case that more than one asbestos sample is collected than the pooled analytical sensitivity is used.

4.3.3 Vapor Assessment

Inhalation exposures for vapors released from soils and groundwater will be evaluated for all worker populations. Several types of data applicable to vapor assessment may be available (e.g., surface emission isolation flux chamber data, soil gas data, soil data, and groundwater data); however, only soil gas data is being considered for the quantitative risk assessments¹¹. The remainder of this section describes generally how soil gas data will be used to develop EPCs for the risk assessments. Details of the modeling approach, including model input parameter values, will be developed in consultation with NDEP for approval prior to completion of the HHRA.

Soil gas data used for the risk assessment will be obtained via NDEP approved methodology and standard operating procedures (SOPs). There are a wide variety of soil gas sampling methods available; however, they can be divided into either active or passive methods. For the purpose of this RAWP it is assumed that only active soil gas sampling will be used in calculating a quantitative estimate of inhalation risk because of the difficulties in converting the passive soil gas measurements to a soil gas concentration. Active soil gas sampling consists of driving a probe into the soil and extracting a soil gas sample for laboratory analysis. The results of active soil gas sampling are reported as a concentration in units of mass over volume (e.g., mg/m³). This soil gas concentration must then be scaled to a representative air EPC for use in the risk assessment. EPCs for ambient and indoor air may be required in order to evaluate the full range of receptors and exposure pathways identified for the Site. Emissions and dispersion modeling will be conducted to scale the soil gas concentrations to the appropriate inhalation EPCs. This modeling will be conducted separately for the indoor and ambient air exposures because of the differences in the infiltration rates and dilution that occur for soil gas entering ambient versus indoor air. Indoor and ambient air EPCs are discussed below.

4.3.3.1 Indoor Air Exposure Point Concentrations

For indoor air concentrations, the soil gas concentration will be scaled through the use of attenuation factors. Indoor air exposures at the Site will be in commercial or industrial buildings. Default attenuation factors are available from the California Environmental Protection Agency (Cal/EPA; 2005) for commercial buildings. The default attenuation factors will be used with the maximum detected soil vapor concentration in the site assessment area to provide an initial screening of the potential inhalation health risks. For chemicals detected in soil gas that present an elevated health risk based on the initial screening, a more refined and site-specific approach based on the Johnson and Ettinger (J&E) vapor intrusion model will be executed.

¹¹ Other data types may be considered for comparative purposes in the data usability evaluation conducted for the source/site assessment area or as a secondary line of evidence discussed in the uncertainty section of the HHRA.

The J&E vapor intrusion model has been used by EPA (2002b) for developing attenuation factors for soil gas infiltration into indoor air. The J&E model predicts the rate of transport of volatile chemicals through the vadose zone and into indoor air. The transport through the vadose zone is a response to the concentration gradient modeled using Fick's First law. The diffusion in soil is described by an effective diffusion coefficient that is based on chemical-specific diffusivity values and soil porosity. At the interface of the vadose zone and building foundation, the J&E model uses an approximation of the convective flux to estimate the rate at which the vapors would be drawn into the indoor air. The infiltration rate of vapors from the soil is balanced with the exfiltration rate of gases from the above-ground portion of the building to estimate the steady-state indoor air concentration.

Several versions of the J&E model are available from EPA (2004b) depending on the nature of the source being modeled. The spreadsheet model developed for use with soil gas will be applied for this evaluation. Inputs will be a mixture of chemical- and site-specific values along with recommended defaults. The chemical-specific inputs are comprised of the soil gas concentration and various chemical properties (e.g., diffusivity and Henry's Law constant). The input soil gas concentrations will be generated from the source area soil vapor sampling. The chemical properties will be the default values for the J&E model. Site-specific parameters will be used when available for the soil and building properties required for the J&E model. Default parameters values from ASTM (2000) for commercial buildings will be used when site-specific information is unavailable.

4.3.3.2 Ambient Air Exposure Point Concentrations

Based on the results of the indoor air risk assessment it may not be necessary to quantitatively evaluate ambient air exposures associated with soil vapor concentrations as indoor air concentrations will be orders-of-magnitude higher than ambient air concentrations. If necessary, the ambient air concentrations will be determined using a steady-state Fickian diffusion model to predict the flux of vapors through the soil and into ambient air. The vapor flux model is based on an effective diffusion coefficient in soil, the soil gas concentration, and the distance the soil gas must travel to reach the soil surface. The effective diffusion coefficient in soil is calculated from the chemical-specific air diffusivity values and soil porosity values. The chemical-specific diffusivity values and soil porosity values will be consistent with the values used in the J&E modeling for the indoor air concentrations. The soil gas sampling for the source area will be used to generate values of the soil gas concentration and travel distance in the vadose zone.

The ambient air concentrations for vapors released from soil to ambient air will be estimated using the dispersion factor presented by EPA (1996a). The dispersion factor proposed for use in the risk assessment is based on meteorological data collected from Las Vegas, NV. The EPA (1996a) provides a range of dispersion factors depending on the size of the source area being

evaluated. The available dispersion factor that is based on a source size that most closely matches the area being considered in a risk assessment will be selected.

5 TOXICITY ASSESSMENT

The purpose of a toxicity assessment is to summarize health effects that may be associated with exposure to the constituents included in the risk assessment and to identify doses that may be associated with those effects. The focus of the toxicity assessment will be on effects associated with repeated long-term exposures and on effects that could be associated with the chemical and radionuclide concentrations and pathways of exposure that are relevant for this Site. Toxicity values developed based on dose-response assessments for these relevant adverse effects will be identified. These toxicity values are numerical expressions of dose and response, and vary based on factors such as the route of exposure (e.g., oral or inhalation) and duration of exposure (e.g., subchronic, chronic).

In assessing the potential toxicity of chemicals and radionuclides, duration of exposure is an important factor because the exposure levels that can cause toxic effects are usually lower when exposures continue for a longer period of time. For example, with continuous exposure to a chemical for many years (typically referred to as chronic exposure), much lower concentrations (and resulting doses) of a chemical could be associated with toxic effects, compared with concentrations that would be identified as causing toxic effects in a person who is exposed to a chemical for only one day (referred to as an acute exposure). Intermediate duration exposures (referred to as subchronic exposures) are more likely to suggest toxic effects at intermediate concentrations. The risk assessments will evaluate risks associated with scenarios involving subchronic and chronic exposures to COPCs on and around the Site; acute exposures will not be evaluated quantitatively.

The following section describes the procedures that will be used to identify and assess toxicity information. Additional discussion is provided for the approach used to assess the toxicity of asbestos and mixtures of dioxins/furans and carcinogenic polycyclic aromatic hydrocarbons (PAHs).

5.1 METHODS FOR TOXICITY ASSESSMENT

Standard procedures, per EPA (1989 and others) will be followed to identify and assess toxicity factors and other relevant toxicity information, such as the weight-of-evidence (WOE) category for carcinogenic potential. As recommended in the EPA memorandum, *Human Health Toxicity Values in Superfund Risk Assessments* (USEPA 2003b), the primary sources that will be consulted for toxicity values are, in order of priority, EPA's Integrated Risk Information System (IRIS; USEPA 2010) and EPA's provisional peer reviewed toxicity values (PPRTVs) from the National Center for Environmental Assessment/Superfund Health Risk Technical Support Center. If neither IRIS toxicity values nor PPRTVs are available, then toxicity values will be obtained from other documented sources, such as EPA's Health Effects Assessment Summary Tables (HEAST; USEPA 1997b), the Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk

levels (MRLs; ATSDR 2007), and Oak Ridge National Laboratory's (ORNL) Risk Assessment Information System (RAIS; USDOE 2007). Toxicity values appropriate to the relevant exposure routes (e.g., oral, inhalation) and exposure times (e.g., subchronic, chronic) determined for the risk assessment will be collected from these sources.

In addition to these sources, human health toxicity criteria developed by Integral Consulting (Integral) for five organic acid SRCs will be used. The toxicity criteria developed by Integral for diethyl phosphorodithioic acid (DEPT) and dimethyl phosphorodithioic acid (DMPT) were submitted and approved with modification by NDEP in 2007 (Integral 2006; NDEP 2007). The toxicity criteria developed for 4-chlorobenzene sulfonic acid (pCBSA), benzenesulphonic acid (BSA), and phthalic acid were submitted to NDEP in November 2007 (Integral 2007) and approved by NDEP in 2008 (NDEP 2008i,j). The final NDEP-approved values will be used in the risk assessment. Following NDEP guidance (NDEP 2009g), the noncarcinogenic toxicity criterion for dichlorobenzil will be based on the toxicity criterion for 4,4'-dichlorobenzophenone (DCBP), adjusted with additional uncertainty factors to account for the likely greater environmental persistence of dichlorobenzil compared to the surrogate, and for database deficiencies. Additionally, in line with NDEP guidance (NDEP 2006c) pyrene will be used as a toxicological surrogate for noncancer toxicity endpoints for PAHs where no noncancer toxicity criterion are available from EPA or the alternative sources listed above. As recommended by NDEP, the noncarcinogenic toxicity criterion for pyrene will be adopted for the following PAHs: benzo(a)anthracene, benzo(a)pyrene (BaP), benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-c,d)pyrene, acenaphthylene, benzo(g,h,i)perylene, and phenanthrene.

Route-to-route extrapolation assumes that once a chemical is absorbed into the bloodstream, the health effects are similar regardless of whether the route of exposure is oral, dermal, or inhalation. This assumption may be valid for some chemicals with pharmacokinetic characteristics that are similar regardless of the route of administration; however, for many chemicals, factors such as absorption, metabolism, distribution, and elimination vary by exposure route, leading to substantial differences in toxicity. Typically, EPA recommends using route-to-route extrapolation to assess risks from absorbed dose following dermal exposures. These recommendations will be followed here and are discussed in the following sections.

EPA (2009a) explicitly warns against extrapolating oral toxicity criteria to inhalation values because the amount of the chemical that reaches the target site through the inhalation pathway is not a simple function of known parameters (i.e., BW, inhalation rate), but rather a complicated set of factors including the physiochemical characteristics of the inhaled contaminant and human physiologic parameters. Therefore, consistent with EPA (2009a) guidance, route-to-route extrapolations will not be conducted to assess inhalation exposures for most chemicals. The only exceptions to this are cases in which EPA has published inhalation toxicity values that were generated using route-to-route extrapolations. Consistent with EPA (2009a) guidance, these values will be used in the risk assessment without adjustment.

In the case that toxicity criteria are not available for a COPC specific to the exposure route being evaluated a quantitative evaluation of risks associated with exposure to the COPC will not be completed. Uncertainties associated with the exclusion of these COPCs from the quantitative risk evaluation will be discussed in the uncertainty section, as relevant.

The following two subsections describe the toxicity values used to assess noncancer and carcinogenic effects of chemicals including radioactive constituents.

5.1.1 Noncancer Effects from Chemical Exposures

The potential for noncancer health effects from chronic exposures (i.e., greater than 7 years) will be evaluated by comparing the estimated daily intake with a reference dose (RfD) for oral exposure routes, and a reference concentration (RfC) for inhalation exposure routes. Chronic toxicity values represent average daily exposure levels at which no adverse health effects are expected to occur with chronic exposures. Subchronic RfDs/RfCs represent average daily exposure levels at which no adverse health effects are expected to occur with subchronic exposures of less than 7 years, as would be the case for the construction worker and trespasser scenarios to be evaluated for the site. RfDs/RfCs reflect the underlying assumption that systemic toxicity occurs as a result of processes that have a threshold.

The RfDs/RfCs for many noncarcinogenic effects are derived based on laboratory animal studies or epidemiological studies in humans. In such studies, the RfD/RfC is typically calculated by identifying the highest concentration or dose that does not cause observable adverse effects (the no-observed-adverse-effects level [NOAEL]) in the study subject. If a NOAEL cannot be identified from the study, a lowest-observed-adverse-effects level (LOAEL) may be used. This dose or concentration is then divided by uncertainty factors to calculate an RfD/RfC.

Uncertainty factors are applied to account for limitations of the underlying data and are intended to ensure that the toxicity value calculated based on the data will be unlikely to result in adverse health effects in exposed human populations. For example, an uncertainty factor of 10 is used to account for interspecies differences (if animal studies were used as the basis for the calculation), and another factor of 10 is used to address the potential that human subpopulations such as children or the elderly may have increased sensitivity to the chemical's adverse effects. Thus, variations in the strength of the underlying data are reflected in the uncertainty factors used to calculate the toxicity values and in the low, medium, or high confidence ratings assigned to those values (USEPA 2010).

For cases in which toxicity values are not available for the specific time-frame, or exposure route being evaluated, in some instances existing values for other time-frames or routes may be used. For example, EPA states that in cases in which a reference value for a desired duration period (e.g., subchronic) is not available, a reference value based on the next longer duration of

exposure may be used as a conservative estimate that would be protective for the shorter-term ED (USEPA 2009a). This procedure will be adopted for the risk assessments.

RfDs are not available for assessing the dermal exposure route. Oral toxicity values are typically used instead. Because oral toxicity values are usually derived from administered doses, while dermal exposure estimates are expressed as absorbed doses, the oral toxicity values must be adjusted to reflect absorbed dose. This adjustment is accomplished by multiplying the oral RfD by a chemical-specific oral absorption rate. The chemical-specific oral absorption rate is an expression of the fraction of contaminant absorbed in the gastrointestinal (GI) tract in the critical toxicity study. This procedure will be used in the risk assessment. GI absorption values (ABS_{GI}) will be obtained from EPA's *RAGS Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)* (USEPA 2004a), NDEP guidance (NDEP 2008k) and the ORNL RAIS (ORNL 2007). Following EPA guidance (2004a) toxicity criteria for chemicals with an ABS_{GI} less than 50% will be adjusted.

A summary of toxicity for each COPC will include the chronic and subchronic RfD or RfC, as well as the target organ of toxicity and uncertainty factors used in deriving the RfD/RfC. Uncertainties in the toxicity values will additionally be described.

5.1.2 Carcinogenic Effects from Chemical Exposures

To assess carcinogenic health effects, CSFs are used for oral and dermal exposures, while IURs are used for inhalation exposures. CSFs and IURs are upper-bound estimates of the carcinogenic potency of chemicals. They are used to estimate the incremental risk of developing cancer, corresponding to a lifetime of exposure at the levels described in the exposure assessment. In standard risk assessment procedures, estimates of carcinogenic potency reflect the conservative assumption that no threshold exists for carcinogenic effects (i.e., that any exposure to a carcinogenic chemical will contribute an incremental amount to an individual's overall risk of developing cancer).

Another component of assessing carcinogenic health effects is a qualitative evaluation of the extent to which a chemical is a human carcinogen. For many chemicals listed in IRIS, this evaluation was conducted by EPA using a classification system for WOE determination.¹² A chemical is assigned a WOE classification based on data obtained from both human and animal studies. Chemicals for which EPA considers adequate human data indicating carcinogenicity are available are categorized as "known human carcinogens" (WOE class A), while other chemicals with various levels of supporting data may be classified as "probable human carcinogens" (WOE class B1 or B2), or "possible human carcinogens" (WOE class C). Where

¹² The WOE categories described in the final *Guidelines for Carcinogen Risk Assessment* (USEPA 2005) as "standard hazard descriptors" differ from and may eventually supersede those used in IRIS (USEPA 2010). These descriptors include "carcinogenic to humans," "likely to be carcinogenic to humans," "suggestive evidence of carcinogenic potential," "inadequate information to assess carcinogenic potential," and "not likely to be carcinogenic to humans."

EPA considers that data are inadequate for determining carcinogenicity, the chemical is “not classifiable as to human carcinogenicity” (WOE class D). When studies provide evidence of noncarcinogenicity, a chemical is assigned a WOE class E (USEPA 2010).

As described for noncarcinogens, toxicity values measuring carcinogenic potency are not readily available for the dermal exposure route. Following EPA guidance, oral CSFs for chemicals with ABS_{GI} less than 50% will be adjusted to determine dermal CSFs. ABS_{GI} will be obtained from EPA’s *RAGS Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)* (2004a), NDEP guidance (2008k), and the ORNL RAIS (ORNL 2007).

A summary of toxicity for each COPC will include the qualitative WOE classification and the CSF or URF. Uncertainties in the incremental risk values will additionally be described.

5.1.3 Effects from Radionuclides

Biological effects associated with exposure to ionizing radiation in the environment may include carcinogenicity, mutagenicity, and teratogenicity. EPA (2001) has determined that cancer risk is the most significant health effect potentially associated with exposure to radionuclides¹³. EPA classifies all radionuclides as WOE Class A, based on their property of emitting ionizing radiation and on the WOE provided by epidemiological studies of radiogenic cancers in humans (USEPA 2001, 2009b).

CSFs for radionuclides are available from HEAST for specific ingestion, inhalation, and external exposures (USEPA 2001, 2009b). The CSFs are derived using models that take into account age- and gender-dependence of radionuclide intake, metabolism, dosimetry, radiogenic risk, and competing causes of death. The model averages the risk over the lifetime of the exposed individual. Consequently the slope factors are not expressed as a function of BW and time.

The resultant CSFs represent central estimates of age-averaged, excess lifetime cancer incidence per unit of activity of a given radionuclide inhaled or ingested, for internal exposure, or per unit time-integrated activity concentration in air or soil for external exposure for an average member of the reference population¹⁴. The CSFs may be used to estimate the lifetime cancer incidence risk attributable to a given radionuclide exposure for an average member of the population, but are not appropriate for assessing the risk to a single individual of a particular age or gender. In addition to the age-averaged values, for the soil ingestion pathway, an adult only CSF is available from HEAST.

¹³ The only exception to this is uranium, which presents both noncarcinogenic chemical hazard and carcinogenic radiological risks. In line with EPA guidance (USEPA 1996b) in the case that uranium is selected as a COPC for a risk assessment, both types of risk will be evaluated. Noncarcinogenic health effects will be evaluated as for other noncarcinogenic chemicals using toxicity criteria specific to uranium.

¹⁴ Current values were calculated using characteristics, mortality statistics, and baseline cancer rates from the 1980s U.S. population

All radionuclides undergo a decay process in which the parent radionuclide is transformed in atomic number, mass, or excitation state. In some cases the resulting decay products are radioactive, and may undergo further decay. Each of these decay products may have different physical and chemical properties which affect their environmental fate and transport, as well as different toxic characteristics and potencies. Because each is unique in its action and toxicity, consideration of all of the decay products is a key element in the risk assessment process. The radiation dose estimates used to calculate the radionuclide CSFs explicitly consider the production of radioactive decay products within the body following ingestion or inhalation; however, only intake or external exposure to the single radionuclide is considered. For certain radionuclides with decay products where contributions of dose and risk from radioactive decay products may be significant, EPA has derived CSFs which incorporate the contribution of short-term decay chain products (i.e., less than 1 year half-life) to the total risk. The resultant CSFs are higher than those which consider the parent radionuclide only, because they additionally consider the risk contribution from the short-lived decay products. The calculation of the CSF from these decay chains assumes the presence of SE.

The CSFs from HEAST will be used to evaluate risks to populations with completed exposures at the Site. The adult only CSF for soil ingestion will be utilized for scenarios where exposure occurs within adulthood only (e.g., worker populations). For all other receptor populations and completed exposure pathways, the age-averaged CSFs will be used. Given that the difference between the age and gender-averaged risk coefficients and the adult-only risk coefficients are slight, the use of the age-averaged values are considered appropriate for evaluating risks to these populations. For the radionuclides for which it is available, the CSF which includes the contribution of short-term decay products will be selected. Any significant uncertainties resulting from the use of an age adjusted CSF, or CSFs which incorporate, or do not incorporate decay products will be discussed in the risk assessments.

5.1.4 Effects from Lead

Adverse health effects associated with exposure to lead include, but are not limited to, neurotoxicity, developmental delays, and reproductive impairment (USEPA 2010). No RfD or RfC is available from EPA for lead, and given the current knowledge regarding background body burdens, lead pharmacokinetics, and low exposure levels associated with some health effects, EPA has determined that it is not appropriate to develop reference levels for lead (USEPA 2010). Given the lack of an RfD and RfC, the method for characterizing risk from lead exposure differs from that utilized for most noncarcinogenic agents. This methodology is presented in Section 6.4.

5.1.5 Effects from Asbestos

Asbestos risks will be assessed in line with the approaches specified in NDEP's (2009f) *Technical Guidance for the Calculation of Asbestos-Related Risk in Soils for the BMI Complex and Common Areas*. The approach relies on exposure-response coefficients that describe the toxicity of different fiber lengths and types of asbestos. These risk coefficients are adopted from the draft, *Technical Support Documents for a Protocol to Assess Asbestos Related Risk* (USEPA 2003c).

The majority of available information indicates that lung cancer and mesothelioma are the most important risks associated with low levels of asbestos (NDEP 2009f, USEPA 2003c). Types and aspect ratios (relative length versus diameter) of asbestos fibers differ, and are known to affect the potency of the material; therefore, deriving conclusions regarding the health effects related to asbestos exposure is complex. In the EPA draft document (USEPA 2003c) studies from environments with asbestos dusts of differing characteristics were reviewed to evaluate asbestos related risks. EPA developed an optimal exposure index, which best reconciles the published literature. The index assigns equal potency to fibers longer than 10 μm and thinner than 0.4 μm and assigns no potency to fibers of other dimensions. The optimal exposure index also assigns unique exposure-response coefficients for chrysotile and amphibole fibers for the endpoints of mesothelioma and lung cancer. Optimum dose response coefficients, based on the body of available data will be assumed for this risk assessment. The coefficients are presented in Appendix E.

5.2 APPROACHES FOR CHEMICAL MIXTURES

For some groups of chemicals, such as polychlorinated dibenzo-p-dioxins (PCDDs)/polychlorinated dibenzofurans (PCDFs)/PCBs and PAHs, information on the toxic potency of individual constituents of the group are expressed in relative terms¹⁵. The approaches for evaluating PCDDs/PCDFs and PAHs are described below.

5.2.1 Toxicity Equivalency Approach for PCDDs/PCDFs/PCBs

Dioxins and furans (PCDDs and PCDFs) are two groups of structurally similar, tricyclic, almost planar, organic compounds that exhibit similar physical and chemical properties. There are 75 dioxins and 135 furans, called congeners, which are differentiated by their number and position of chlorine atoms. Researchers in the early 1980s concluded that a subset of PCDDs, PCDFs, and PCB congeners shared a common mechanism of action and induced comparable biological and toxic responses (USEPA 2003d). However, the potency of the different congeners varies considerably.

¹⁵ Other chemical mixtures have toxicity criteria that represent the potency of the entire mixture (i.e., various Aroclors). These mixtures will be treated in line with the protocols described in Section 5.1.

Seventeen PCDD and PCDF congeners (7 PCDDs, 10 PCDFs) exhibit what is termed “dioxin-like” toxicity. These 17 congeners have chlorine atoms present in the 2, 3, 7, and 8 positions on the ring structure of the molecule and are more toxic than other congeners with fewer chlorine atoms or with chlorine atoms in different positions on the ring structure. The congener 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) is the most widely studied and has been found to exhibit the most potent toxic response. Similarly, 12 coplanar PCB congeners have been shown to exhibit dioxin-like toxicity and are grouped with the 17 dioxin/furan congeners that exhibit toxicity similar to 2,3,7,8-TCDD (Van den Berg et al. 1998).

Human health risk estimates for exposures to PCDDs/PCDFs traditionally require conversion of concentrations of individual dioxin and furan congeners to their 2,3,7,8-TCDD TEQ concentration using congener-specific toxic equivalency factors (TEFs). The 2,3,7,8-TCDD TEQ concentration for each sample is calculated by multiplying concentrations of individual congeners by their congener-specific TEFs, and summing the results for all congeners as shown in Equation 5-1, below. The 2,3,7,8-TCDD TEQ concentration is assumed to express the total potency of the mixture of PCDDs/PCDFs in a sample to exert the toxicity of 2,3,7,8-TCDD.

$$\text{TEQ} = \Sigma(C_1 \times \text{TEF}_1) + (C_2 \times \text{TEF}_2) + \dots(C_n \times \text{TEF}_n) \quad \text{Eq. 5-1}$$

where,

C = congener specific concentration (e.g., mg/kg)
TEF = congener specific TEF (unitless).

For assessment of human health risks, TEFs developed by the World Health Organization (WHO; Van den Berg et al. 1998) and adopted by NDEP for deriving BCLs (NDEP 2009e) will be to calculate TEQs. These TEFs are the most widely accepted equivalency factors and are typically expressed as “WHO98 TEFs”. Table 5-1 presents the TEFs that will be used in the risk assessment.

Risk from TEQ concentrations are calculated similarly to that from other COPCs by combining calculated exposure with a risk-based criteria.

5.2.2 Relative Potency Approach for PAHs

The cancer potencies of individual carcinogenic PAH chemicals are expressed relative to the cancer potency of BaP. This procedure involves applying chemical-specific relative potency factors (RPFs) to the CSF for BaP, resulting in a CSF adjusted for the toxicity of each PAH relative to BaP. Table 5-1 presents the RPFs provided by EPA (1993) that will be used in the risk assessment if PAHs are selected as a COPC.

Risks associated with PAHs will be evaluated in a compound specific manner using toxicity criteria based on the RPFs outlined above. In order to retain the ability to more fully understand the contributions of various PAHs to estimates of risk, the individual PAHs for a given sample will not be summed in an *a priori* manner. However, to avoid reducing the effects of multiple PAHs that may act via a similar mode of action, in the case that any single carcinogenic PAH is selected as a COPC, the full suite of carcinogenic PAHs will be evaluated using one half the SQL for non-detects. This could in certain situations lead to risks that are dominated by non-detect values. If this occurs, the uncertainty associated with this approach will be discussed in the risk report.

Despite the wide use of RPFs in health risk assessments at Superfund and Resource Conservation and Recovery Act (RCRA) sites to express the toxicity of carcinogenic PAHs in relation to the toxicity of BaP, numerous limitations of its use have been identified. These limitations contribute to uncertainty in the estimation of risks for the Site. The uncertainties associated with this approach will be discussed in the risk assessment.

6 RISK CHARACTERIZATION

The goal of risk characterization is to present and interpret the key findings of the risk assessment, along with their limitations and uncertainties, for use in risk management decision making. In the process of risk characterization, quantitative estimates of exposure and toxicity are compared to yield estimates of potential health risk. Risks for noncancer and cancer effects are estimated separately because of differences in calculation methods.

With the exception of lead, risks associated with exposure to multiple non-carcinogens will be considered cumulatively. Similarly, risks associated with exposure to multiple chemical carcinogens will be added. The methods for combining risk estimates to non-carcinogens and chemical carcinogens for a given exposure pathway is discussed below. Cancer risks from chemical, radionuclide, and asbestos will be calculated and presented separately.

As presented in Section 3.4.1, if statistical analyses indicate that a particular SRC is within background soil levels, then the SRC will not be identified as a COPC to be quantified in the HHRA. Background risks for COPCs may be calculated separately and discussed in the uncertainty evaluation to provide context to the HHRA results.

This section describes the methods that will be used for quantifying and interpreting risks and for characterizing uncertainties associated with the risk estimates.

6.1 NONCANCER RISKS FROM CHEMICAL EXPOSURES

Health risks other than cancer are characterized as the increased likelihood that an individual will suffer adverse health effects as a result of chemical exposure. To evaluate noncancer risks, the ratio of the exposure term (i.e., average daily intake or EC) to the corresponding noncarcinogenic toxicity reference value (i.e., RfD or RfC) is calculated. It is most appropriate to apply reference values that correspond with the duration of exposure assumed for a specific receptor (e.g., where ED is less than 7 years, a subchronic RfD or RfC is ideally used). This ratio is referred to as the HQ. If the calculated value of the HQ is less than or equal to 1, no adverse health effects are expected. If the calculated value of the HQ is greater than 1, then further risk evaluation is needed.

The HQ is calculated for oral and dermal exposure pathways using the following equation:

$$HQ(\text{unitless}) = \frac{ADD}{RfD} \quad \text{Eq. 6-1 (adapted from USEPA 1989)}$$

where,

ADD¹⁶ = average daily dose of the chemical via the specified exposure route (mg/kg-day)
RfD = reference dose (mg/kg-day).

The HQ is calculated for the inhalation exposure pathway using the following equation:

$$HQ(\text{unitless}) = \frac{EC}{RfC} \quad \text{Eq. 6-2 (adapted from USEPA 2009a)}$$

where,

EC = exposure concentration ($\mu\text{g}/\text{m}^3$)
RfC = reference concentration ($\mu\text{g}/\text{m}^3$).

To evaluate the effect of exposure to multiple chemicals that act on the body in a similar manner, the HQs for each exposure pathway for individual chemicals are typically summed to determine a noncancer HI using the following formula:

$$HI(\text{unitless}) = HQ_1 + HQ_2 + \dots + HQ_i \quad \text{Eq. 6-3 (adapted from USEPA 1989)}$$

where,

HQ = hazard quotient for specified exposure pathway (unitless).

HIs for multiple chemicals are generally not summed if the reference doses for the chemicals are based on effects on different target organs. This is because the noncancer health risks associated with chemicals that affect different target organs are not likely to be additive. For this reason, in the case that the total HI exceeds 1 for all COPCs combined, a more refined analysis based on target organ may be conducted.

6.2 CANCER RISKS FROM CHEMICAL EXPOSURES

The cancer risk estimates derived using standard risk assessment methods are characterized as the incremental probability that an individual will develop cancer during his or her lifetime due

¹⁶ For exposure via dermal contact, the ADD is referred to as the dermally absorbed dose (DAD); however, for simplicity, intakes are referred to as the ADD for all exposure routes.

to exposure to SRCs resulting from the specific exposure scenarios that are going to be evaluated. The term "incremental" reflects the fact that the calculated risk associated with site-related exposure is in addition to the background risk of cancer experienced by all individuals in the course of daily life.

Excess incremental lifetime cancer risks are calculated as the product of the exposure term (i.e., lifetime average daily intake or EC) and the expression of the carcinogenic potency of chemicals (i.e., CSF or IUR).

Excess incremental lifetime cancer risk from oral and dermal exposures is calculated using the following equation:

$$\text{Cancer Risk}(\text{unitless}) = \text{LADD} \times \text{CSF} \quad \text{Eq. 6-4 (adapted from USEPA 1989)}$$

where,

LADD = lifetime average daily dose of the chemical via the specified exposure route (mg/kg-day)
CSF = cancer slope factor (kg-day/mg).

Excess incremental lifetime cancer risk from inhalation exposures is calculated using the following equation:

$$\text{Cancer Risk}(\text{unitless}) = \text{EC} \times \text{IUR} \quad \text{Eq. 6-5 (adapted from USEPA 2009a)}$$

where,

EC = exposure concentration ($\mu\text{g}/\text{m}^3$)
IUR = inhalation unit risk ($\text{m}^3/\mu\text{g}$).

6.3 RADIONUCLIDE RISKS

Cancer risks resulting from intakes of radionuclides are calculated in a similar manner to cancer risks for chemicals. The primary difference in the characterization is that equations used to characterize risks from radionuclides rely on intake parameters, and risk coefficients, expressed in units of activity.

For internal exposure excess cancer risk will be calculated as:

$$\text{Cancer Risk}(\text{unitless}) = \text{Dose} \times \text{CSF} \quad \text{Eq. 6-6 (adapted from USEPA 1996b)}$$

where,

Dose = total dose of a radionuclide via the specified exposure route
(pCi)
CSF = cancer slope factor (pCi⁻¹).

For external exposure excess cancer risk will be calculated as:

$$\text{Cancer Risk (unitless)} = EET \times CSF \quad \text{Eq. 6-7 (adapted from USEPA 1996b)}$$

where,

EET = external exposure term for a radionuclide (pCi – year/g)
CSF = cancer slope factor (g/pCi - year).

6.4 LEAD RISKS

In the case that lead analytical results exceed the NDEP BCL of 800 mg/kg, the ALM will be used to estimate risks associated with lead exposure. The ALM predicts the blood lead level in an adult with a site-related lead exposure by summing the “baseline” blood lead level (PbB₀) (i.e., that which would occur in the absence of any site-related exposures) with the increment in blood lead concentration that is expected as a result of increased exposure due to contact with lead-contaminated soil at the Site (USEPA 2003a). According to EPA (2003a), protection of the fetus is the most health-sensitive endpoint for adults. In-line with assessing this endpoint the ALM includes a module to predict fetal blood lead levels. In the case that the ALM is applied, following EPA guidance (2003a), central estimates of exposure will be used. An arithmetic mean concentration will be used for the EPC in the model. Baseline blood lead concentrations and geometric standard deviations of blood lead for the ALM will be obtained from U.S. population data presented in the National Health and Nutrition Examination Survey (NHANES) III. A target risk level of no more than a five percent probability that a fetus exposed to lead will exceed a blood lead level of 10 µg/dL will be applied as the risk threshold.

6.5 ASBESTOS RISKS

Risks associated with asbestos will be evaluated using NDEP (2009f) assessment methodology. This methodology details procedures to calculate the risk of additional deaths from lung cancer and mesothelioma from inhalation exposures to asbestos and is discussed in detail below.

NDEP guidance adopts the approaches recommended in EPA’s draft protocol (USEPA 2003c) for evaluating asbestos-related cancer risk. Under the approach risk is estimated as the product

of a risk coefficient and a mathematical function that depends on the level of exposure, the duration of exposure, and time. Estimates of additional deaths attributable to asbestos from lung cancer, from mesothelioma, and from both combined, are based on the optimum risk coefficients, described in Section 5.1.4. Lifetime asbestos induced risk of both lung cancer and mesothelioma differ between males and females, and smokers and non-smokers, and individual risk coefficients have been derived for each of these sub-populations. Risk estimates for each subgroup are combined with population statistics to determine a population averaged risk.

Asbestos-related risk (ARR) will be calculated as:

$$ARR \text{ (unitless)} = EC \times URF \quad \text{Eq. 6-8 (adapted from NDEP 2009f)}$$

where,

EC = exposure concentration (f/cm³)
URF = unit risk factor (cm³/f).

and,

$$URF = \frac{10^{-5}}{0.0001} \times R = \frac{1}{10} \times R \quad \text{Eq. 6-9 (NDEP 2009f)}$$

where,

R = estimated additional deaths from lung cancer or mesothelioma per 100,000 persons from continuous, lifetime exposure to an asbestos concentration of 0.0001 f/cm³ (for fibrous structures longer than 10 μm and thinner than 0.4 μm) as determined using transmission electron microscopy (TEM) methods.

and,

$$R = 0.5 \times [0.786 \times (NSM + NSF) + 0.214 \times (SM + SF)] \quad \text{Eq. 6-10 (NDEP 2009f)}$$

where,

NSM = corresponding risk for male non-smokers
NSF = corresponding risk for female non-smokers
SM = corresponding risk for male smokers
SF = corresponding risk for female smokers.

The numerator value (10⁻⁵) and denominator value (0.0001) in equation 6-9 allow for an adjustment for the units embedded within the risk coefficients in equation 6-10 which refer to risk per 100,000 persons for exposure to an asbestos air concentration of 0.0001 f/cm³ to be made.

Risks of additional deaths by sub-population to be used for the risk calculations are included in Appendix E. In line with NDEP guidance, in order to be protective of exposure to second hand smoke, the same R value will be used for child receptors in the offsite residential scenario. The combined risks of lung cancer and mesothelioma will be calculated.

6.6 DATA QUALITY ASSESSMENT

A data quality assessment (DQA) is an analysis performed at the completion of a risk assessment in order to determine if a sufficient amount of data were available to support the risk-based decisions evaluated. A DQA of the sampling data used in the HHRA will be presented in the risk assessment report. The sample size calculations will be conducted for the risk driving COPCs. The formula used for the sample size calculation is based on a non-parametric test (the Wilcoxon signed rank test) and on simulation studies performed by the Pacific Northwest National Laboratories (PNNL 2009) that formed the basis for an approximate formula that is based on the normal distribution. Essentially, the formula is the one that would be used if a normal-base test were being performed, but an adjustment is made (multiply by 1.16) to account for the intent to perform a non-parametric test. The formula is as follows:

$$n = 1.16 \left[\frac{s^2}{\Delta^2} \right] \left(z_{1-\alpha} + z_{1-\beta(\mu)} \right)^2 + 0.5 z_{1-\alpha}^2 \quad \text{Eq. 6-11 (PNNL 2009)}$$

where,

n	=	number of samples
s	=	estimated standard deviation of concentrations/fibers/activities
Δ	=	the difference between the threshold value stated in the null hypothesis and the point at which β is specified
α	=	significance level or Type I error tolerance
$\beta(\mu)$	=	Type II error tolerance
z	=	a quantile from the standard normal distribution.

For the selected risk drivers, inputs for the calculations will include an estimate of the variance from the measured data, a desired significance level, and desired power of the test that must be specified at a concentration of interest (which determines the tolerable difference from the threshold value), typically the NDEP BCL. The calculations will cover a range of Type I and Type II error tolerances, and the point at which the Type II error is specified. Accordingly, various combinations of input values will be used, including: values of α of 5%, 10% and 15%; values of β of 15%, 20% and 25%; and, a gray region of width 10%, 20% and 30% of the threshold level.

The results of the DQA will be used to support the uncertainty evaluation, described below.

6.7 UNCERTAINTY EVALUATION

The final element of the risk assessments will be an assessment of the uncertainty in the estimated noncarcinogenic and carcinogenic risks. Uncertainty is inherent in many aspects of the risk assessment process, and generally arises from a lack of knowledge of 1) site conditions and future site use, 2) toxicity and dose-response for COPCs, 3) the extent to which an individual may be exposed to COPCs, and/or 4) the representativeness of modeled EPCs. This lack of knowledge means that assumptions must be made based on information presented in the scientific literature or professional judgment. In general, such assumptions will be made in a manner that intentionally biases the process towards health protection.

Uncertainties in the risk will be identified and addressed qualitatively in general, although some quantitative measures of uncertainty (e.g., probabilistic analyses using Monte Carlo analysis) may be provided. Descriptions of the uncertainty inherent in analytical data and toxicity and exposure parameters used to characterize risks will be provided in the risk assessment reports. The uncertainty analyses will conclude with a discussion of the overall impact of uncertainty in the risk assessment on the risk characterization for the site assessment area.

6.8 PRESENTATION AND INTERPRETATION OF FINDINGS

The risk assessments results will be presented in tabular format and include key supporting information used to calculate the risks. Key pathways and COPCs that drive risk estimates will be identified and discussed. Reports will include discussions of the results in the context of their implications for risk management actions at the site assessment area. Key uncertainties or data gaps and their influence on risk management decisions also will be discussed. Risk assessment reports will include the following:

- Background - description of the site assessment area being addressed including relevant history; relevant geographical information.
- Exposure scenarios - description of receptor groups and pathways for which risks will be characterized.
- Data evaluation - description of data sources selected for use in the risk assessment; details of data treatment.
- COPC selection - description of methodology for selecting COPCs; list of COPCs that will be evaluated.
- Exposure assessment - presentation of exposure parameters and media-specific EPCs; presentation of methodology for calculating exposures; resulting exposures.

- Toxicity assessment - presentation of noncarcinogenic and carcinogenic toxicity criteria; discussion of human health effects associated with risk-driving COPCs.
- Risk characterization - presentation of methodologies for characterizing risks; calculated noncarcinogenic and carcinogenic risks.
- Uncertainties - qualitative and quantitative assessments of key uncertainties and data gaps; a description of the impacts of uncertainties on resulting risk estimates.
- Conclusions.
- References.

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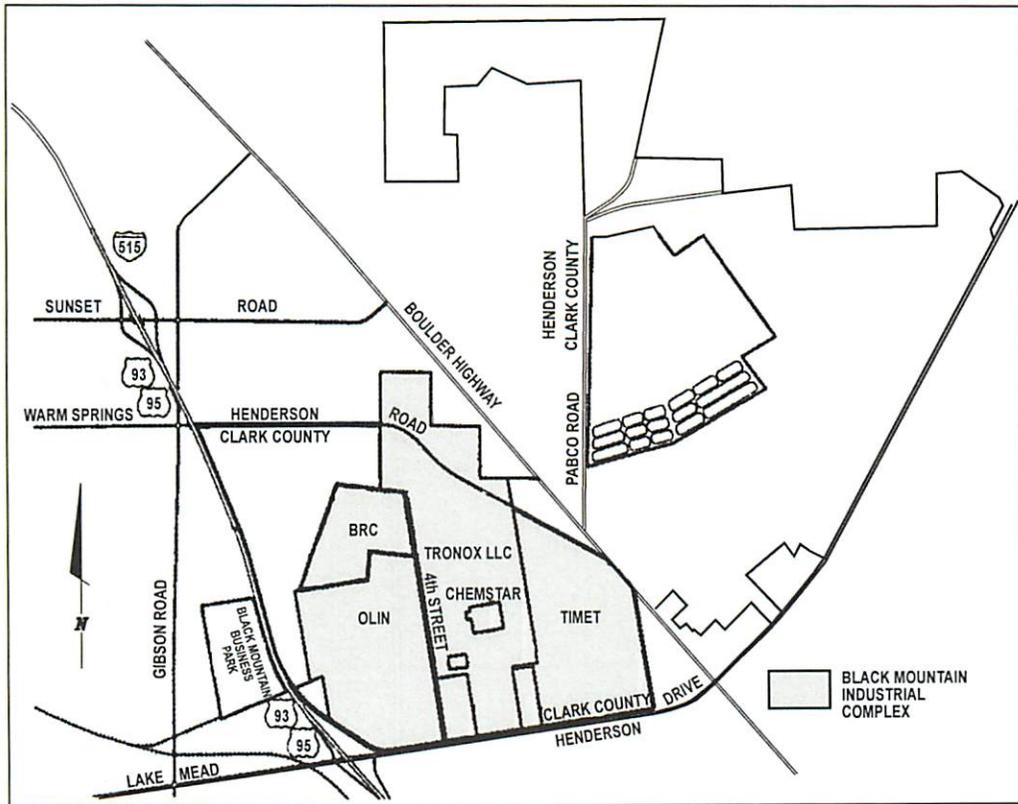
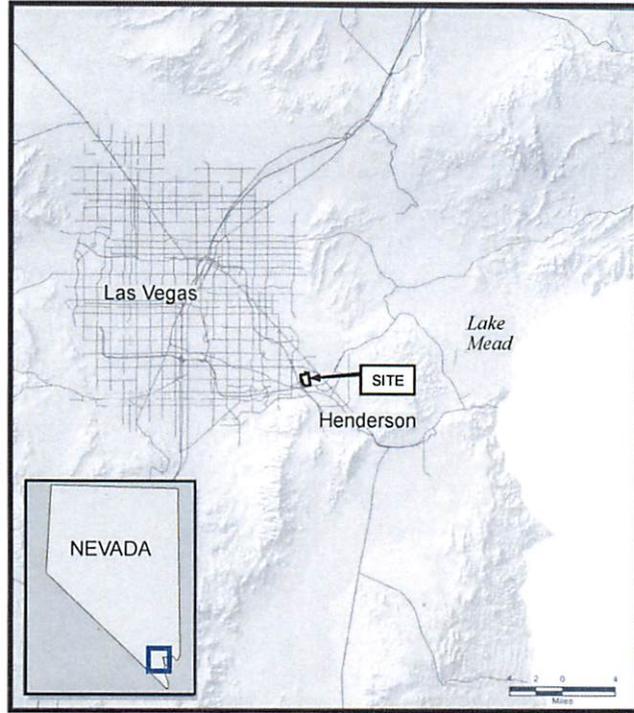
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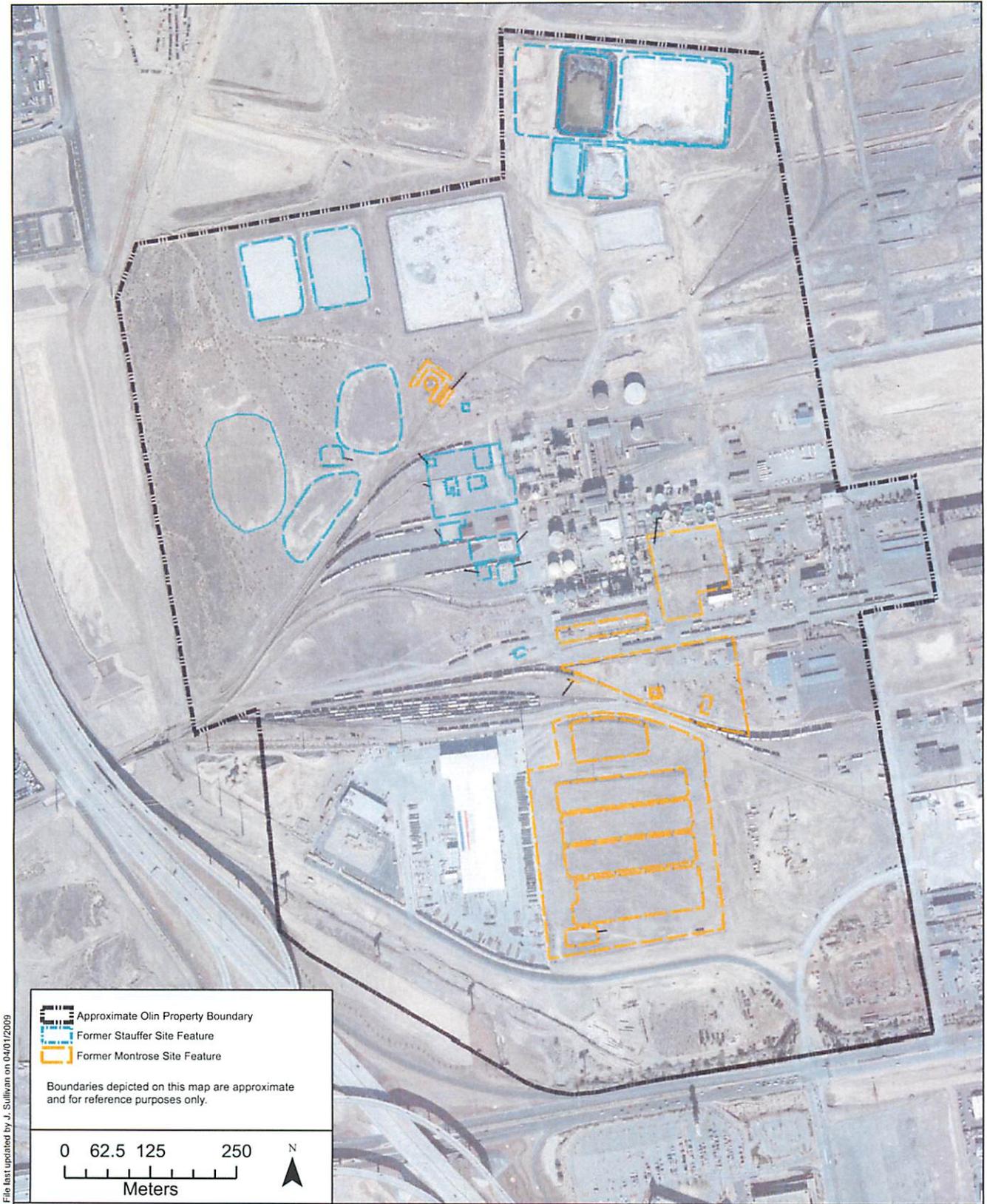
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FIGURES





File last updated by J. Sullivan on 04/01/2009



Figure 1-2.
Former Montrose and Stauffer Site.
Henderson, NV.

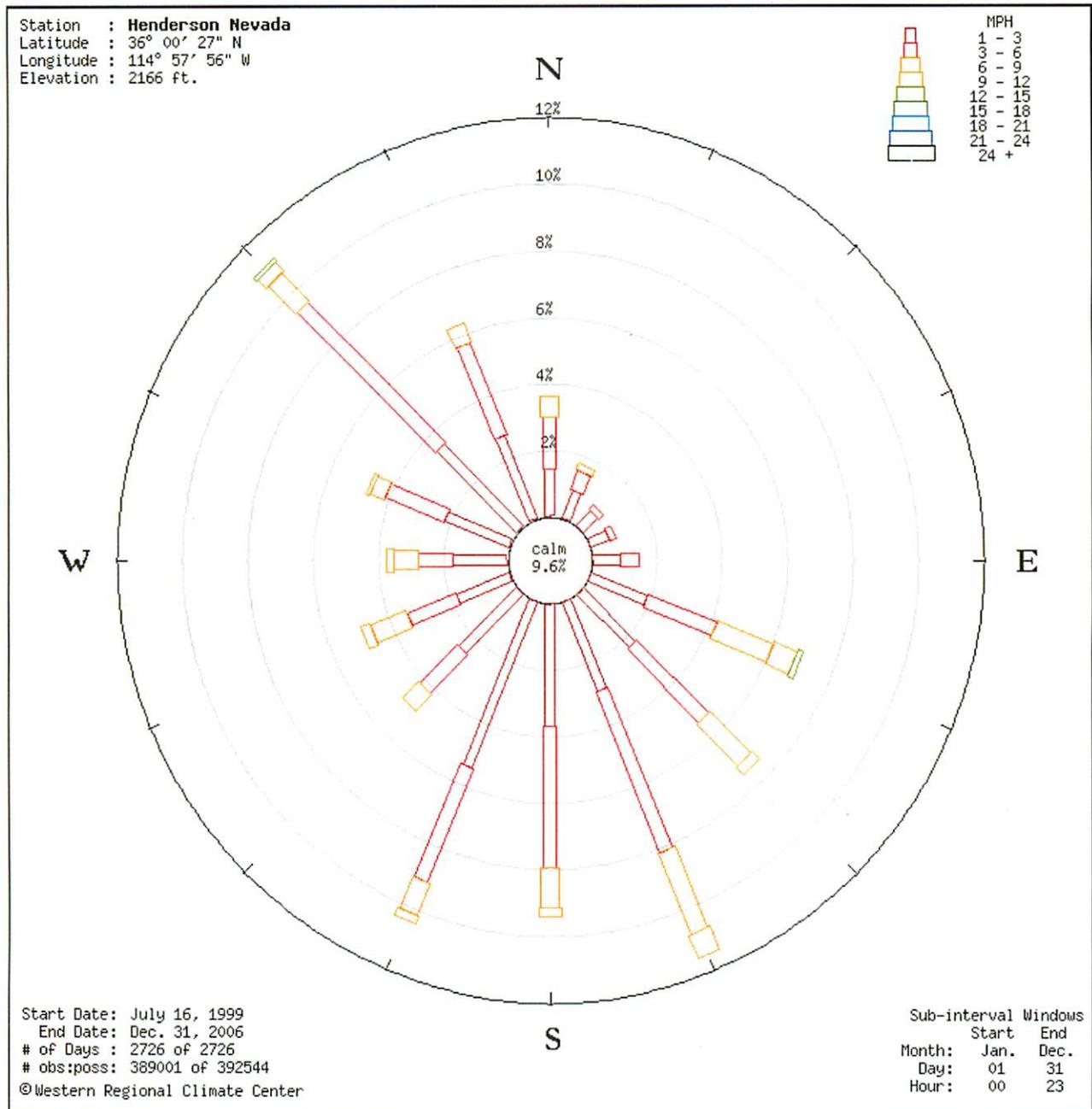


Figure 1-3.
 Windrose for Henderson, Nevada
 Risk Assessment Work Plan
 Former Stauffer Chemical Company Facility
 Henderson Nevada

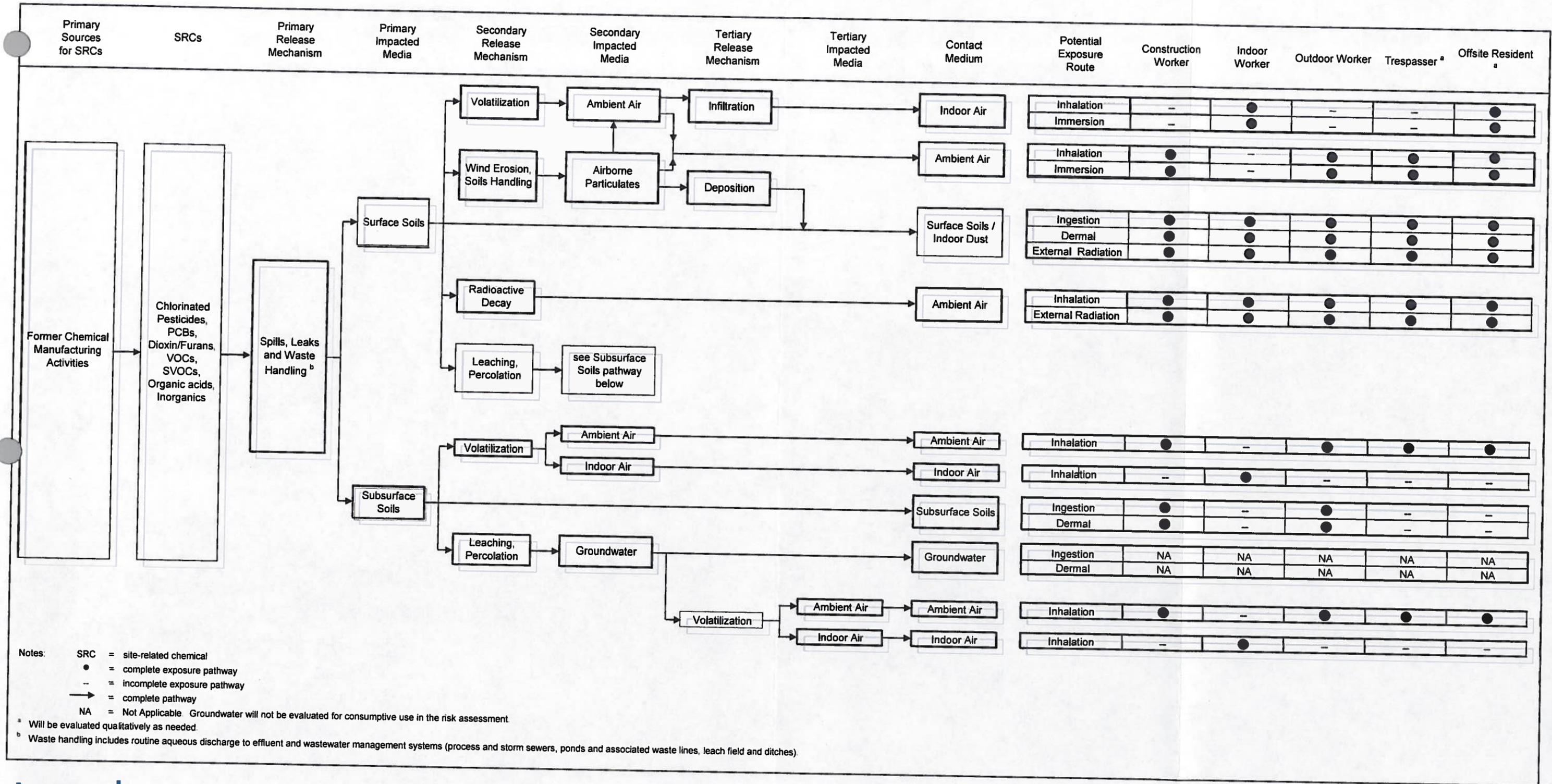


Figure 2-1.
Potential Human Exposure Pathways
Former Montrose and Stauffer Site,
Henderson, Nevada

TABLES

Table 5-1. Toxic Equivalency Factors and Relative Potency Factors.

Dioxin/Furan Congener	Toxic Equivalency Factor ^a
PCDDs	
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.0001
PCDFs	
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0001
Non-Ortho PCBs	
PCB 77	0.0001
PCB 81	0.0001
PCB 126	0.1
PCB 169	0.01
Mono-Ortho PCBs	
PCB 105	0.0001
PCB 114	0.0005
PCB 118	0.0001
PCB 123	0.0001
PCB 156	0.0005
PCB 157	0.0005
PCB 167	0.00001
PCB 189	0.0001
PAHs	
Benzo(a)pyrene	1.0
Benzo(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.01
Dibenzo(a,h)anthracene	1
Indeno(1,2,3-cd)pyrene	0.1
Chrysene	0.001

Notes:

^a Source: Van den Berg et al. (1998).

^b Source: USEPA (1993).

APPENDIX A

RESPONSE TO COMMENTS LOG

NDEP COMMENT RESPONSE LOG, FEBRUARY 2010 COMMENTS			
RISK ASSESSMENT WORKPLAN FORMER MONTROSE AND STAUFFER FACILITIES HENDERSON, NEVADA			
Comment No.	Applicable Section/ Text	Comment	RTC
1	General	The text is not clear about what risks will be calculated in terms of background risk, and incremental lifetime cancer risks. Some discussion up front would be helpful, with consistency thereafter. [ADDITIONAL NOTE FROM B. RAKVICA] NDEP would allow the Companies to qualitatively address the trespasser scenario and off-site resident and we can discuss this on the call	Part 1 - We have added text to Section 6 that described that background risks will not necessarily be calculated, but may be calculated in order to provide context to the risk results. In the case that they are calculated they will be discussed as part of the uncertainty evaluation. Part 2 - We have removed the text throughout the main document and appendices that details the quantitative assessment of the trespasser and off-site resident.
2	General	The points of departure for the deterministic risk assessments should be described up front . [ADDITIONAL NOTE FROM B. RAKVICA] Insert "A" is suggested to be included in the document as suitable text.	We have added Section 1.3 to outline the points of departure for the deterministic risk assessment.
3	General	Some discussion is needed up front that defines terms such as site assessment area, waste management area, exposure area and decision unit. The discussion needs to explain the boundaries or scope of a risk assessment. Sometimes this is alluded to in some places in the Deliverable, but some clear definitions and discussions upfront would help.	We have added text to Section 1.2 that defines the terms site assessment area and exposure area as they relate to the RAS process and the risk assessment process in particular.
4	General	NDEP would like to discuss the Companies' plan to present CTEs for the risk assessment. [ADDITIONAL NOTE FROM B. RAKVICA] NDEP would like to discuss the purpose and utility of CTEs	We have added text to Section 4.1.3, that clarifies that CTE estimates will not be calculated in all situations, but may be calculated in some in order to provide context to the RME results.
5	General	Radon has not been discussed anywhere, please clarify what is planned.	Recognition that receptor groups may be exposed to radon has been added to Sections 2.2.1, 2.2.2, and 2.2.3. In line with the agreements made at the March 24 meeting with the companies and NDEP text stating that the prediction of future radon risk should be deferred for the time being (given that risk assessment methodology from NDEP is not available) was added.
6	Section 1.1.2 pp.1-2	Please identify that imidan is phosmet and that trithion is carbofenothion. This could be provided as a footnote to the text. The relevance of this comment pertains to the fact that the toxicity database does not use trade names. Therefore, this requested edit will reduce any confusion in the future.	These names were added in the requested section.
7	Section 1.2 top of pp. 1-4	Add the soil depth interval for leaching assessment.	At the March 24 meeting with the companies and NDEP it was agreed upon that soil data would be compared to leaching based BCLs, and the results of that comparison presented in the risk assessments, however that any formal evaluation and discussion of leaching would be addressed in the groundwater remedial alternative study. Text describing the scope of the leaching assessment that will be included in the risk assessment was added to Section 1.2.
8	pp. 2-3 2nd paragraph	Please clarify why radionuclides are not part of the SRC list. Based upon data collected since the SRC list was developed, it is now known that radionuclide concentrations in the subsurface in some areas are elevated.	Text in Section 2.2.1 explains that although radionuclides were not part of the formal SRC list defined in Hargis, that for the purpose of the RAWP it is included as an SRC and will be evaluated in the RAs. No change to the text was made. The language in the RAWP was reviewed and approved at the March 24 meeting with the Companies and NDEP.
9	Section 3.1 pp. 3-1, 3rd par. Last sentence	For consistency with measurement units, please edit this sentence to read " If measured soil vapor concentrations (from soil vapor probes) or surface flux rates (from flux chamber measurements) are used to assess...". Please also confirm that, if groundwater data are used as a source term for vapor exposures, whether all detected chemicals will be evaluated or if a screening step will be employed.	The companies have agreed upon a revised approach that focuses on the use of soil gas data for the vapor assessment at the exclusion of surface flux chambers. Matrix data (i.e., soil, groundwater) may be utilized as a secondary line of evidence for the risk assessments. The text in this section has been revised to reflect this approach, and therefore this comment is no longer relevant.

NDEP COMMENT RESPONSE LOG, FEBRUARY 2010 COMMENTS			
RISK ASSESSMENT WORKPLAN FORMER MONTROSE AND STAUFFER FACILITIES HENDERSON, NEVADA			
Comment No.	Applicable Section/ Text	Comment	RTC
10	- Pp. 3-2 1st partial sentence	For clarification, please edit the sentence as follows (please add the word "data" at the end of the sentence [bold used only for purposes of the comment]: "EPA guidance for data usability in risk assessment (USEPA 1992a,b) and NDEP procedures outlined in guidance issued for assessing data usability for environmental investigations at the BM Complex and Common Areas (NDEP 2008d) will guide the data (risk) assessments.	The requested edit has been made.
11	- pp. 3-3 1st paragraph 2nd sentence	Although the MDA might be reported, NDEP guidance requires the use of all radionuclide data as reported, and not censored at an MDA or any other form of detection limit. This is a global comment that will not be repeated.	The text has been clarified to indicate that a value lower than the MDA, if reported, may be adopted for the RA. The language in the RAWP was reviewed and approved at the March 24 meeting with the Companies and NDEP.
12	- pp. 3-5 Section 3.4.1 1st paragraph 1st sentence	Please note that if there are also non-detects that are significantly greater than background, then such constituents cannot be eliminated. That is, the detection limits that are used for non-detects are important as well.	It was agreed at the March 24 meeting with the Companies and NDEP that the data usability evaluation will address the issue of the relationship between detection limits and background. Section 3.1 briefly overviews the guidance that will be utilized for the data usability assessment and the reporting that will be completed for that assessment. No change was made to the text.
13	- pp. 3-6 Section 3.4.2 1st paragraph last sentence	Reword to "All volatile SRCs that are detected will be evaluated".	Text in this section has been revised to read "All detected SRCs will be evaluated". The change is in line with the decision to use only soil vapor data and not flux or groundwater measurements.
14	- pp. 3-7 Section 3.4.2 last paragraph	This seems insufficient for asbestos. This is about COPC selection, so it would be helpful if some discussion was provided of how or why chrysotile or amphibole will be chosen as a COPC.	Current text was reviewed and approved at the March 24 meeting with the companies and NDEP. No change to the text was made.
15	- Sections 4.2.1 and 4.2.3	An exposure time (hrs/day) is not described. Please add discussion on exposure time for all receptor scenarios.	The requested text was added as the new Section 4.2.3.
16	- pp. 4-7 top	Please clarify CTE versus RME parameters and the relationship between the two.	See RTC #4.
17	- pp. 4-7 bottom	EnviroGiSdT does not force use of 1/2 DL. However, it is the default option. Please reword.	Text in Section 4.3.1 has been revised to clarify that 1/2 of the DL is the programs default option.
18	- pp. 4-8 last paragraph 2nd sentence	Replace the end of the sentence with "are defined for non-construction scenarios".	Text revised as per comment; 1st paragraph in Section 4.3.2.1.
19	- pp. 4-9 1st paragraph	Please note that the dust emissions modeling described here is PEF modeling. PEF is not mentioned in this paragraph, yet it is used immediately in the last paragraph of this page. Please include a sentence after the USEPA, 2002a citation to mention the PEF and direct the reader to Appendix D for details.	Text revised as per comment; 2nd paragraph in Section 4.3.2.1.
20	- pp. 4-9 last paragraph 4th sentence	The PEF equation does not depend on the exposure area so much as on the source area. Please clarify the relationship between source areas and exposure areas.	Text revised as per comment; 1st paragraph of Non-Construction Dust Emissions subsection (of Section 4.3.2.1).
21	- pp. 4-9 last sentence	Setting the vegetative cover at 0 seems overly conservative. This parameter is used to represent that there is no bare dirt, and can include paved parking lots, etc. It seems that using USEPA's default of 0.5 is reasonable. Also, note that use of a value of zero does not "optimize the potential for wind erosion emissions". Please reword. It artificially represents 100% bare ground, which supports a conservative estimate of wind erosion emissions.	In line with the agreement reached at the March 24 meeting with the companies and NDEP the text in Section 4.3.2.1 was revised to state that a value for the vegetative cover will be selected in the risk assessment.

NDEP COMMENT RESPONSE LOG, FEBRUARY 2010 COMMENTS			
RISK ASSESSMENT WORKPLAN FORMER MONTROSE AND STAUFFER FACILITIES HENDERSON, NEVADA			
Comment No.	Applicable Section/ Text	Comment	RTC
22	pp. 4-10 1st full para. 2nd last sent.	Please change "The dispersion factor equation provides estimates of the ambient air concentrations" to "The PEF model generates estimates of the ambient air concentrations".	The suggested text has been incorporated; 2nd paragraph of Non-Construction Dust Emissions subsection (of Section 4.3.2.1).
23	pp. 4-10 1st full para. Last sentence	Please change "dilution factor" to "attenuation factor".	The term has been revised as requested; 2nd paragraph of Non-Construction Dust Emissions subsection.
24	Section 4.3.2.1 pp. 4-11	In accordance with USEPA 2002, please define the "decision unit" as the exposure area.	The requested change has been made in Section 4.3.2.1. The change is in line with the terminology introduced in Section 1.2, and outlined in RTC#3.
25	pp. 4-14 Section 4.3.3	Please note that air EPCs for VOCs should be based on soil vapor or flux data when available. Groundwater or soil matrix are not acceptable for calculation of an air EPC.	The companies have agreed to move forward with assessing volatiles using soil vapor data that will be gathered via NDEP approved methodologies. Revisions have been made in Section 4.3.3 to reflect this approach consistent with recommendation in comment.
26	pp. 4-14 last line	Change SOP to SAP.	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
27	Pp. 4-15 Section 4.3.3.1 2nd paragraph 3rd sentence	(this comment applies in several places). The ASTM model that is referenced here (and see next paragraph) is not a dispersion model. It is a diffusion model that might be better characterized as a transport model (or box model).	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
28	pp. 4-15 Section 4.3.3.1 2nd paragraph last 2 sentence	The term "site assessment area" needs to be defined. To what area will the flux data be applied? Also, the maximum is a statistic that is very unreliable because of its strong dependence on sample size. Consideration of a 95% UCL as well as the maximum is worthwhile, but in both cases the area to which these statistics are defined needs to be made clearer. [RAKVICA] NDEP would like to discuss if the Companies are going to utilize BRC SOP 16 and 37 or develop their own. If the BRC SOPs are used many of these comments go away as noted below.	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
29	pp. 4-15 Section 4.3.3.1 3rd paragraph 2nd last sentence	It is not clear that it is refinement of "ambient air modeling" that is under consideration here. It is refinement of modeling indoor air concentrations from flux chamber data.	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
30	pp. 4-16 Section 4.3.3.2 2nd paragraph	Will the same building parameters be used in the CAL/EPA attenuation factors, the J&E attenuation factors, and the ASTM model used on the flux chamber data? [ADDITIONAL NOTE FROM B. RAKVICA] Addressed by BRC SOP 16 and 37	Text of 4.3.3.1 has been revised to clarify that building parameters will reflect site-specific values or defaults for commercial buildings based on ASTM (2000).
31	pp. 4-16 Section 4.3.3.2 3rd paragraph	The J&E model makes use of both diffusion and advection. Will the flux chamber data also be modeled to indoor air with a model that admits advection?	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
32	pp. 4-16 Section 4.3.3.2 4th paragraph	The USEPA J&E model is developed for a residential building. Will the building parameters be adjusted for a commercial building? If so, how?	Text of 4.3.3.1 has been revised to clarify that building parameters will reflect site-specific values or defaults for commercial buildings based on ASTM (2000).

NDEP COMMENT RESPONSE LOG, FEBRUARY 2010 COMMENTS			
RISK ASSESSMENT WORKPLAN FORMER MONTROSE AND STAUFFER FACILITIES HENDERSON, NEVADA			
Comment No.	Applicable Section/ Text	Comment	RTC
33	pp. 4-17 Section 4.3.3.2 4th paragraph	It is not clear that it will make sense to define such small areas to support the analysis. There will be less data in smaller areas. This brings up the question of how areas will be defined to support characterization and risk assessment. Some discussion of this upfront would be helpful. In general, the multiple lines of evidence approach, and the path forward for defining areas of interest needs to be described to put this in perspective. VI analysis is usually iterative, and the risk assessment areas and approach will depend on what is found during the iterative characterization stages of data collection	Text of 4.3.3 has been revised to indicate that source area soil gas data will be used to characterize area considered in the model.
34	pp. 4-17 2nd full para. 2nd sentence	The equation needs to be presented or the source cited where it is presented. There are many different forms of the equation. It does not appear to be discussed in Appendix E. [ADDITIONAL NOTE FROM B. RAKVICA] Addressed by SOP 16 and 37	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
35	Section 4.3.3.3 Soil Data	The text describes how soil matrix data will be used to estimate air EPCs for VOCs – this is a lower tier approach and not necessary when either soil vapor or flux data are available. Please clarify in text. [ADDITIONAL NOTE FROM B. RAKVICA] Addressed by SOP 16 and 37	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
36	pp. 4-17 Section 4.3.3.3 3rd sentence	Flux chamber and soil gas measurements are also not direct measurements. The medium of interest is indoor air. Neither of these methods measure indoor air. Instead they sample another medium that is then modeled to indoor air. This is not different than for soil data. [ADDITIONAL NOTE FROM B. RAKVICA] Addressed by SOP 16 and 37	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
37	Section 4.3.3.4 pp. 4-19 & 4-20 Groundwater Data	The text describes how groundwater data will be used to estimate air EPCs for VOCs – this is a lower tier approach and not necessary when either soil vapor or flux data are available. [ADDITIONAL NOTE FROM B. RAKVICA] Addressed by SOP 16 and 37	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
38	pp. 4-18 2nd half of 2nd paragraph	It is not clear why averaging time is being discussed here? There is no AT term in the VF equation (there is an exposure interval term). AT is relevant to all of the data with respect to risk assessment. The time period relevant to the model appears to be time relative to reaching equilibrium with an infinite source. As long as the exposure interval (or exposure duration) is greater than this, exposure duration does not matter. It also appears that exposure duration (ED) and averaging time (AT) terminology have been mixed up. AT is only relevant with respect to the health endpoint (e.g. cancer or non-cancer). It is suggested that AT be replaced with "time-weighted emission rate".	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
39	pp. 4-19 2nd paragraph 2nd sentence	A UCL is proposed for data that cover the building footprint (note comments on issues with this above), however a maximum is proposed as an alternative. Over what area will the maximum be taken?	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
40	pp. 4-19 Section 4.3.3.4 1st paragraph 3rd sentence	Where in the soil screening guidance is a model presented for the evaluation of soil emissions from contaminants in groundwater?	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
41	pp. 4-19 Section 4.3.3.4 last paragraph on page	It is not clear what this "dilution factor" refers to. What is the exact factor name in the soil screening guidance?	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.
42	pp. 4-20 last paragraph 1st sentence	It is not clear that there is any justification for using the maximum groundwater concentration. It is also not clear that it will make sense to only look at data below an (undefined) building footprint. Page 5-2, 1st (full) paragraph, 14th line.	In line with RTC#25 above, this text has been removed. Therefore this comment is no longer relevant.

NDEP COMMENT RESPONSE LOG, FEBRUARY 2010 COMMENTS			
RISK ASSESSMENT WORKPLAN FORMER MONTROSE AND STAUFFER FACILITIES HENDERSON, NEVADA			
Comment No.	Applicable Section/ Text	Comment	RTC
43	- Section 5.2 Approaches for Chemical Mixtures	Coplanar PCBs are mentioned in this section. Please also describe how Aroclor data will be evaluated.	The intent of this section is to describe the manner in which the toxicity of mixtures for which no RfD or CSF is available to calculate risks. In these cases the TEQ or RPF approach will be utilized. For aroclors, toxicity criteria derived for the aroclor mixture will be utilized, and therefore their treatment falls within the discussions within Section 5.1.1 and 5.1.2, Noncancer and Carcinogenic effects from chemical exposures. A footnote clarifying this fact was added to the beginning of Section 5.2. The revised language was reviewed and accepted at the March 24 meeting with the Companies and NDEP.
44	- pp. 5-6 Section 5.1.4 last sentence on page	It is not clear where this is discussed below. A subsection should be referenced.	Text in Section 5.1.5 was revised slightly to provide a clearer transition between the two paragraphs. The revised text was reviewed and agreed upon at the March 24 meeting with the Companies and NDEP.
45	- pp. 5-7 Section 5.2	PCBs should also be mentioned in this introduction paragraph.	Text clarifying the inclusion of dioxin like PCBs in the TEQ approach was added to the introductory paragraph of Section 5.2. The revised text was reviewed and agreed upon at the March 24 meeting with the Companies and NDEP.
46	- pp. 6-4	The section on lead toxicity should be moved to Chapter 5 and given its own subsection similar to asbestos. The default GSD from the model should be used unless robust population data from the site can be used to derive an appropriate GSD.	The text presented in Section 6.2 is appropriately placed as it does not describe the toxicity of lead, rather the process by which risk characterization will be completed for lead. The exposure parameters that will be used in the model are described in Section 4.1.3. These sections of text were not changed. However, in order to be consistent with the other SRCs presented in ch 5 and 6, text briefly describing the toxicity of lead has been added as Section 5.1.4.
47	- Chapter 5	Note also that there is no discussion of asbestos toxicity in Chapter 5.	The current text was reviewed at the March 24 meeting with the companies and NDEP. It was agreed that no change to the text is warranted.
48	- pp. 6-7	COPC selection is not obviously included in this list.	This item was added to the list of items that will be presented in the risk assessments in Section 6.8
49	- pp.6-7	The (final) Data Quality Assessment step is not included. DQA is needed to justify that enough samples have been taken to support the risk assessment in each case.	A DQA for risk drivers will be completed. Text describing this assessment has been added to Section 6 (as Section 6.6).
50	a Appendix C Tables	Inhalation, Offsite resident – ETi is set to 14 hrs/day, but should be 16.7 hrs/day.	In line with RTC#1 above, the offsite resident will no longer be evaluated quantitatively in the risk assessment. Therefore this comment is no longer relevant.
50	b Appendix C Tables	Equation C-1 – The units do not cancel. CF (if in numerator) should be kg/mg, as shown in the table.	This edit has been made.
50	c Appendix C Tables	Equation C-2 – The units do not cancel. As shown in Part A RAGS, SA should be in units of cm ² /event.	The equation is consistent with USEPA 2004 guidance on dermal contact (note EV accounts for the frequency of event). No change was made to the text.
50	d Appendix C Tables	Exposure Factor Table, Inhalation – The 0.4 indoor air dilution factor should be applicable only to particulates.	Tables have been clarified to show dilution factor only applies to indoor workers.

NDEP COMMENT RESPONSE LOG, FEBRUARY 2010 COMMENTS			
RISK ASSESSMENT WORKPLAN FORMER MONTROSE AND STAUFFER FACILITIES HENDERSON, NEVADA			
Comment No.	Applicable Section/ Text	Comment	RTC
50	e	Appendix C Tables Exposure Factor Table, Inhalation – The 0.4 indoor air dilution factor should not be applied for outdoor receptors.	Disagree. The equation utilized allows the term that this factor is included in to go to 0, when ETI (exposure time, indoors) is equal to 0 (such as for the outdoor worker and construction workers). The full terms were included for transparency. No changes to the tables were made.
50	f	Appendix C Tables Exposure Factor Table, Inhalation – 8 hours per day is quite a bit of time away from home for a pre-schooler. EPA's default residential scenario assumes less time away from home for children. Accordingly, the rationale for the 0-6 year old being gone 365 days per year for at least 8 hr (15 days per year totally gone and 350 days per year gone for 8 hours per day) should be provided.	In line with RTC#1 above, the offsite resident will no longer be evaluated quantitatively in the risk assessment. Therefore this comment is no longer relevant.
51	a	Appendix D Tables Page D-2, top – It is stated that the mean annual wind speed, UM, is based on observations from the National Weather Service station in Las Vegas, NV. Please provide the specific value to be used.	The specific wind speed to be used is 4.07 m/s (WRCC, 2010). This value has been added to the text. It is noted that all of the values for modeling parameters are included in the Appendix D table.
51	b	Appendix D Tables The text in this appendix refers to PEFs that will be calculated for "Site sources", "exposure areas" and "site assessment" areas. Please avoid the term "site assessment areas" unless it can be explained how they factor in to the determination of exposure areas. Exposure areas should be the basis for the PEFs and exposure areas should incorporate key CSM issues related to sources. The term "post-construction" is used in this appendix but this scenario is not specifically defined in the work plan. Please clarify.	Discussion revised to provide clarification on source area.
51	c	Appendix D Tables The Mean annual wind speed is given as: Table has it set to 3.9 m/s, but it should be 4.0 m/s.	The specific wind speed to be used is 4.07 m/s (WRCC, 2010). This value has been added to the appendix table.
51	d	Appendix D Tables Number of days with >= 0.01 inches of precipitation: Table has it set to 29 days/yr, but it should be 27 days/yr.	Table has been revised as per NDEP reference.
51	e	Appendix D Tables Wet soil bulk density: Table has it set to 2 Mg/m ³ , but it should be 1.74 Mg/m ³ .	Table has been revised to show additional significant digits as per NDEP request.
51	f	Appendix D Tables Mean dozing speed: Table has it set to 11 km/hr, but it should be 11.4 km/hr.	Table has been revised to show additional significant digits as per NDEP request.
51	g	Appendix D Tables Width of unpaved road segment: The table has this value set to 20 ft, but it should be 6.1 meters. It translates into the same thing, but for consistency it should be reported in meters.	The length in meters has been added as a parenthetical. This approach was agreed to at the March 24 meeting with the Companies and NDEP.
51	h	Appendix D Tables Length of unpaved road segment: The units for this parameter should be changed from feet to meters.	In line with RTC #51h above, the units of feet and meters are both shown.
52	a	Appendix E The way this appendix is written, all outdoor VOC EPCs will be based on soil matrix or groundwater data. If soil vapor data or flux chamber data are available, these are the preferred source terms for outdoor air assessment.	Text in Section 4.3 has been revised to indicate that soil gas data collected using an approved NDEP SOP will be used for vapor risk assessment. This clarification addressed the comment and eliminated the need for the Appendix.
52	b	Appendix E Page E-1, 2nd paragraph – Please reference Tables E-1 through E-3 for outdoor workers, offsite residents, and construction workers (outdoor air parameters) and Table E-4 for indoor workers (J & E model parameters).	Technical Appendix eliminated due to revised approach based on NDEP approved SOP for soil gas. Therefore this comment is no longer relevant.
52	c	Appendix E Paragraph under Equation 3, 1st sentence. This is only the case for non-carcinogens. Please clarify.	Text in Section 4.3.3.3 has been updated to clarify this terminology. As stated above, Appendix E was eliminated.
52	d	Appendix E VAPOR RELEASED FROM SOIL section, 2nd last paragraph, last sentence. Values relate to a residential building and will need to be changed for a commercial building. Please clarify.	Technical Appendix E was eliminated due to revised approach based on NDEP approved SOP for soil gas.
52	e	Appendix E VAPOR RELEASED FROM SOIL section, last paragraph, 1st sentence. Issue with definition of areas (similar to main text). Please clarify over which area UCLs will be calculated. This is a comment that applies to other parts of Appendix E.	Technical Appendix E was eliminated due to revised approach based on NDEP approved SOP for soil gas.

NDEP COMMENT RESPONSE LOG, FEBRUARY 2010 COMMENTS			
RISK ASSESSMENT WORKPLAN FORMER MONTROSE AND STAUFFER FACILITIES HENDERSON, NEVADA			
Comment No.	Applicable Section/ Text	Comment	RTC
52	f Appendix E	"Water-Filled Porosity" must be measured using ASTM D2216 and not calculated.	Comment noted and recommended approach will be followed. No revision to document as Technical Appendix E was eliminated due to revised approach based on NDEP approved SOP for soil gas.
52	g Appendix E	"Sample USCS Classification" – for risk assessment purposes the NDEP recommends soil classification based on sieve analysis following ASTM D422 and C117 (fines <200 sieve size).	Comment noted and recommended approach will be followed. No revision to document as Technical Appendix E was eliminated due to revised approach based on NDEP approved SOP for soil gas.
52	h Appendix E	Calculation of Site-Specific Fraction Organic Carbon (foc) – Neither PES nor Stauffer have indicated where these samples were taken in relation to on-site contamination. The NDEP has repeatedly indicated to PES/Stauffer that Foc samples must be taken from uncontaminated areas.	Comment noted and recommended approach will be followed. No revision to document as Technical Appendix E was eliminated due to revised approach based on NDEP approved SOP for soil gas.

APPENDIX B

SITE-RELATED CHEMICALS LIST AS
DETAILED IN HARGIS +
ASSOCIATES, INC. (2008)

Table B-1. Site-Related Chemical List.

Group	Chemical	CAS Number	Montrose	SMC	Water	Soil
			SRC ^a	SRC ^b		
ALDEHYDES	Acetaldehyde	75-07-0	✓		Y	Y
ALDEHYDES	Chloral	75-87-6	✓			
ALDEHYDES	Chloral Hydrate	302-17-0	✓		Y	Y
ALDEHYDES	Chloroacetaldehyde	107-20-0	✓		Y	Y
ALDEHYDES	Dichloroacetaldehyde	79-02-7	✓			
ALDEHYDES	Formaldehyde	50-00-0		✓	Y	Y
ASBESTOS	Asbestos	1332-21-4		✓		
CHEMICALS UNDER REVIEW	Total Petroleum Hydrocarbons	NA		✓	Y	Y
DIOXINS/FURANS	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	39001-02-0		✓	Y	Y
DIOXINS/FURANS	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	3268-87-9		✓	Y	Y
DIOXINS/FURANS	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	35822-46-9		✓	Y	Y
DIOXINS/FURANS	1,2,3,4,6,7,8-Heptachlorodibenzofuran	67562-39-4		✓	Y	Y
DIOXINS/FURANS	1,2,3,4,7,8,9-Heptachlorodibenzofuran	55673-89-7		✓	Y	Y
DIOXINS/FURANS	1,2,3,4,7,8-Hexachlorodibenzofuran	70648-26-9		✓	Y	Y
DIOXINS/FURANS	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	39227-28-6		✓	Y	Y
DIOXINS/FURANS	1,2,3,6,7,8-Hexachlorodibenzofuran	57117-44-9		✓	Y	Y
DIOXINS/FURANS	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	57653-85-7		✓	Y	Y
DIOXINS/FURANS	1,2,3,7,8,9-Hexachlorodibenzofuran	72918-21-9		✓	Y	Y
DIOXINS/FURANS	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	19408-74-3		✓	Y	Y
DIOXINS/FURANS	1,2,3,7,8-Pentachlorodibenzofuran	57117-41-6		✓	Y	Y
DIOXINS/FURANS	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	40321-76-4		✓	Y	Y
DIOXINS/FURANS	2,3,4,6,7,8-Hexachlorodibenzofuran	60851-34-5		✓	Y	Y
DIOXINS/FURANS	2,3,4,7,8-Pentachlorodibenzofuran	57117-31-4		✓	Y	Y
DIOXINS/FURANS	2,3,7,8-Tetrachlorodibenzofuran	51207-31-9		✓	Y	Y
DIOXINS/FURANS	2,3,7,8-Tetrachlorodibenzo-p-dioxin	1746-01-6		✓	Y	Y
GENERAL CHEMICAL	Total Dissolved Solids	NA	✓	✓	Y	Y
INDICATOR CHEMICAL	Alkalinity as CaCO ₃ [Sodium Hydroxide]	NA	✓		Y	Y
INDICATOR CHEMICAL	Ammonia-N ₂	7664-41-7	✓	✓	Y	Y
INDICATOR CHEMICAL	Chloride	16887-00-6	✓		Y	Y
INDICATOR CHEMICAL	Iron (Total)	7439-89-6	✓	✓	Y	Y
INDICATOR CHEMICAL	pH (in Soil)	NA	✓		Y	Y
INDICATOR CHEMICAL	pH (in Water)	NA	✓		Y	Y
INDICATOR CHEMICAL	Sodium	7440-23-5	✓	✓	Y	Y
INDICATOR CHEMICAL	Sulfate	14808-79-8	✓		Y	Y
INDICATOR CHEMICAL	Sulfur - total	63705-05-5	✓			
INDICATOR CHEMICAL	Total Phosphorus	7723-14-0	✓	✓	Y	Y
INORGANIC	Ammonium Chloride	12125-02-9	✓		Y	Y
INORGANIC	Bicarbonate	71-52-3		✓	Y	Y
INORGANIC	Calcium carbonate	471-34-1		✓	Y	Y
INORGANIC	Calcium hydroxides	1305-62-0		✓	Y	Y
INORGANIC	Chloride	16887-00-6	✓		Y	Y
INORGANIC	Chlorine	7782-50-5	✓	✓	Y	Y
INORGANIC	Cyanide	57-12-5	✓	✓	Y	Y
INORGANIC	Ferric Chloride	7705-08-0	✓		Y	Y
INORGANIC	Fluoride	16984-48-8	✓		Y	Y
INORGANIC	Graphite	7782-42-5		✓	Y	Y
INORGANIC	Hydrochloric Acid	7647-01-0	✓	✓	Y	Y
INORGANIC	Iodine	7553-56-2		✓	Y	Y
INORGANIC	Iodine chloride	7790-99-0		✓	Y	Y
INORGANIC	Magnesium hydroxide	1309-42-8		✓	Y	Y
INORGANIC	Magnesium oxide	1309-48-4		✓	Y	Y
INORGANIC	Nitrate	14797-55-8		✓	Y	Y
INORGANIC	Nitrogen Chloride	10025-85-1	✓	✓		
INORGANIC	Phosphonic Acid	7664-38-2	✓	✓	Y	Y
INORGANIC	Phosphorus pentasulfide	1314-80-3		✓	Y	Y
INORGANIC	Phosphorus Trichloride	7719-12-2	✓		Y	Y
INORGANIC	Sodium carbonate	497-19-8		✓	Y	Y
INORGANIC	Sodium chlorate	7775-09-9		✓	Y	Y
INORGANIC	Sodium chloride	7647-14-5		✓	Y	Y
INORGANIC	Sodium Hydroxide	1310-73-2	✓	✓	Y	Y
INORGANIC	Sodium Hypochlorite	7681-52-9	✓	✓	Y	Y
INORGANIC	Sodium salt of Diethyl phosphorodithioic acid	3338-24-7		✓		

Table B-1. Site-Related Chemical List.

Group	Chemical	CAS Number	Montrose	SMC	Water	Soil
			SRC ^a	SRC ^b		
INORGANIC	Sodium salt of Dimethyl phosphorodithioic acid	26377-29-7		✓		
INORGANIC	Sodium sulfate	7757-82-6		✓	Y	Y
INORGANIC	Sulfate	14808-79-8	✓		Y	Y
INORGANIC	Sulfuric Acid	7664-93-9	✓	✓	Y	Y
INORGANIC	Total Dissolved Solids	TDS		✓	Y	Y
INORGANIC	White phosphorus	12185-10-3		✓	Y	Y
METAL	Aluminum	7429-90-5	✓	✓	Y	Y
METAL	Antimony	7440-36-0	✓	✓	Y	Y
METAL	Arsenic	7440-38-2	✓	✓	Y	Y
METAL	Barium	7440-39-3	✓		Y	Y
METAL	Beryllium	7440-41-7	✓	✓	Y	Y
METAL	Cadmium	7440-43-9	✓	✓	Y	Y
METAL	Calcium	7440-70-2	✓	✓	Y	Y
METAL	Chromium (Total)	7440-47-3	✓	✓	Y	Y
METAL	Chromium VI (in Soil)	18540-29-9	✓	✓	Y	Y
METAL	Chromium VI (in Water)	18540-29-9	✓	✓	Y	Y
METAL	Cobalt	7440-48-4	✓	✓	Y	Y
METAL	Copper	7440-50-8	✓	✓	Y	Y
METAL	Iron (Total)	7439-89-6	✓	✓	Y	Y
METAL	Lead	7439-92-1	✓	✓	Y	Y
METAL	Magnesium	7439-95-4	✓	✓	Y	Y
METAL	Manganese	7439-96-5	✓	✓	Y	Y
METAL	Mercury [Mercury (in Soil)]	7439-97-6	✓	✓	Y	Y
METAL	Mercury [Mercury (in Water)]	7439-97-6	✓	✓	Y	Y
METAL	Molybdenum	7439-98-7	✓		Y	Y
METAL	Nickel	7440-02-0	✓	✓	Y	Y
METAL	Phosphorus	7723-14-0	✓	✓	Y	Y
METAL	Phosphorus, white	7723-14-0	✓	✓	Y	Y
METAL	Potassium	7440-09-7	✓	✓	Y	Y
METAL	Selenium	7782-49-2	✓	✓	Y	Y
METAL	Silver	7440-22-4	✓	✓	Y	Y
METAL	Sodium	7440-23-5	✓	✓	Y	Y
METAL	Sulfur, molecular	7704-34-9	✓		Y	Y
METAL	Thallium	7440-28-0	✓	✓	Y	Y
METAL	Tin	7440-31-5	✓	✓	Y	Y
METAL	Titanium	7440-32-6		✓	Y	Y
METAL	Vanadium	7440-62-2	✓	✓	Y	Y
METAL	Zinc	7440-66-6	✓	✓	Y	Y
ORGANIC ACIDS	4-Chlorobenzene Sulfonic Acid (pCBSA)	98-68-8	✓	✓		
ORGANIC ACIDS	Benzenesulfonic acid	98-11-3		✓		
ORGANIC ACIDS	Diethyl phosphorodithioic acid	298-06-6		✓		
ORGANIC ACIDS	Dimethyl phosphorodithioic acid	756-80-9		✓		
ORGANIC ACIDS	Phthalic acid	88-99-3		✓		
PCB	2,2'-Dichlorobiphenyl	13029-08-8	✓		Y	
PCB	2,3'-Dichlorobiphenyl	25569-80-6	✓		Y	
PCB	2,3-Dichlorobiphenyl	16605-91-7	✓		Y	
PCB	2,4'-Dichlorobiphenyl	34883-43-7	✓		Y	
PCB	2,4-Dichlorobiphenyl	33284-50-3	✓		Y	
PCB	2,5-Dichlorobiphenyl	34883-39-1	✓		Y	
PCB	2,6-Dichlorobiphenyl	33146-45-1	✓		Y	
PCB	3,3'-Dichlorobiphenyl	2050-67-1	✓		Y	
PCB	3,4'-Dichlorobiphenyl	2974-90-5	✓		Y	
PCB	3,4-Dichlorobiphenyl	2974-92-7	✓		Y	
PCB	3,5-Dichlorobiphenyl	34883-41-5	✓		Y	
PCB	4,4'-Dichlorobiphenyl	2050-68-2	✓		Y	
PCB	Aroclor 1016	12674-11-2	✓	✓	Y	Y
PCB	Aroclor 1221	11104-28-2	✓	✓	Y	Y
PCB	Aroclor 1232	11141-16-5	✓	✓	Y	Y
PCB	Aroclor 1242	53469-21-9	✓	✓	Y	Y
PCB	Aroclor 1248	12672-29-6	✓	✓	Y	Y
PCB	Aroclor 1254	11097-69-1	✓	✓	Y	Y
PCB	Aroclor 1260	11096-82-5	✓	✓	Y	Y

Table B-1. Site-Related Chemical List.

Group	Chemical	CAS Number	Montrose	SMC	Water	Soil
			SRC ^a	SRC ^b		
PCB	PCB 077	32598-13-3	✓	✓	Y	
PCB	PCB 081	70362-50-4	✓	✓	Y	
PCB	PCB 105	32598-14-4	✓	✓	Y	
PCB	PCB 114	74472-37-0	✓	✓	Y	
PCB	PCB 118	31508-00-6	✓	✓	Y	
PCB	PCB 123	65510-44-3	✓	✓	Y	
PCB	PCB 126	57465-28-8	✓	✓	Y	
PCB	PCB 156	38380-08-4	✓	✓	Y	
PCB	PCB 157	69782-90-7	✓	✓	Y	
PCB	PCB 167	52663-72-6	✓	✓	Y	
PCB	PCB 169	32774-16-6	✓	✓	Y	
PCB	PCB 189	39635-31-9	✓	✓	Y	
PESTICIDE	2,4'-DDD	53-19-0	✓	✓		
PESTICIDE	2,4'-DDE	3424-82-6	✓	✓		
PESTICIDE	2,4'-DDT	789-02-6	✓			
PESTICIDE	4,4'-DDD	72-54-8	✓	✓	Y	Y
PESTICIDE	4,4'-DDE	72-55-9	✓	✓	Y	Y
PESTICIDE	4,4'-DDT	50-29-3	✓	✓	Y	Y
PESTICIDE	A-BHC	319-84-6	✓	✓	Y	Y
PESTICIDE	Aldrin	309-00-2	✓	✓	Y	Y
PESTICIDE	B-BHC	319-85-7	✓	✓	Y	Y
PESTICIDE	Carbophenothion	786-19-6	✓	✓		
PESTICIDE	D-BHC	319-86-8	✓		Y	Y
PESTICIDE	Dieldrin	60-57-1	✓	✓	Y	Y
PESTICIDE	Endosulfan I	959-98-8	✓	✓	Y	Y
PESTICIDE	Endosulfan Sulfate	1031-07-8	✓	✓	Y	Y
PESTICIDE	Endrin	72-20-8	✓	✓	Y	Y
PESTICIDE	Endrin Aldehyde	7421-93-4	✓	✓	Y	Y
PESTICIDE	G-BHC	58-89-9	✓	✓	Y	Y
PESTICIDE	Heptachlor Epoxide	1024-57-3	✓	✓	Y	Y
PESTICIDE	Methyl-carbophenothion	953-17-3		✓		
PESTICIDE	Phosmet	732-11-6		✓		
SVOC	1-(4-chlorophenyl)-1-propanone	6285-05-8	✓			
SVOC	1,1'-Sulfonybis benzene	127-63-9		✓		
SVOC	1,2,3,4-Tetrachlorobenzene	634-66-2	✓	✓		
SVOC	1,2,4,5-Tetrachlorobenzene	95-94-3	✓	✓		
SVOC	1,2-Diphenyl hydrazine	122-66-7	✓			
SVOC	1-chloro-4-(methylsulfonyl) benzene	98-57-7		✓		
SVOC	2,2'-Dichlorobenzil	21854-95-5	✓		Y	Y
SVOC	2,4,5-Trichlorophenol	95-95-4	✓	✓	Y	Y
SVOC	2,4,6-Trichlorophenol	88-06-2	✓	✓		
SVOC	2,4-Dichlorophenol	120-83-2	✓	✓	Y	Y
SVOC	2,4-Dimethylphenol	105-67-9		✓	Y	Y
SVOC	2-Chlorophenol	95-57-8	✓	✓	Y	Y
SVOC	4,4'-Dichlorobenzil	3457-46-3	✓			
SVOC	4-Chloro-3-methylphenol	35421-08-0	✓		Y	Y
SVOC	4-Chlorophenyl methyl sulfide	123-09-1		✓	Y	Y
SVOC	4-Nitrophenol	100-02-7	✓			
SVOC	Benzo(a)anthracene	56-55-3		✓		
SVOC	Benzo(a)pyrene	50-32-8	✓	✓		
SVOC	Benzo(b)fluoranthene	205-99-2		✓	Y	Y
SVOC	Benzo(g,h,i)perylene	191-24-2		✓	Y	Y
SVOC	Benzo(k)fluoranthene	207-08-9		✓	Y	Y
SVOC	Benzoic Acid	65-85-0	✓	✓		
SVOC	Benzophenone	119-61-9		✓		
SVOC	Bis(2-ethylhexyl)phthalate	117-81-7	✓	✓		
SVOC	Bis-(chloromethyl)ether	542-88-1		✓	Y	Y
SVOC	Bis(p-chlorophenyl)disulfide	1142-19-4		✓		
SVOC	Chlorobenzenethiol	106-54-7		✓		
SVOC	Chrysene	218-01-9	✓	✓	Y	Y
SVOC	Dibenzo(a,h)anthracene	53-70-3		✓	Y	Y
SVOC	Diethyl phthalate	84-66-2		✓	Y	Y

Table B-1. Site-Related Chemical List.

Group	Chemical	CAS Number	Montrose	SMC	Water	Soil
			SRC ^a	SRC ^b		
SVOC	Di-n-butyl phthalate	84-74-2	✓	✓	Y	Y
SVOC	Di-n-octyl phthalate	117-84-0		✓		
SVOC	Fluoranthene	206-44-0	✓	✓	Y	Y
SVOC	Hexachlorobenzene	118-74-1	✓	✓	Y	Y
SVOC	Hexachloroethane	67-72-1		✓	Y	Y
SVOC	Indeno(1,2,3-cd)pyrene	193-39-5		✓		
SVOC	N-Hydroxymethylphthalimide	118-29-6		✓		
SVOC	Octachlorostyrene	29082-74-4	✓	✓		
SVOC	p-Chlorophenyl sulfone	80-07-9		✓		
SVOC	Pentachlorobenzene	608-93-5	✓	✓		
SVOC	Pentachlorophenol	87-86-5	✓		Y	Y
SVOC	Phenanthrene	85-01-8		✓	Y	Y
SVOC	Phenol (Total)	108-95-2	✓	✓	Y	Y
SVOC	Phenyl disulfide	882-33-7		✓		
SVOC	Phenyl sulfide	139-66-2		✓		
SVOC	Pyrene	129-00-0	✓	✓	Y	Y
SVOC	Pyridine	110-86-1		✓	Y	Y
SVOC	Thiophenol	108-98-5		✓		
TIC	1,1'-Thiobis [4-Chloro]Benzene	5181-10-2		✓		
TIC	1,2,3,5-Tetrachlorobenzene	634-90-2	✓	✓		
TIC	1,2,3-Trimethylbenzene	526-73-8		✓		
TIC	1,2,4-Trithiolane	289-16-7		✓		
TIC	1,5-Dichloroanthracene	6406-96-8	✓			
TIC	1,8-Dichloroanthracene	14381-66-9	✓			
TIC	1-Nitropropane	108-03-2	✓			
TIC	2,2,2-Trichloroethanol	115-20-8	✓			
TIC	2,3-Dichloroanthracene	613-07-0	✓			
TIC	2,3-Dichlorostyrene	213-28-6	✓			
TIC	2,4-Dichlorostyrene	21-27-5	✓			
TIC	2,5-Dichlorostyrene	1123-84-8	✓			
TIC	2,6-dichlorostyrene	28469-92-3	✓			
TIC	2-Chloro benzenethiol	6320-03-2		✓		
TIC	2-Chlorobenzyl chloride	611-19-8	✓			
TIC	2-Nitropropane	79-46-9	✓			
TIC	3,4-dichlorostyrene	2039-83-0	✓			
TIC	3,5-Heptanedione, 2,6-Dimethyl	18362-64-6		✓		
TIC	3-Chloro-Benzenethiol	2037-31-2		✓		
TIC	3-Chlorobenzyl chloride	620-20-2	✓			
TIC	3-Hexene-2,5-Dione (cis and trans)	4436-75-3		✓		
TIC	4-Chloro benzoylchloride	122-01-0		✓		
TIC	4-Chlorobenzaldehyde	104-88-1		✓		
TIC	4-Chloro-benzoic Acid	26264-09-5		✓		
TIC	4-Chlorobenzyl chloride	104-83-6	✓			
TIC	9,10-Dichloroanthracene	605-48-1	✓			
TIC	Alkane	NA	✓			
TIC	Alkyl Alkane	NA	✓			
TIC	Benzenesulfinothioic acid, phenylester	1208-20-4		✓		
TIC	bis-(2-chlorophenylmethanone)	NA	✓			
TIC	bis-(3-chlorophenylmethanone)	NA	✓			
TIC	bis-(4-chlorophenylmethanone)	90-98-2	✓	✓		
TIC	Chloroalkylbenzene	NA	✓			
TIC	Chloro-iodo-Benzene	615-41-8		✓		
TIC	Chloromethyl phthalimide	17564-64-6		✓		
TIC	Cyclododecene (CDEN)	1501-82-2	✓			
TIC	Ethyl Ether	60-29-7	✓			
TIC	Heptachlorostyrene	61255-81-0	✓			
TIC	Hexachlorostyrene	61128-00-5	✓			
TIC	Isoheptane	31394-54-4		✓		
TIC	Methanone, (3-Chlorophenyl)(4-Chlorophenyl)	7498-66-0		✓		
TIC	Methylsulfinyl benzene	1193-82-4		✓		
TIC	Methylthio-Benzene	100-68-5		✓		
TIC	O,O,A-Trimethylester phosphorodithioic acid	2953-29-9		✓		

Table B-1. Site-Related Chemical List.

Group	Chemical	CAS Number	Montrose	SMC	Water	Soil
			SRC ^a	SRC ^b		
TIC	Octasulfur	10544-50-0		✓		
TIC	Paraformaldehyde	30525-89-4		✓		
TIC	Pentachlorocyclohexane	22138-39-2	✓			
TIC	Pentachlorostyrene	83484-75-7	✓			
TIC	Phthalimide	85-41-6		✓		
TIC	Polyethylene Glycol	25322-68-3	✓			
TIC	Sodium thiophenate	930-69-8		✓		
TIC	Sulfenone	80-00-2		✓		
TIC	Tetrachlorocyclohexane	129-00-0	✓	✓		
TIC	Tetrachlorostyrene	NA	✓			
TIC	Tetrachlorothiophene	6012-97-1		✓		
TIC	Trichlorostyrene	NA	✓			
TIC	Unknown	NA	✓			
TIC	Unknown Brominated Hydrocarbon	NA	✓			
TIC	Unknown Chlorinated Aromatics	NA	✓			
TIC	Unknown Chlorinated Benzene	NA	✓			
TIC	Unknown Chlorinated Compound	NA	✓			
TIC	Unknown Chlorinated Hydrocarbon	NA	✓			
TIC	Unknown Chlorinated Ketone	NA	✓			
TIC	Unknown Hydrocarbon	NA	✓			
TIC	Unknown Organic Acid	NA	✓			
VOC	1,1,1,2-Tetrachloroethane	630-20-6	✓		Y	Y
VOC	1,1,2,2-Tetrachloroethane	79-34-5	✓		Y	Y
VOC	1,1,2-Trichloroethane	79-00-5	✓		Y	Y
VOC	1,1-Dichloroethane	75-34-3	✓		Y	Y
VOC	1,1-Dichloroethene	75-35-4	✓		Y	Y
VOC	1,1-Dichloropropene	563-58-6	✓			
VOC	1,2,3-Trichlorobenzene	87-61-6	✓	✓	Y	Y
VOC	1,2,3-Trichloropropane	96-18-4	✓			
VOC	1,2,4-Trichlorobenzene	120-82-1	✓	✓		
VOC	1,2,4-Trimethylbenzene	95-63-6	✓	✓		
VOC	1,2-Dichlorobenzene	95-50-1	✓	✓	Y	Y
VOC	1,2-Dichloroethane	107-06-2	✓		Y	Y
VOC	1,3,5-Trichlorobenzene	108-70-3	✓	✓		
VOC	1,3,5-Trimethylbenzene	108-67-8	✓	✓		
VOC	1,3-Dichlorobenzene	541-73-1	✓	✓	Y	Y
VOC	1,3-Dichloropropane	142-28-9	✓			
VOC	1,4-Dichlorobenzene	108-46-7	✓	✓	Y	Y
VOC	1-Methylethylbenzene	98-82-8	✓		Y	Y
VOC	2,2,3-Trimethylbutane	464-06-2		✓		
VOC	2,2-Dimethylpentane	590-35-2		✓		
VOC	2,3-Dimethylpentane	565-59-3		✓		
VOC	2,4-Dimethylpentane	108-08-7		✓		
VOC	2-Butanone	78-93-3	✓		Y	Y
VOC	2-Chlorotoluene	95-49-8	✓			
VOC	2-Hexanone	591-78-6	✓		Y	Y
VOC	2-Methylhexane	591-76-4		✓		
VOC	3,3-Dimethylpentane	562-49-2		✓		
VOC	3-Chlorobenzoic Acid	535-80-8	✓			
VOC	3-Ethylpentane	617-78-7		✓		
VOC	3-Methylhexane	589-34-4		✓		
VOC	4-Chlorobenzoic Acid	74-11-3	✓			
VOC	4-Chlorotoluene	106-43-4	✓			
VOC	4-methyl-2-pentanone	108-10-1	✓		Y	Y
VOC	Acetone	67-64-1	✓	✓	Y	Y
VOC	Benzene	71-43-2	✓	✓	Y	Y
VOC	Bromobenzene	108-86-1	✓		Y	Y
VOC	Bromochloromethane	74-97-5	✓		Y	Y
VOC	Bromoform	75-25-2	✓		Y	Y
VOC	Carbon disulfide	75-15-0		✓		
VOC	Carbon Tetrachloride	56-23-5	✓	✓	Y	Y
VOC	Chlorobenzene	108-90-7	✓	✓	Y	Y
VOC	Chloroform	67-66-3	✓	✓	Y	Y

Table B-1. Site-Related Chemical List.

Group	Chemical	CAS Number	Montrose	SMC	Water	Soil
			SRC ^a	SRC ^b		
VOC	Chloromethane	74-87-3	✓		Y	Y
VOC	cis-1,2-Dichloroethene	156-59-2	✓		Y	Y
VOC	Dibromochloroethane	73506-94-2	✓			
VOC	Dibromochloromethane	124-48-1	✓		Y	Y
VOC	Dibromochloropropane (DBCP)	98-12-8	✓		Y	Y
VOC	Dichlorodifluoromethane	75-71-8	✓		Y	Y
VOC	Dimethyldisulfide	624-92-0		✓		
VOC	Ethyl Alcohol (Ethanol)	64-17-5	✓	✓		
VOC	Ethyl Chloride	75-00-3	✓		Y	Y
VOC	Ethylbenzene	100-41-4	✓		Y	Y
VOC	Hexachlorobutadiene	87-68-3	✓	✓	Y	Y
VOC	Isopropyl toluene	25155-15-1	✓			
VOC	m,p-Xylene	136777-61-2		✓	Y	Y
VOC	Methanol (Methyl alcohol)	67-56-1		✓		
VOC	Methylene Chloride	75-09-2	✓	✓	Y	Y
VOC	Naphthalene	91-20-3	✓	✓	Y	Y
VOC	n-Butyl benzene	104-51-8	✓			
VOC	n-Heptane	142-82-5		✓		
VOC	Nonanal	124-19-6		✓		
VOC	n-Propyl benzene	103-65-1	✓			
VOC	Rubber hydrocarbon solvent	64475-85-0	✓		Y	Y
VOC	sec-Butyl benzene	135-98-8	✓			
VOC	Styrene	100-42-5	✓			
VOC	tert-Butyl benzene	98-06-6	✓			
VOC	Tetrachloroethene (PCE)	127-18-4	✓		Y	Y
VOC	Toluene	108-88-3	✓	✓	Y	Y
VOC	trans-1,2-Dichloroethene	156-60-5	✓		Y	Y
VOC	trans-1,3-Dichloropropene	10061-02-6	✓		Y	Y
VOC	Trichloroethene (TCE)	79-01-6	✓		Y	Y
VOC	Trichlorofluoromethane	75-69-4	✓		Y	Y
VOC	Vinyl chloride	75-01-4	✓		Y	Y
VOC	Xylene (o)	1330-20-7	✓	✓	Y	Y
VOC	Xylenes (m,p)	1330-20-7	✓	✓	Y	Y

Source: Hargis. 2008. Conceptual site model, former Montrose and Stauffer facilities and downgradient areas to Las Vegas Wash, Henderson, Clark County, Nevada. Revision 1.0. DRAFT. Prepared for Montrose Chemical Corporation of California, Stauffer Management Company, LLC., Syngenta Crop Protection, Inc., and Pioneer Americas, LLC. Hargis + Associates, Inc.

- Notes:
- CAS = chemical abstract service
 - NA = not applicable, due to impracticable analysis or certification does not exist
 - PCB = polychlorinated biphenyl
 - SRC = site-related chemical
 - SMC = Stauffer Management Company
 - SVOC = semi-volatile organic compound
 - TIC = tentatively identified compound
 - VOC = volatile organic compound
 - Y = yes
 - ✓ = applicable

^a List of Montrose SRCs approved by NDEP. 2006. Personal communication (letter to J. Kelly, Montrose Chemical Corporation of California, Bainbridge Island, WA, dated July 26, 2006, regarding NDEP's approval of the Montrose SRC list). Nevada Division of Environmental Protection.

^b List of Stauffer SRCs approved by NDEP. 2006. Personal communication (letter to G. Crouse, Syngenta Crop Protection, Inc., Greensboro, NC, L. Erickson, Stauffer Management Company, Golden, CO, and C. Sylvia, Pioneer Americas LLC, Henderson, NV, dated June 5, 2006, regarding NDEP's approval of the Stauffer SRC list). Nevada Division of Environmental Protection.

APPENDIX C

EXPOSURE EQUATIONS AND ASSUMPTIONS

EXHIBIT C-1. INCIDENTAL INGESTION OF CHEMICALS IN SOIL

$$LADD_{soil}, ADD_{soil} = \frac{C_{soil} \times IR_{soil} \times CF \times EF \times ED}{AT \times BW} \quad \text{Eq. C-1}$$

where,

- LADD_{soil} = lifetime average daily dose from incidental ingestion of soil (mg/kg-day)
- ADD_{soil} = average daily dose from incidental ingestion of soil (mg/kg-day)
- C_{soil} = concentration of chemical in soil (mg/kg)
- IR_{soil} = soil ingestion rate (mg/day)
- CF = unit conversion factor (kg/mg)
- EF = exposure frequency for soil (days/year)
- ED = exposure duration (years)
- AT = averaging time (days)
- BW = body weight (kg).

Parameter Values

Exposure Factor	Value		Units	Source
	CTE	RME		
Indoor Worker				
IR _{soil}	50	50	mg/day	USEPA (2002a), Exhibit 4-1
CF	1E-06	1E-06	kg/mg	Unit conversion factor
EF	250	250	days/year	USEPA (2002a), Exhibit 4-1
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a), Exhibit 4-1
AT _{nc}	2,555	9,125	days	ED x 365 days/year
AT _c	25,550	25,550	days	70 years x 365 days/year
BW	70	70	kg	USEPA (2002a), Exhibit 4-1
Outdoor Worker				
IR _{soil}	50	100	mg/day	USEPA (1991); USEPA (2002a), Exhibit 4-1
CF	1E-06	1E-06	kg/mg	Unit conversion factor
EF	225	225	days/year	USEPA (2002a), Exhibit 4-1
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a), Exhibit 4-1

Exposure Factor	Value		Units	Source
	CTE	RME		
AT _{nc}	2,555	9,125	days	ED x 365 days/year
AT _c	25,550	25,550	days	70 years x 365 days/year
BW	70	70	kg	USEPA (2002a), Exhibit 4-1
Construction Worker				
IR _{soil}	100	330	mg/day	BPJ; USEPA (2002a), Exhibit 5-1
CF	1E-06	1E-06	kg/mg	Unit conversion factor
EF	250	250	days/year	USEPA (2002a), Exhibit 5-1
ED	0.5	1	years	BPJ; USEPA (2002a), Exhibit 5-1
AT _{nc}	183	365	days	ED x 365 days/year
AT _c	25,550	25,550	days	70 years x 365 days/ year
BW	70	70	kg	USEPA (2002a), Exhibit 5-1

EXHIBIT C-2. DERMAL CONTACT WITH CHEMICALS IN SOIL

$$LDAD_{soil}, DAD_{soil} = \frac{DA_{event} \times SA \times EF \times ED \times EV}{AT \times BW} \quad \text{Eq. C-2}$$

where,

LDAD _{soil}	=	lifetime dermal absorbed dose (mg/kg-day)
DAD _{soil}	=	dermal absorbed dose (mg/kg-day)
DA _{event}	=	absorbed dose per event (mg/cm ²)
SA	=	skin surface area available for contact (cm ²)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
EV	=	event frequency (day ⁻¹)
AT	=	averaging time (days)
BW	=	body weight (kg)

and,

$$DA_{event} = C_{soil} \times CF \times AF \times ABS_d \quad \text{Eq. C-3}$$

where,

DA _{event}	=	absorbed dose per event (mg/cm ²)
C _{soil}	=	concentration of chemical in soil (mg/kg)
CF	=	unit conversion factor (kg/mg)
AF	=	adherence factor (mg/cm ²)
ABS _d	=	dermal absorption fraction, chemical-specific (unitless).

Parameter Values

Exposure Factor	Value		Units	Source
	CTE	RME		
Outdoor Worker				
AF	0.02	0.2	mg/cm ²	USEPA (2004a), Exhibit 3-5
ABS _d	Chemical specific		unitless	–
SA	3,300	3,300	cm ²	USEPA (2004a), Exhibit 3-5 (Assumes short sleeved shirt, pants, and shoes are worn)
EF	225	225	days/year	USEPA (2002a), Exhibit 4-1
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a), Exhibit 4-1

Exposure Factor	Value		Units	Source
	CTE	RME		
EV	1	1	day ⁻¹	USEPA (2004a), Exhibit 3-5
AT _{nc}	2,555	9,125	days	ED x 365 day/year
AT _c	25,550	25,550	days	70 years x 365 days/year
BW	70	70	kg	USEPA (2002a), Exhibit 4-1
CF	1E-06	1E-06	kg/mg	Unit conversion factor
Construction Worker				
AF	0.1	0.3	mg/cm ²	USEPA (2004a), Exhibit 3-3 (Study on construction workers)
ABS _d	Chemical specific		unitless	--
SA	3,300	3,300	cm ²	USEPA (2004a), Exhibit 3-5 (Assumes short sleeved shirt, pants, and shoes are worn)
EF	250	250	days/year	USEPA (2002a), Exhibit 5-1
ED	0.5	1	years	BPJ; USEPA (2002a), Exhibit 5-1
EV	1	1	day ⁻¹	USEPA (2004a), Exhibit 3-5
AT _{nc}	183	365	days	ED x 365 day/year
AT _c	25,550	25,550	days	70 years x 365 days/year
BW	70	70	kg	USEPA (2002a), Exhibit 5-1
CF	1E-06	1E-06	kg/mg	Unit conversion factor

EXHIBIT C-3. INHALATION OF INDOOR AND OUTDOOR AIR (CHEMICAL PARTICULATES, VOLATILES, AND ASBESTOS)

$$EC = \frac{C_{air} \times [ET_o + (ET_i \times DF_i)] \times EF \times ED}{AT} \quad \text{Eq. C-4}$$

where,

- EC = exposure concentration ($\mu\text{g}/\text{m}^3$, f/cm^3)
- C_{air} = chemical concentration in air ($\mu\text{g}/\text{m}^3$, f/cm^3)
- ET_i = exposure time indoors (hours/day)
- ET_o = exposure time outdoors (hours/day)
- DF_i = particulate dilution factor for outdoor to indoor air (unitless)
- EF = exposure frequency (days/year)
- ED = exposure duration (years)
- AT = averaging time (hours).

Parameter Values

Exposure Factor	Value		Units	Source
	CTE	RME		
Indoor Worker				
ET_i	8	8	hours/day	Assumed as work day duration
ET_o	0	0	hours/day	Entire work day is assumed to occur indoors
DF_i	0.4	0.4	unitless	BRC et al. (2009)
EF	250	250	days/year	USEPA (2002a), Exhibit 4-1
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a); Exhibit 4-1
AT_{nc}	61,320	219,000	hours	$ED \times 365 \text{ day/year} \times 24 \text{ hours/day}$
AT_c	613,200	613,200	hours	$70 \text{ years} \times 365 \text{ days/year} \times 24 \text{ hours/day}$
Outdoor Worker				
ET_i	0	0	hours/day	Entire work day is assumed to occur outdoors
ET_o	8	8	hours/day	Assumed as work day duration
DF_i	NA	NA	NA	NA
EF	225	225	days/year	USEPA (2002a), Exhibit 4-1
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a),

Exposure Factor	Value		Units	Source
	CTE	RME		
				Exhibit 4-1
AT _{nc}	61,320	219,000	hours	ED x 365 day/year x 24 hours/day
AT _c	613,200	613,200	hours	70 years x 365 days/year x 24 hours/day
Construction Worker				
ET _i	0	0	hours/day	Entire work day is assumed to occur outdoors
ET _o	8	8	hours/day	Assumed as work day duration
DF _i	NA	NA	NA	NA
EF	250	250	days/year	USEPA (2002a), Exhibit 5-1
ED	0.5	1	years	BPJ; USEPA (2002a), Exhibit 5-1
AT _{nc}	4,392	8,760	hours	ED x 365 day/year x 24 hours/day
AT _c	613,200	613,200	hours	70 years x 365 days/year x 24 hours/day

NA = not applicable.

EXHIBIT C-4. INCIDENTAL INGESTION OF RADIONUCLIDES IN SOIL

$$Dose_{soil} = C_{soil} \times IR_{soil} \times EF \times ED \times CF \qquad \text{Eq. C-5}$$

where,

- Dose_{soil} = internal dose from incidental ingestion of soil (pCi)
- C_{soil} = concentration of radionuclide in soil (pCi/g)
- IR_{soil} = soil ingestion rate (mg/day)
- EF = exposure frequency (days/year)
- ED = exposure duration (years)
- CF = unit conversion factor (g/mg).

Parameter Values

Exposure Factor	Value		Units	Source
	CTE	RME		
Indoor Worker				
IR _{soil}	50	50	mg/day	USEPA (2002a), Exhibit 4-1
EF	250	250	days/year	USEPA (2002a), Exhibit 4-1
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a); Exhibit 4-1
CF	1E-03	1E-03	g/mg	Unit conversion factor
Outdoor Worker				
IR _{soil}	50	100	mg/day	USEPA (1991); USEPA (2002a), Exhibit 4-1
EF	225	225	days/year	USEPA (2002a), Exhibit 4-1
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a); Exhibit 4-1
CF	1E-03	1E-03	g/mg	Unit conversion factor
Construction Worker				
IR _{soil}	100	330	mg/day	BPJ; USEPA (2002a), Exhibit 5-1
EF	250	250	days/year	USEPA (2002a), Exhibit 5-1
ED	0.5	1	years	BPJ; USEPA (2002a), Exhibit 5-1
CF	1E-03	1E-03	g/mg	Unit conversion factor

EXHIBIT C-5. INHALATION OF RADIONUCLIDES IN INDOOR AND OUTDOOR AIR PARTICULATES

$$Dose_{inhal} = C_{air} \times InhR \times [ET_o + (ET_i \times DF_i)] \times EF \times ED \quad \text{Eq. C-6}$$

where,

- Dose_{inhal} = internal dose from inhalation (pCi)
- C_{air} = concentration of radionuclide in air (pCi/m³)
- InhR = inhalation rate (m³/day)
- ET_o = exposure time outdoors (hours/day)
- ET_i = exposure time indoors (hours/day)
- DF_i = particulate dilution factor for outdoor to indoor air (unitless)
- EF = exposure frequency (days/year)
- ED = exposure duration (years).

Parameter Values

Exposure Factor	Value		Units	Source
	CTE	RME		
Indoor Worker				
InhR	13	13	m ³ /day	USEPA (1997a), Table 5-23 (Assumes moderate activity, 8 hours/day)
ET _o	0	0	hours/day	Entire work day is assumed to occur indoors
ET _i	8	8	hours/day	Assumed as work day duration
DF _i	0.4	0.4	unitless	BRC et al. (2009)
EF	250	250	days/year	USEPA (2002a), Exhibit 4-1
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a); Exhibit 4-1
Outdoor Worker				
InhR	20	20	m ³ /day	USEPA (1997a), Table 5-23 (Assumes heavy activity, 8 hours/day)
ET _o	8	8	hours/day	Assumed as work day duration
ET _i	0	0	hours/day	Entire work day is assumed to occur outdoors
DF _i	NA	NA	NA	NA
EF	225	225	days/year	USEPA (2002a), Exhibit 4-1
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a);

Exposure Factor	Value		Units	Source
	CTE	RME		
				Exhibit 4-1
Construction Worker				
InhR	20	20	m ³ /day	USEPA (1997a), Table 5-23 (Assumes heavy activity, 8 hours/day)
ET _o	8	8	hours/day	Assumed as work day duration
ET _i	0	0	hours/day	Entire work day is assumed to occur outdoors
DF _i	NA	NA	NA	NA
EF	250	250	days/year	USEPA (2002a), Exhibit 5-1
ED	0.5	1	years	BPJ; USEPA(2002a), Exhibit 5-1

NA = not applicable.

EXHIBIT C-6. EXTERNAL EXPOSURE FROM RADIONUCLIDES

$$Dose_{ext} = C_{soil} \times [EF/CF_{DY}] \times ED \times ACF \times [ET_{fo} + (ET_{fi} \times GSF)] \quad \text{Eq. C-7}$$

where,

- Dose_{ext} = dose from external exposure (pCi-yr/g)
- C_{soil} = concentration of radionuclide in soil (pCi/g)
- EF = exposure frequency (days/year)
- CF_{DY} = conversion factor (days/year)
- ED = exposure duration (years)
- ACF = area correction factor (unitless)
- ET_{fo} = fraction of time spent outdoors (unitless)
- ET_{fi} = fraction of time spent indoors (unitless)
- GSF = gamma shielding factor (unitless).

Parameter Values

Exposure Factor	Value		Units	Source
	CTE	RME		
Indoor Worker				
EF	250	250	days/year	USEPA (2002a), Exhibit 4-1
CF _{DY}	365	365	days/year	Conversion factor
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a); Exhibit 4-1
ACF	Specific to risk assessment study area		unitless	–
ET _{fi}	0.33	0.33	unitless	USEPA (2009b), Table 1 (Assumes 100% of 8 hour work day)
ET _{fo}	0	0	unitless	USEPA (2009b), Table 1
GSF	0.4	0.4	unitless	USEPA (2009b), Table 1
Outdoor Worker				
EF	225	225	days/year	USEPA (2002a), Exhibit 4-1
CF _{DY}	365	365	days/year	Conversion factor
ED	7	25	years	USEPA (1997a), Table 1-2; USEPA (2002a); Exhibit 4-1
ACF	Specific to risk assessment study		unitless	–

Exposure Factor	Value		Units	Source
	CTE	RME		
	area			
ET _f	0	0	unitless	USEPA (2009b), Table 1
ET _{fo}	0.33	0.33	unitless	USEPA (2009b), Table 1 (Assumes 100% of 8 hour work day)
GSF	0.4	0.4	unitless	USEPA (2009b), Table 1
Construction Worker				
EF	250	250	days/year	USEPA (2002a), Exhibit 5-1
CF _{DY}	365	365	days/year	Conversion factor
ED	0.5	1	years	BPJ; USEPA(2002a), Exhibit 5-1
ACF	Specific to risk assessment study area		unitless	--
ET _f	0	0	unitless	USEPA (2009b), Table 1
ET _{fo}	0.33	0.33	unitless	USEPA (2009b), Table 1 (Assumes 100% of 8 hour work day)
GSF	0.4	0.4	unitless	USEPA (2009b), Table 1

APPENDIX D

PARTICULATE MODELING APPROACH

Airborne particulate concentrations will be calculated for dust emissions sources at the Site. There are two primary sources of dust emissions for the Site: wind erosion and soil disturbances associated with construction activities. A tiered modeling approach that progresses from a simplified and upper-bound assessment to a refined and more accurate estimate of potential health risks is proposed for evaluating inhalation exposures related to airborne particulates released from the Site. This appendix provides details on the initial tier proposed in the risk assessment work plan (RAWP). The first tier for estimating airborne particulate concentrations will be based on the simplified site-specific methods provided in the EPA *Supplemental Soil Screening Guidance* (USEPA 2002a). Under this methodology, particulate emission factors (PEFs) are calculated for each receptor exposed to airborne dust generated from Site sources. The PEF value generates an estimate of the ambient air concentration associated with a dust emission mechanism (e.g., wind erosion, excavation, grading). The following discussion provides details of how PEFs will be calculated for the receptors identified in the RAWP. First the equations for calculating emissions for the various fugitive dust sources are presented. Then the PEF equations for each receptor are provided. A complete listing of the PEF equation inputs and values are provided in Table D-1.

DUST EMISSION EQUATIONS

This section provides details on the equations and inputs that will be used to estimate the emissions of fugitive dust sources identified in this RAWP. These dust emission equations were obtained from EPA (2002a) and are consistent with Nevada Division of Environmental Protection (NDEP) guidance (2009f).

Wind Erosion

For the wind erosion component of the fugitive dust emissions, M_{WIND} , the calculation will follow Equation E-20 from EPA (2002a).

$$M_{wind} = 0.036 \times (1 - V) \times \left(\frac{U_m}{U_t} \right)^3 \times F(x) \times A_{surf} \times ED \times 8,760 \text{ hr / yr} \quad \text{Eq. D-1}$$

where,

M_{wind}	=	unit mass emitted from wind erosion (g)
V	=	fraction of vegetative cover (unitless)
U_m	=	mean annual windspeed (m/s)
U_t	=	equivalent threshold value of windspeed at 7 m (m/s)
$F(x)$	=	function dependent on U_m/U_t derived from Cowherd et al. 1985 (as cited in USEPA 2002a); (unitless)
A_{surf}	=	areal extent of site with surface soil contamination (m ²)

ED = exposure duration (years).

The fraction of vegetative cover in the exposure area will be determined for the individual source area being assessed, and described in the risk assessment report. The mean annual wind speed, U_m , is based on data from the Western Regional Climate Center (WRCC; 2010) for Las Vegas, NV. A value of 4.07 m/s will be used for this parameter. For the equivalent threshold windspeed at height, U , the EPA default value of 11.32 m/s will be used (Equation 5-11, USEPA 2002a). The EPA default value will also be used for the related $F(x)$ term (default = 0.194; Equation 5-11, USEPA 2002a). The areal extent for the wind erosion PEF will be based on the size of the source area. The exposure duration (ED) will match the site-specific value assumed for each receptor.

Fugitive dust emissions in the form of wind erosion will be considered at a source area even if construction activities are not planned. If construction activities are included as dust emission sources for a source area, then wind erosion emissions during and post construction will be calculated separately. The dust emission equation shown as Equation D-1 will be used for both calculations, but the ED value will vary accordingly. These separate calculations for wind erosion are needed because some receptors are exposed to these emissions only during or after construction activities. Further details of the ED value in the dust emission calculations for wind erosion are provided in the relevant PEF discussions. All of the remaining dust emission sources to be presented operate only during the period of construction activities.

Vehicle Traffic on Unpaved Roads

For source areas where construction activities are contemplated, dust emissions from vehicle traffic over the unpaved surface soil will be calculated using Equation E-27 from EPA (2002a).

$$M_{road} = 556 \times (W / 3)^{0.4} \times \left[\frac{(365 - p)}{365} \right] \times \sum VKT \quad \text{Eq. D-2}$$

where,

- M_{road} = unit mass emitted from traffic on unpaved roads (g)
- W = mean vehicle weight (tons)
- p = number of days per year with at least 0.01 in. of precipitation (days/year)
- $\sum VKT_R$ = sum of fleet vehicle kilometers traveled over the unpaved road surface during the period of construction activities (km).

$$\sum VKT_R = \frac{N_V \times L_D \times t_w \times 5 \text{ days/week}}{1000 \text{ m/km}} \quad \text{Eq. D-3}$$

where,

- N_V = number of vehicles that cross the road surface daily during construction activities (unitless)
- L_D = length of unpaved road traveled by vehicles per day (m/day)
- t_w = duration of unpaved construction activity in weeks (weeks/year).

The M_{ROAD} equation above assumes the EPA (2002a) default values for road surface silt content (8.5%) and moisture content (0.2%). Values for the other input parameters in the vehicle traffic dust emission equation M_{ROAD} are provided in Table D-1.

Excavation

The fugitive dust released from excavation during construction activities will be calculated using Equation E-21 from EPA (2002a) as follows.

$$M_{excav} = 0.35 \times 0.0016 \times \left(\frac{U_m}{2.2}\right)^{1.3} \times \rho_{soil} \times A_{excav} \times d_{excav} \times N_A \times 10^3 \text{ g/kg} \quad \text{Eq. D-4}$$

where,

- M_{excav} = amount of dust released from excavation during construction (g)
- U_m = mean annual wind speed (m/s)
- M = gravimetric soil moisture content (%)
- ρ_{soil} = wet soil bulk density (Mg/m³)
- A_{excav} = areal extent of the site excavation (m²)
- d_{excav} = average depth of the site excavation (m)
- N_A = number of times the soil is dumped (unitless).

With the exception of the mean annual wind speed, U_m, the input parameters for the excavation equation are site-specific and will be specified for the particular exposure area being evaluated.

Dozing

The fugitive dust equation for dozing during construction activities is based on Equation E-22 of EPA (2002a).

$$M_{doz} = 0.75 \times \left(\frac{0.45 \times s^{1.5}}{M^{1.4}} \right) \times \frac{\sum VKT_{doz}}{S_{doz}} \times 10^3 \text{ g/kg} \quad \text{Eq. D-5}$$

where,

- M_{doz} = unit mass released from dozing operations (g)
- s = soil silt content (%)
- M = gravimetric soil moisture content (%)
- S_{doz} = mean vehicle speed (km/hr)
- $\sum VKT_{doz}$ = sum of kilometers traveled during dozing operations (km).

$$\sum VKT_{doz} = \frac{\left(\frac{A_{gd}^{0.5}}{B_d} \right) \times (A_{gd})^{0.5} \times N_{doz}}{1000 \text{ m/km}} \quad \text{Eq. D-6}$$

where,

- A_{gd} = surface area of grading and dozing operations (m²)
- B_d = width of the blade used for dozing (m)
- N_{doz} = number of times the area is dozed during construction period (unitless).

The values to be used to calculate dust emissions for the dozing operations are provided in Table D-1.

Grading

For grading operations during construction activities the dust emissions will be calculated using Equation E-23 from EPA (2002a).

$$M_{grade} = 0.60 \times 0.0056 \times (S_{grade})^2 \times \sum VKT_{grade} \times 10^3 \text{ g/kg} \quad \text{Eq. D-7}$$

where,

- M_{grade} = the amount of dust released from grading operations during construction activities (g)
- S_{grade} = average speed of grader (km/hr)
- $\sum VKT_{grade}$ = sum of kilometers traveled during grading operations (km).

$$\sum VKT_{grade} = \frac{\left(\frac{A_{gd}^{0.5}}{B_g}\right) \times (A_{gd})^{0.5} \times N_{grade}}{1000 \text{ m/km}} \quad \text{Eq. D-8}$$

where,

- A_{gd} = surface area of the grading and dozing activity (m²)
- B_g = width of the blade used for grading (m)
- N_{grade} = number of times the area is graded during construction period (unitless).

The values to be used to calculate dust emissions for the dozing operations are provided in Table D-1.

Tilling

For tilling operations during construction activities the dust emissions will be calculated using Equation E-24 from EPA (2002a).

$$M_{till} = 1.1 \times s_t^{0.6} \times A_{till} \times 4047 \text{ m}^2 / \text{acre} \times 10^{-4} \text{ ha} / \text{m}^2 \times 10^3 \text{ g} / \text{kg} \times N_A \quad \text{Eq. D-9}$$

where,

- M_{till} = mass of dust released from tilling operations (g)
- s_t = silt content for tilled soil (%)
- A_{till} = areal extent of tilling operations (acres)
- N_A = number of times soil is tilled (unitless).

The values to be used to calculate dust emissions for the tilling operations are provided in Table D-1.

PEF EQUATIONS

PEF equations provide the means for estimating the concentration of dust in ambient air due to dust emissions from the soil. Separate PEF equations are provided for construction workers and onsite workers (i.e., indoor and outdoor). The two major elements of the PEF equation are the dust emission term and a dispersion factor term.

The dispersion factor term, identified as Q/C for all PEF equations presented in this appendix, has the same general form.

$$Q/C = A \times \exp \left[\frac{(\ln A_s - B)^2}{C} \right] \quad \text{Eq. D-10}$$

The dispersion factor expresses the estimated ambient air concentration for the particular dust source being evaluated. Three of the four variables in the dispersion factor equation (i.e., A, B, and C), describe the dilution expected for the source-receptor relationship being modeled; primarily whether the receptor is located on or downwind of the dust emission source. EPA (2002a) has developed recommended values of these three factors for all of the source-receptor relationships discussed in this RAWP. In some cases EPA (2002a) provides climate-specific values for these three factors. The values for Las Vegas, NV, will be used whenever climate-specific values can be selected. The fourth factor, A_s , describes the size of the dust emission source or the exposure area and is therefore related to the site-specific exposure area or decision unit being evaluated. The discussion of PEF equations below will provide details on the dispersion factor equation for each of the receptors considered in this RAWP.

Construction Worker PEF

The PEF for construction workers assumes they are exposed to dust emissions from vehicle traffic and construction activities. These inhalation exposures are assumed to occur only during the period of construction activities. It is assumed that construction workers are located at the center of the dust emission source for the period of exposure. For construction workers NDEP provides the following equation for the PEF based on the combined impact of dust emissions from vehicle traffic over unpaved roads and construction activities (Equation 18; NDEP 2009f).

$$PEF_{CW} = \frac{1}{\left(\frac{1}{PEF_{CWR}} \right) + \left(\frac{1}{PEF_{CWC}} \right)} \quad \text{Eq. D-11}$$

The term PEF_{CWR} represents construction worker exposure to dust from vehicle traffic over unpaved roads during construction and comes from Equation E-18 of EPA (2002a).

$$PEF_{CWR} = \frac{Q}{C_{SR}} \times \frac{1}{F_D} \times \frac{T \times A_R}{M_{ROAD}} \quad \text{Eq. D-12}$$

where,

PEF_{CWR} = subchronic PEF for dust emissions from vehicle traffic over unpaved roads (m^3/kg)

- Q/C_{SR} = inverse of the ratio of the 1-hour geometric mean air concentration to the emission flux along a straight road section bisecting a square site ($g/m^2\text{-s}$ per kg/m^3)
- F_D = dispersion correction factor (unitless)
- T = total time over which construction occurs (s)
- A_R = surface area of the contaminated road segment (m^2)
- M_{ROAD} = dust emission from vehicle traffic on unpaved roads (g), see Equation D-2 of this appendix.

The dispersion factor for this source-receptor combination, from Equation E-19 of EPA (2002a), has the general form shown in Equation D-10 of this appendix. The area term in the equation, A_s , will reflect the size of the source area, in acres. The input values for factors A, B, and C in Equation D-10 are constants that cannot be modified to match the climatic region being investigated.

The dispersion correction factor, F_D , is calculated using Equation E-16 of EPA (2002a) as:

$$F_D = 0.1852 + \frac{5.3537}{t_c} + \frac{-9.6318}{t_c^2} \quad \text{Eq. D-13}$$

where,

- t_c = duration of construction activity (hours).

The A_R term in Equation D-12 defines surface area of the unpaved road over which vehicle traffic occurs. This term is given in Equation E-18 of EPA (2002a) as:

$$A_R = L_R \times W_R \times 0.092903 \text{ m}^2 / \text{ft}^2 \quad \text{Eq. D-14}$$

where,

- A_R = surface area of contaminated unpaved road segment (m^2)
- L_R = length of unpaved road segment (ft) = square root of the source surface area configured as a square
- W_R = width of unpaved road segment (ft).

The PEF for construction workers for dusts generated from construction activities other than vehicle traffic on unpaved roads is given by Equation E-26 of EPA (2002a) as:

$$PEF_{CWC} = \frac{Q}{C_{SC}} \times \frac{1}{F_D} \times \frac{1}{J_{T-CW}} \quad \text{Eq. D-15}$$

where,

- PEF_{CWC} = subchronic PEF for construction activities other than traffic on unpaved roads (m³/kg)
- Q/C_{sc} = inverse of the ratio of the 1-hour geometric mean air concentration and the emission flux at the center of the square emission source (g/m²-s per kg/m³)
- F_D = dispersion correction factor, see Equation D-13 of this appendix
- J_{T-CW} = total time-averaged dust unit emission flux for construction activities other than traffic on unpaved roads (g/m²-s).

The dispersion factor Q/C_{sc} is given by Equation E-15 from EPA (2002a) and follows the general form shown in Equation D-10 of this appendix. The area term in the equation, A_s, will reflect the size of the source area. The input values for factors A, B, and C in Equation D-10 are constants that cannot be modified to match the climatic region being investigated (USEPA 2002a). Construction workers are assumed to be located at the center of the dust emission source throughout the exposure.

The dispersion correction factor, F_D, is the same as described in Equation D-13 of this appendix.

The J_{T-CW} value in Equation D-15, which represents the total time-averaged dust emission flux for the construction worker exposure to dust from construction activities other than vehicle traffic on unpaved roads, is given by Equation E-25 of EPA (2002a).

$$J_{T-CW} = \frac{M_{WIND} + M_{Excav} + M_{doz} + M_{grade} + M_{fill}}{A_{site} \times T} \quad \text{Eq. D-16}$$

where,

- J_{T-CW} = total time-averaged dust unit emission flux for construction activities other than traffic on unpaved roads (g/m²-s)
- A_{site} = areal extent of soil contamination (m²)
- T = total time over which construction occurs (s).

The calculations for the individual construction related dust emission sources (i.e., M_{WIND}, M_{Excav}, M_{doz}, M_{grade}, and M_{fill}) are as described earlier in Dust Emissions Equations section of this appendix.

Onsite Workers

Onsite workers (i.e., outdoor and indoor workers) are not exposed to fugitive dusts from construction activities in the site assessment area. If construction dust emissions are evaluated for an area, the onsite workers are assumed to arrive in the area post construction. Thus, the PEF for the onsite workers is the same whether or not construction activities occur in an exposure area. The following PEF equation for the onsite worker is taken from Equation 24 of NDEP (2009f).

$$PEF_{ow} = \frac{Q}{C_{ow}} \times \frac{3600s/hr}{0.036 \times (1-V) \times \left(\frac{U_m}{U_t}\right)^3 \times F_x} \quad \text{Eq. D-17}$$

where,

- PEF_{ow} = PEF for onsite workers exposed to wind erosion dust emissions in the absence of construction activities (m³/kg)
- Q/C_{ow} = inverse of the ratio of the 1-hour geometric mean air concentration and the emission flux at the center of the square emission source (g/m²-s per kg/m³)
- V = fraction of vegetative cover (unitless)
- U_m = mean annual windspeed (m/s)
- U_t = equivalent threshold value of windspeed at 7 m (m/s)
- F(x) = function dependent on U_m/U_t derived from Cowherd et al. 1985 (as cited in USEPA 2002a); (unitless).

The dispersion factor for the onsite worker, Q/C_{ow}, is given by Equation 25 from NDEP (2009f) and follows the general form shown in Equation D-10 of this appendix. The value of A_s is the site-specific areal extent of the dust emission source. The workers are assumed to be located at the center of the dust emission source. The values of constants A, B, and C for Las Vegas, NV provided by EPA (Exhibit E-3, 2002a) were selected for use in the RAWP.

The fraction of vegetative cover will be set at zero unless construction has occurred in the exposure area, in which case the EPA (2002a) post-construction default value of 0.5 will be assumed. The mean annual wind speed, U_m, is based on observations from the National Weather Service station in Las Vegas, NV. For the equivalent threshold windspeed at height, U_t, the EPA default value of 11.32 m/s will be used (Equation 5-11, USEPA 2002a). The EPA default value will also be used for the related F(x) term (default = 0.194; Equation 5-11, USEPA 2002a).

The PEF equation shown in D-19 above provides an estimate of the ambient air dust concentration. This value is appropriate for use in evaluating outdoor worker exposures

without modification. Indoor workers are assumed to be exposed to dust in indoor air. To be consistent with NDEP approved assumptions for other human health risk assessments in the BMI Complex (BRC et al. 2009), a dilution factor of 0.4 will be used to scale from ambient air dust concentrations to indoor air concentrations. This is the only modification that will be required to use the PEF equation in D-19 in evaluating risks for onsite indoor workers.

Table D-1. Input Parameter Values Used to Develop Particulate Emission Factors.

Parameter	Abbreviation	Units	CTE	RME	Source
Fraction of vegetative cover	V	unitless	--	--	Site-specific
Fraction of vegetative cover post construction	V _{pc}	unitless	--	--	Site-specific
Mean annual wind speed	U _m	m/s	4.07	4.07	WRCC (2010)
Equivalent threshold value of windspeed at 7 m	U _t	m/s	11.32	11.32	Equation 5-11, EPA (2002a)
Function dependent on U _m /U _T derived from Cowherd et al. (1985)	F(X)	unitless	0.194	0.194	Equation 5-11, EPA (2002a)
Areal extent of exposure area or decision unit	A _{surf}	m ²	--	--	Site-specific
Mean vehicle weight	W	tons	8	8	Assumed ^a
Number of days with ≥0.01 in. of precipitation	p	days/year	27	27	WRCC (2010) ^b
Number of vehicles that cross the unpaved road surface daily during construction activities	N _v	unitless	30	30	Assumed ^a
Length of unpaved road traveled by vehicles each day	L _D	m/day	--	--	Site-specific
Total construction time in weeks	t _w	weeks/year	26	52	Assumed
Percent moisture in soil	M	%	--	--	Site-specific
Wet soil bulk density	r _{soil}	mg/m ³	1.74	1.74	(1)
Areal extent of site excavation	A _{excav}	m ²	--	--	Site-specific
Depth of site excavation	d _{excav}	m	--	--	Site-specific
Number of times soil is dumped	N _A	unitless	2	2	EPA (2002a)
Silt content in soil for dozing emissions	s	%	--	--	Site-specific
Mean dozing speed	S _{doz}	km/hr	11.4	11.4	EPA (2002a)
Areal extent of grading and dozing activities	A _{gd}	m ²	--	--	Site-specific
Width of dozer blade	B _d	m	2.44	2.44	EPA (2002a)
Number of times area is dozed	N _{doz}	unitless	--	--	Site-specific
Average grading speed	S _{grade}	km/hr	--	--	Site-specific
Width of grader blade	B _g	m	2.44	2.44	EPA (2002a)
Number of times area is graded	N _{grade}	unitless	--	--	Site-specific
Soil silt content for soil tilled	s _t	%	--	--	Site-specific
Areal extent of site tilling	A _{till}	acres	--	--	Site-specific
Number of times soil is tilled	N _A	unitless	2	2	EPA (2002a)
Dispersion factor constants for construction worker exposure to vehicle emissions over unpaved road surfaces	A	unitless	12.9351	12.9351	Equation E-19, EPA (2002a)
	B	unitless	5.7383	5.7383	Equation E-19, EPA (2002a)
	C	unitless	71.7711	71.7711	Equation E-19, EPA (2002a)
Areal extent of site surface soil contamination	A _S	acres	--	--	Site-specific

Table D-1. Input Parameter Values Used to Develop Particulate Emission Factors.

Parameter	Abbreviation	Units	CTE	RME	Source
Dispersion correction factor ^c	F _D	unitless	0.186	0.186	Equation E-16, EPA (2002a)
Total time over which construction occurs	T	seconds	15,768,000	31,536,000	Assumed
Total hours over which construction occurs	t _c	hr	4,380	8,760	Assumed
Surface area of contaminated unpaved road segment ^d	A _R	m ²	--	--	Site-specific
Width of unpaved road segment	W _R	ft, (m)	20, (6.1)	20 (6.1)	Assumed
Length of unpaved road segment ^e	L _R	ft, (m)	--	--	Site-specific
Dispersion factor constants for construction worker exposure to dust emissions from construction activities	A	unitless	2.4538	2.4538	Equation E-15, EPA (2002a)
	B	unitless	17.5660	17.5660	Equation E-15, EPA (2002a)
	C	unitless	189.0426	189.0426	Equation E-15, EPA (2002a)
Dispersion factor constants for offsite resident exposed to fugitive dust emissions from the modeled dust emission sources	A	unitless	12.1784	12.1784	Exhibit E-5 ^f , EPA (2002a)
	B	unitless	24.5606	24.5606	Exhibit E-5 ^f , EPA (2002a)
	C	unitless	296.4751	296.4751	Exhibit E-5 ^f , EPA (2002a)
Dispersion factor constants for onsite workers exposed to fugitive dust emissions from the modeled dust emission sources	A	unitless	13.3093	13.3093	Exhibit E-3 ^f , EPA (2002a)
	B	unitless	19.8387	19.8387	Exhibit E-3 ^f , EPA (2002a)
	C	unitless	230.1652	230.1652	Exhibit E-3 ^f , EPA (2002a)
Dispersion factor constants for onsite trespassers to fugitive dust emissions from the modeled dust emission sources	A	unitless	13.3093	13.3093	Exhibit E-3 ^f , EPA (2002a)
	B	unitless	19.8387	19.8387	Exhibit E-3 ^f , EPA (2002a)
	C	unitless	230.1652	230.1652	Exhibit E-3 ^f , EPA (2002a)
Exposure Duration	ED	years	Construction Worker = 0.5 Onsite Worker = 7	Construction Worker = 1.0 Onsite Worker = 25	Appendix C, Exhibit C-3

Sources: Cowherd, C.G., G. Muleski, P. Engelhart, and D. Gillette. 1985. Rapid assessment of exposure to particulate emissions from surface contamination sites. EPA/600/8-85/002. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, DC. (not seen, as cited in USEPA 2002a).
 Environ. 2003. Risk assessment for the water reclamation facility expansion site, Henderson, Nevada. Prepared for the City of Henderson, Nevada. October.
 USEPA. 2002a. Supplemental guidance for developing soil screening levels for Superfund sites. OSWER 9355.4-24. U.S. Environmental Protection Agency. December.
 WRCC. 2009. Average wind speeds for Las Vegas. Available at: www.wrcc.dri.edu/htmlfiles/westwind.final.html#NEVADA. Western Regional Climate Center, Desert Research Institute.
 (1) Based on data from vicinity investigations (from data collected in the BMI Common Areas in 2004 and Environ [2003]).

Notes: CTE = central tendency exposure
 RME = reasonable maximum exposure
 -- = site-specific

^a Assumes twenty 2-ton cars and ten 20-ton trucks traverse the Site daily.
^b Based on normal precipitation data provided NDEP for site area.
^c Calculated as $F_D = 0.1852 + (5.3537 / t) + (-9.6318 / t^2)$ where t is the construction time in units of hours.
^d Calculated as $A_R = L_R \times W_R \times 0.092903 \text{ m}^2 / \text{ft}^2$
^e Calculated as the square root of the areal extent of the surface soil contamination configured as a square.
^f Data for Las Vegas, NV used in calculations.

APPENDIX E

PARAMETERS FOR EVALUATING ASBESTOS-INDUCED RISKS

Table E-1. Optimized Dose-Response Coefficients for Pure Fiber Types.

Fiber Type	$K_L \times 100$	$K_M \times 10^8$
Chrysotile	0.6	0.04
Amphibole	3	30

Source: Table 7-18 from USEPA. 2003c. Technical support document for a protocol to assess asbestos-related risk. Final Draft. U.S. Environmental Protection Agency.

Notes: K_L = coefficient for lung cancer
 K_M = coefficient for mesothelioma

Coefficients apply to exposures quantified in terms of concentrations (f/ml) of fibers longer than 10 μm and thinner than 0.4 μm

Table E-2. Estimated Additional Deaths^a from Lung Cancer or Mesothelioma from Constant Lifetime Exposure to 0.0001 TEM f/cm³ Longer than 10 μm and Thinner than 0.04 μm Based on Optimum Risk Coefficients.

	Non-smokers		Smokers	
	Males	Females	Males	Females
Chrysotile				
Lung Cancer	0.185	0.207	1.6	1.5
Mesothelioma	0.0836	0.096	0.0482	0.0702
Combined	0.269	0.303	1.65	1.57
Amphibole				
Lung Cancer	0.2	0.286	2.22	2.47
Mesothelioma	62.7	72.3	36.1	52.7
Combined	62.9	72.5	38.3	55.1

Source: Table 8-2 from USEPA. 2003c. Technical support document for a protocol to assess asbestos-related risk. Final Draft. U.S. Environmental Protection Agency.

Notes: TEM = transmission electron microscopy
^a Estimated additional deaths are per 100,000 persons.

**RISK ASSESSMENT WORK PLAN
FORMER MONTROSE AND STAUFFER FACILITIES
HENDERSON, NEVADA**

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Appendix A. Response to Comments Log

~~Site-Related Chemicals List as Detailed in Hargis + Associates, Inc. (2008)~~

~~Appendix B. Site-Related Chemicals List as Detailed in Hargis + Associates, Inc. (2008)~~

~~Appendix B. Chemicals to be Evaluated for Vapor Intrusion Potential~~

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Table 5-1. Toxic Equivalency Factors and Relative Potency Factors

ACRONYMS AND ABBREVIATIONS

ABSGI	fraction of contaminant absorbed in gastrointestinal tract
ACD	Agricultural Chemical Division
ACF	area correction factor
ADD	average daily dose
ALM	adult lead methodology
ARR	asbestos-related risk
AT	averaging time
ATSDR	Agency for Toxic Substances and Disease Registry
BaP	benzo(a)pyrene
BCL	basic comparison level
bgs	below ground surface
BHC	benzene hexachloride
BMI	Black Mountain Industrial
BRC	Basic Remediation Company
BSA	benzenesulfonic acid
BW	body weight
Cal/EPA	California Environmental Protection Agency
cm	<u>centimeter</u>
COPC	constituent of potential concern
CSF	cancer slope factor
CSM	conceptual site model
CTE	central tendency exposure
DAD	<u>daily average dose</u>
DAD	<u>dermally absorbed dose</u>
DCBP	dichlorobenzophenone
DEPT	diethyl phosphorodithioic acid
DMPT	dimethyl phosphorodithioic acid
DQA	<u>data quality assessment</u>

EC	exposure concentration
ED	exposure duration
EF	exposure frequency
EPA	U.S. Environmental Protection Agency
EPC	exposure point concentration
ET_{β}	fraction of time spent indoors
ET_{α}	fraction of time spent outdoors
f/cm^3	fiber per cubic centimeter
ft	foot
GI	gastrointestinal tract
GSF	gamma shielding factor
HCL	hydrochloric acid
HEAST	Health Effects Assessment Summary Tables
HHRA	human health risk assessment
HI	hazard index
HQ	hazard quotient
in.	inch
Integral	Integral Consulting Inc.
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
I & E	Johnson & Ettinger
kg	kilogram
LADD	lifetime average daily dose
lb	pound
LOAEL	lowest-observed-adverse-effects level
m^2	square meter
MDA	minimum detectable activity
MDL	method detection limit
mg/kg	milligram per kilogram
mg/kg-day	milligram per kilogram per day

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<u>mg/m³</u>	<u>milligram per cubic meter</u>
Montrose	Montrose Chemical Company of California
MRL	minimal risk level
NDEP	Nevada Division of Environmental Protection
NHANES	National Health and Nutrition Examination Survey
NOAEL	no-observed-adverse-effects level
Olin	Olin Corporation
ORNL	Oak Ridge National Laboratory
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PbB ₀	baseline blood lead level
PCB	polychlorinated biphenyl
pCBSA	4-chlorobenzene sulfonic acid
PCDD	polychlorinated dibenzo- <i>p</i> -dioxin
PCDF	polychlorinated dibenzofuran
pCi	picocurie
PEF	particulate emission factor
<u>PEF</u>	<u>particulate emission factor</u>
POD	point of departure
<u>POD</u>	<u>point of departure</u>
Pioneer	Pioneer Americas, LLC
POD	point of departure
<u>ppb</u>	<u>part per billion</u>
PPRTV	provisional peer reviewed toxicity values
PRG	preliminary remediation goal
QC	quality control
RAGS	Risk Assessment Guidance for Superfund
RAIS	Risk Assessment Information System

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RAS	remedial alternatives study
RAWP	risk assessment work plan
RCRA	Resource Conservation and Recovery Act
RfC	reference concentration
RfD	reference dose
RME	reasonable maximum exposure
RPF	relative potency factor
SE	secular equilibrium
SMC	Stauffer Management Company LLC
<u>SOP</u>	<u>standard operating procedure</u>
SQL	sample quantitation limit
SRC	site-related chemical
Stauffer	Stauffer Chemical Company
SVOC	semivolatile organic compound
Syngenta	Syngenta Crop Protection, Inc.
TCDD	tetrachlorodibenzo- <i>p</i> -dioxin
TEF	toxic equivalency factor
TEQ	toxic equivalent
TEM	transmission electron microscopy
TIC	tentatively identified compound
Timet	Titanium Metals Corporation
TPH	total petroleum hydrocarbon
Tronox	Tronox, Inc.
<u>UCL</u>	<u>upper confidence limit</u>
<u>UF</u>	<u>uncertainty factor</u>
<u>µg/dL</u>	<u>microgram per deciliter</u>
<u>µg/m³</u>	<u>microgram per cubic meters</u>
<u>µm</u>	<u>micrometer</u>
<u>VF</u>	<u>volatilization factor</u>
VOC	volatile organic compound

WHO World Health Organization
WOE weight-of-evidence

1 INTRODUCTION

This risk assessment work plan (RAWP) outlines the proposed approach for assessing potential human health risks at the former Montrose and Stauffer facilities in Henderson, Nevada (the Site). This RAWP has been prepared on behalf of Montrose Chemical Company of California (Montrose), Stauffer Management Company LLC/Syngenta Crop Protection, Inc. (SMC/Syngenta) and Olin Corporation (Olin) (the Companies) as part of the overall effort to characterize the nature and extent of contamination at this former facility and determine the need for, and effectiveness of, remedial actions to address overall risks.

1.1 SITE DESCRIPTION¹

The former Montrose and Stauffer facilities are located in the southwest portion of a heavily industrialized area currently referred to as the Black Mountain Industrial (BMI) Complex. The BMI Complex is located within an unincorporated portion of Clark County surrounded by the City of Henderson, NV. Under current operations, the BMI Complex includes property owned, leased, or administered by Olin (and formerly Pioneer Americas LLC [Pioneer]), Tronox, Inc. (Tronox), Titanium Metals Corporation (Timet), Chemstar Lime Company, and Basic Remediation Company (BRC) and its affiliates (Figure 1-1). The Site, as referred to in this work plan, comprises the portion of the BMI complex previously utilized by Montrose and Stauffer Chemical Company (Stauffer) and currently owned and operated by Olin for the production of liquid chlorine, caustic soda, hydrochloric acid (HCL), and bleach (Figure 1-2). The total acreage of the Site is approximately 315 acres.

1.1.1 Site Setting

The Site is located within the Las Vegas Valley and the southwestern part of the Basin and Range physiographic province. The climate is arid with precipitation averaging slightly less than 4.5 in. per year (NOAA 2009). Winters are mild and summers are hot with temperatures often above 100 degrees Fahrenheit (°F). The average annual daily temperatures range from a low of approximately 56°F to a high of approximately 80°F (NOAA 2009).

Land surface at the Site is a mixture of natural and non-native materials. Some portions of the Site are paved. Outside of these areas, most of the land surface is bare soil or sparsely vegetated. Surface and near-surface soils at the Site are generally coarse-grained, comprised of quaternary alluvium deposits consisting of sands and gravels, with varying amounts of silts and occasional cobbles (Hargis 2008). In some areas, caliche is present on the surface (PES 2006, 2007).

¹ The information summarized in this section is largely excerpted from previous reports prepared by PES (2006, 2007) and Hargis (2008).

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Natural site drainage is to the north, but no perennially wet drainages or other natural water bodies exist on the Site. Wind direction is variable, but predominately from the northwest, south, southwest, and southeast (Figure 1-3).

The Site is currently used exclusively for industrial processes. Site access to Olin's current operating facility is controlled by gates and a guard house. A 6-ft high chain-linked razor wire-topped perimeter fence exits around portions of the Site.

Areas immediately adjacent to the Site are undeveloped or industrial/commercial. The nearest residences occur to the west-northwest, south, and southeast and are located more than ~~one-half~~ 1/2 mile from the Site.

1.1.2 Site History

The Site was first developed as part of the original BMI Complex, which was constructed under a contract with the U.S. Defense Plant Corporation and operated by BMI to produce magnesium for the World War II effort from 1942 through 1944. Chlorine was essential to magnesium production and a chlorine and caustic soda plant was constructed at the Site (PES 2006).

From 1945 through 1984, the Site was operated by Stauffer for production of chlorine, sodium hydroxide, HCL, and agricultural chemical products (PES 2006). The most extensive operations included the manufacture of chlorine and caustic soda from 1945 through 1984, and the production of HCL from 1954 to 1984. Stauffer also manufactured the pesticides trithion® (carbophenothion) (1958 through 1984), imidan® (phosmet) (1964 through 1982), parachlorothiophenol (1960 through 1984), and thiophenol (1967 through 1982) at its Agricultural Chemical Division (ACD) Plant. Lindane (gamma-benzene hexachloride [BHC]) was produced at the former Lindane Plant from 1946 through 1958. The Stauffer manufacturing facilities were largely demolished in 1984.

Montrose constructed and operated a manufacturing plant to produce a variety of organic chemicals from 1947 through 1983 (Hargis 2008). Organic chemical products included chlorobenzene, polychlorinated benzenes (PCBs), chloral, and 4,4'-dichlorobenzil. Montrose ceased operations at the organic chemical plant in 1983 and demolished the plant in 1984. Montrose also constructed a manufacturing plant for the production of synthetic HCL in 1954 and at an expanded facility constructed in 1977 (Hargis 2008). Montrose produced HCL at these production facilities until 1985.

Olin currently operates chlor alkali production facilities at the Site and manufactures liquid chlorine, caustic soda, HCL, and bleach. Olin began operation in 2007 when they acquired Pioneer.

1.2 SCOPE AND FOCUS OF THE HUMAN HEALTH RISK ASSESSMENT

This document has been prepared to satisfy the Nevada Division of Environmental Protection (NDEP) requirements to provide a RAWP detailing the human health risk assessment (HHRA) methodology as part of the overall remedial alternatives studies (RAS) to be conducted at the Site (NDEP 2008a). As outlined by NDEP, RAS will be conducted at various source areas and potential source areas ~~site assessment and/or waste management areas~~ at the Site. This RAWP has been developed to detail the procedures to be used to evaluate human health risks at the areas where risk-based closure may be sought by the Companies, and/or where the evaluation of human health risks is appropriate to support the evaluation of remedial action alternatives.

The HHRA approach outlined here is consistent with basic procedures recommended by the U.S. Environmental Protection Agency (EPA) for conducting risk assessments at waste sites. Documents that will guide the risk assessment include, but are not limited to, the following:

- *Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual, Part A* (USEPA 1989)
- *Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual, Part E* (USEPA 2004a)
- *Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual, Part F* (USEPA 2009a)
- *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites* (USEPA 2002a).

In addition, the risk assessment will follow guidance developed by NDEP applicable to risk assessment, including data evaluation to support risk assessment, provided at the following website: <http://ndep.nv.gov/bmi/technical.htm>.

The focus of the HHRA will be to evaluate risks associated with conditions that exist, or are anticipated to exist, at the various source areas and potential source ~~site assessment and/or waste management areas~~ following implementation of the remedial decision (i.e., conditions at "closure"). Remedial decisions may include an active remedy and/or no further action. For purposes of the NDEP RAS process, these "post-remedy" or "closure" conditions constitute the baseline condition for each ~~site assessment area~~ that will be evaluated in the risk assessment.

The various source areas and potential source ~~site assessment areas at the Site that may be evaluated via a HHRA are identified in the RAS document. The RAS document also identifies site assessment areas that were developed by grouping various source areas defined in order to simplify and organize future investigation and RAS activities. The HHRA will focus on potential exposures within ½-acre areas across the source/site assessment area. If sampling data for multiple ½-acre exposure areas exhibit similar concentration distributions they may be combined for evaluation in the HHRA. Sampling data may not be available within each of the~~

1/2-acre exposure areas of a source/site assessment area; however, assumptions of similar concentration distributions across areas larger than 1/2-acre may allow the risk assessment to be applied to combined exposure areas. Such aggregation of 1/2-acre exposure areas, as supported by the data, would become decision units for the risk assessment. Use of the decision units would allow for risk management decisions to be made simultaneously for many 1/2-acre exposure areas within a source/site assessment area based on a similarity in the contaminant concentration distribution that allows for aggregation of individual exposure areas. Details on the manner in which data will be treated and risk will be characterized for source/site assessment areas is described in more detail in the remainder of this RAWP.

The HHRA will address potential exposures and risks assuming that the overall site will remain an industrial property after closure. As such, the assessment assumes that deed restrictions and institutional controls that limit the use of the site to industrial activity will be put in place as part of remedial actions. If such restrictions and controls are not implemented the conclusions of the risk assessment cannot be used to predict risks to receptors under alternate future use scenarios.

The HHRA will address potential exposures to onsite industrial/commercial workers, construction workers, and maintenance workers quantitatively. A qualitative analysis assessment will be conducted to evaluate potential exposures to trespassers, and offsite residents. Potential exposures to constituents of potential concern (COPCs) detected in surface (i.e., 0-6 in. below ground surface [bgs]) and shallow soils (i.e., 0-10 ft bgs) will be evaluated for the direct contact pathways, as well as inhalation of vapor-phase and particulate-sorbed contaminants in indoor and outdoor air. For deeper vadose zone soils (i.e., > 10 ft bgs) and groundwater, the potential for vapors to migrate from the subsurface to indoor and outdoor air also will be evaluated.

Groundwater is being addressed from a non-degradation standpoint and only factors into the HHRA via indirect exposures related to inhalation of volatiles released from groundwater beneath the Site. Direct exposures to groundwater via consumptive use will not be subject to a formal risk assessment. Instead, to support management decisions regarding remedial actions, groundwater quality data will be compared with chemical- and radionuclide-specific standards that define acceptable risk levels for consumptive use. Additionally, in order to characterize potential impacts of soils on groundwater quality, soil data (0-10 ft bgs) will be compared to NDEP basic comparison levels (BCLs) for leaching. Tables presenting the comparisons to leaching BCLs will be included in the risk assessment. However, evaluations related to protection of the overall quality of groundwater as a resource will be evaluated separately as part of the remedial alternatives assessment, but not in the risk assessment.

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1.3 RISK AND CHEMICAL-SPECIFIC GOALS

Remediation goals for a source/site assessment area will be developed on a case-by-case basis as part of the overall RAS process. The following conditions will be applied in development of the remediation goals.

1. Post-remediation chemical concentrations and radionuclide activities in Site soils will have a cumulative theoretical upper-bound incremental carcinogenic risk level point of departure (POD) of 10^{-6} . For cases where NDEP concurs that this goal is unfeasible, the goal may be re-evaluated in accordance with USEPA guidance (USEPA 1991, 1995). The POD risk goal will be evaluated separately for chemicals, asbestos, and radionuclides.
2. Post-remediation chemical concentrations in Site soils are targeted to have an associated cumulative, noncancer hazard index (HI) of 1.0 or less. If the HI is determined to be greater than 1.0, target organ-specific HIs may be calculated for primary and secondary organs. The final risk goal will be to achieve target organ-specific noncancer HIs of 1.0 or less.
3. The risk-based target goal for lead in soil is 800 mg/kg for industrial/commercial land use. This is based on the USEPA's Adult Lead Methodology (ALM) using default input factors for an industrial/commercial worker (USEPA 1996a, NDEP 2009a).
4. Where background levels exceed risk-based levels, Site soils are targeted to have risks no greater than those associated with background conditions.
5. ~~Asbestos cancer risks~~Cancer risks from asbestos are based on the estimated additional deaths from lung cancer or mesothelioma due to constant lifetime exposure. The risk-based POD for asbestos is 10^{-6} . As mentioned above, risk from asbestos is evaluated separately from other chemicals and radionuclides. For cases where NDEP concurs that this goal is unfeasible, the goal may be re-evaluated in accordance with USEPA guidance (USEPA 1991, 1995).
6. The target goal for dioxin/furan toxic equivalents (TEQ) for commercial and industrial land use is 1 ppb. This value is based on the 1998 USEPA OSWER Directive with a modification to address identified uncertainties (10-fold uncertainty factor) regarding cancer potency in humans that results in a screening range of 0.5-2 ppb. A single value of 1 ppb was selected (NDEP 2009a). Risks related to TEQs will only be quantified and presented if residual

concentrations exceed the target goal. If risks are quantified the uncertainty analysis will explain (at a minimum) the portion of the risks that are related to non-detected congeners as well as the risks associated with the NDEP 1 ppb TEQ target goal.

~~6. The target goal for dioxin/furan toxic equivalents (TEQ) for commercial and industrial land use is 1 part per billion (ppb). This value is based on the 1998 USEPA OSWER Directive with a modification to address identified uncertainties (10 fold uncertainty factor) regarding cancer potency in humans that results in a screening range of 0.5 to 2 ppb. A single value of 1 ppb was selected (NDEP 2009). Risks related to TEQs will only be quantified and presented if residual concentrations exceed the target goal. If risks are quantified the uncertainty analysis will explain (at a minimum) the portion of the risks that are related to non-detected congeners as well as the risks associated with the NDEP 1 ppb TEQ target goal.~~

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4:31.4 ORGANIZATION OF THIS REPORT

The remainder of this document provides a detailed overview of the approaches that will be used to address potential human health risks associated with constituents that are present in soils or groundwater at the Site. It is organized into the following sections:

- Section 2 – Exposure Scenarios for the Site
- Section 3 – Data Evaluation
- Section 4 – Exposure Assessment
- Section 5 – Toxicity Assessment
- Section 6 – Risk Characterization
- Section 7 – References.

2 EXPOSURE SCENARIOS FOR THE SITE

The exposure scenarios to be considered in the HHRA are dependent upon the exposure pathways relevant to the Site and receptor populations that use the Site. As discussed previously, the exposure scenarios to be evaluated assume that the Site will remain as an industrial facility at closure.

Figure 2-1 summarizes the exposure pathways and receptor populations that will be considered in any HHRA to be conducted at the Site. Importantly, this summary figure is meant to provide a comprehensive listing of the suite of potential exposure pathways and receptors at the Site as a whole. Not all exposure pathways and receptor groups will necessarily be applicable for every site assessment area (i.e., exposure area). The HHRA conducted for each exposure site assessment area or decision unit will discuss the selection of exposure pathways and receptor groups evaluated and provide the rationale for exclusion of any exposure pathways and receptor groups.

2.1 EXPOSURE PATHWAYS

EPA (1989) has developed the concept of an exposure pathway to define the ways in which receptors might be exposed to constituents. Exposure pathways combine information on the source and transport of a constituent to a point of contact with a receptor and the exposure routes at that point. To be considered complete, an exposure pathway must contain the following elements (USEPA 1989):

1. A source and mechanism of release
2. A retention or transport medium
3. A point of potential contact with the affected medium
4. An exposure route at the contact point.

If any of these elements is missing, exposure will not occur, and the exposure pathway is not complete. Only complete exposure pathways are selected for evaluation in risk assessments.

2.1.1 Sources, Transport, and Contaminated Media

A conceptual site model (CSM) is a tool used to describe the source, release, distribution, and transport of chemical constituents to potential receptor populations. As such, a CSM provides detail related to development of exposure pathways for the Site. A draft site-wide CSM has been developed to address contamination associated with the Site. As part of the overall RAS process (NDEP 2008a), this site-wide CSM is being supplemented by the development of area-specific CSMs. These focused CSMs are being used to guide data collection and remedy design

at the various ~~source and/or site assessment and/or waste management~~ areas, and also will be useful for determining the potentially complete exposure pathways that are relevant at such areas.

The area-specific CSMs will be updated as additional information is collected during site investigation and the evaluation of remedial actions. The draft site-wide CSM (Hargis 2008), however, provides sufficient background information to support a conceptualization of the range of sources, release, fate and transport mechanisms, chemicals, and contaminated media that could be considered in the subject risk assessments.

Briefly, past manufacturing and waste management activities resulted in the release of chemicals to soil and/or groundwater at the former Montrose and Stauffer facilities. These chemicals can be transported in the environment by a variety of mechanisms and reach potential human receptors who contact contaminated media.

Montrose manufactured organic chemicals including chlorobenzene, polychlorinated benzenes, PCBs, choral and 4,4'-dichlorobenzil and HCL at the Site from 1947 to 1983. Stauffer manufactured chlorine, sodium hydroxide, hydrochloric acid, and agricultural chemical products including pesticides and herbicides at the Site from 1954 to 1984. Olin currently operates chlor alkali production facilities at the Site and manufactures liquid chlorine, caustic soda, HCL, and bleach. Historical perspective regarding the impact of these operations on environmental contamination will be provided within the area-specific CSMs.

In cooperation with NDEP a list of site-related chemicals (SRCs) was agreed upon based on a review of historic Site operations, practices, and analytical data (NDEP 2006a,b). SRCs have been detected in surface soils, subsurface soils, and groundwater at the Site. SRCs for the Site include:

- Volatile organic chemicals (VOCs)
- Semivolatile organic chemicals (SVOCs)
- Pesticides and related by-products
- Polychlorinated biphenyls (PCBs)
- Dioxins/furans
- Organic acids
- Metals; and
-

- Asbestos.

In addition to this list, a number of tentatively identified compounds (TICs) have been selected as SRCs for the Site. Hargis (2008) provided a list of SRCs that have been identified for the former Montrose and Stauffer operations. This list is included as Appendix A-B of this RAWP.

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NDEP additionally has requested that radionuclides be addressed in the risk assessment (NDEP 2008b,c), and this RAWP therefore also includes procedures for evaluating radionuclide exposures and associated risks.

For ease of discussion, the previously identified SRCs along with radionuclides are collectively referred to as SRCs in this RAWP. The RAWP provides details on how all SRCs would be evaluated in an HHRA; however, it may be the case that only a subset of this full suite of SRCs will be addressed for a particular ~~exposure site assessment or waste management area~~. Site-specific conditions that warrant deviation from the list of SRCs presented in this RAWP will be discussed with NDEP prior to generating the HHRA.

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SRCs in soil can be directly contacted by persons using the Site. In addition, constituents that are sorbed to soils can be transported to air via wind erosion or due to other physical disturbances of the soil (e.g., vehicle traffic, excavation). Once in air, the soil-sorbed SRCs (i.e., particulates) can be transported to potential receptors both on and off the Site. In addition, vapors that are present in subsurface soils can be transported to the surface and subsequently be dispersed and reach receptors either on- or offsite. Volatile constituents in groundwater also can reach potential receptors as the result of vapor transport through vadose zone soils to surface environments. (As discussed earlier, direct consumptive uses of groundwater will not be evaluated in the risk assessment.) Radioactive elements in soil can additionally release gamma, beta, and alpha radiation to which receptors can be externally exposed.

2.1.2 Exposure Routes

Human receptors can be exposed to SRCs in contaminated media by the following exposure routes:

- Ingestion of contaminated media (e.g., soils)
- Dermal contact (e.g., with soils)
- Inhalation (i.e., vapor or particulate phase constituents).

In addition, human receptors can be exposed externally to certain radionuclides without direct contact or inhalation. These exposures are termed "external exposures".

2.2 POTENTIAL RECEPTOR POPULATIONS

As discussed earlier, the HHRA will address potential exposures and risks assuming that the overall site will remain an industrial property after remedial actions have been implemented (i.e., at closure). As such, the primary receptor populations that could be exposed to SRCs at the Site are site workers. Other potential onsite receptors, such as trespassers, will not be evaluated quantitatively. As stated by USEPA (2002a) evaluation of exposures to members of the public under a non-residential land use scenario is not warranted for two reasons; first, public access is generally restricted at industrial sites, and second, while the public may have access to commercial sites, onsite workers have a much higher exposure potential because they spend substantially more time at the site. Additional onsite exposures could occur for trespassers that illegally enter the Site. Onsite workers and trespassers will be considered in the HHRA. No other onsite receptors will exist.

Some offsite receptors may exist under certain conditions. For example, SRCs that are transported from the Site in air (either as particulate or vapors) also could reach offsite receptors. The principal offsite receptors are nearby workers and residents. Exposures to offsite workers will be lower than those to onsite workers (due to fewer exposure routes and lower exposure levels); Based on a comparison of key exposure factors for the onsite and offsite receptors, exposure to offsite residents is additionally anticipated to be lower than for workers onsite. The conclusion is exemplified by the 100-fold difference in the default particulate emission factor (PEF) from construction for onsite receptors versus offsite receptors as recommended by USEPA (2002a). Potential exposures to onsite workers will be higher because this parameter has a much larger influence on the inhalation pathway evaluation compared to other exposure factors that may be higher for the offsite resident. Therefore offsite receptors will not be evaluated quantitatively; a discussion of the rationale for the decision will be included in the risk assessments. therefore, risks to offsite workers will not be evaluated in a quantitative manner in the risk assessments. Offsite residents, however, constitute a unique receptor population different from onsite workers and so will also be considered for evaluation in the risk assessments. In addition, a qualitative evaluation of the offsite worker for the construction scenario will be completed; the risk results for onsite workers will provide the foundation for this evaluation.

The principal receptor populations that will be quantitatively evaluated in the HHRA and the routes by which they might be exposed are discussed below. The particular receptors and exposure pathways to be evaluated for any exposure individual site assessment area within the Site will be discussed in the HHRA conducted for the relevant source/site assessment area.

2.2.1 Indoor Worker

The indoor worker is defined as a long-term, full-time employee who spends most of the day working indoors. Workers may be exposed to outdoor dusts that have infiltrated the building,

outdoor soils that have been tracked in, and to contaminants present in indoor air as the result of vapor intrusion.

Potentially complete exposure pathways for the indoor worker are:

- Inhalation of indoor dust
- Inhalation of vapors and radon² in indoor air released from soil and groundwater
- Incidental ingestion of surface soil that has been tracked indoors
- External radiation exposure from surface soil that is outdoors, and surface soil that has been tracked indoors (radionuclides only).

Surface soils defined as the top 6 in. of the soil column, are used to define the potential concentrations of SRCs in dust/soils that reach indoors. The vapor inhalation pathway is based on volatile concentrations in the full soil column (i.e., from surface down to groundwater) and in the groundwater. External radiation exposure to radionuclides that are present in outdoor soil is limited to materials within the top 6 in. of soil; radionuclides found below this level are shielded by the top layer of soil and do not contribute to external radiation exposure (USEPA 2000).

Workers can additionally be exposed to radiation via physical immersion in airborne particulates containing radionuclides. This is a complete exposure pathway (as noted in Figure 2-1) but consistent with EPA guidance for developing preliminary remediation goals (PRGs) for radionuclides (USEPA 2009b), contributes negligibly to overall exposures and will not be evaluated in the risk assessments conducted for the Site.

2.2.2 Outdoor Worker

The outdoor worker is defined as a long-term, full-time employee who spends most of the day working outdoors. This receptor is assumed to participate in relatively low-intensity activities such as building maintenance, unloading and loading materials and supplies, or occasional digging. Soil exposure for this receptor group is limited to surface soils. Inhalation of vapors as well as dust generated by wind erosion and construction activities also may occur.

Potentially complete exposure pathways for the outdoor worker are:

- Inhalation of outdoor dust
- Inhalation of vapors and radon in ambient air released from soil and groundwater³

²An NDEP approved risk assessment methodology for radon is currently not available; risks that may occur via exposure to radon will be addressed in a future guidance document from NDEP, and will not be quantified in the risk assessments for the Sites completed prior to that guidance.

³Pathway will be evaluated quantitatively only if needed based on results of indoor air evaluation.

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- Incidental ingestion of surface soil
- External radiation exposure from surface soil (radionuclides only)
- Dermal contact with soil.

Again, external radiation exposure via immersion is also a complete pathway, but contributes negligibly to exposure, and will not be evaluated.

2.2.3 Construction Worker

Construction workers are expected to participate in shorter term, intermittent work at the Site. Work completed by this group might include demolition or construction activities completed as part of developing infrastructure for future onsite activities. The activities for this receptor may involve substantial onsite exposures to surface and subsurface soils. Workers are assumed to have potential for direct contact with soil from 0 to 10 ft bgs. Inhalation of dust and vapors also may occur.

The construction workers may contact exposure media via the following exposure pathways:

- Inhalation of outdoor dust
- Inhalation of ~~vapors~~ vapors and radon in ambient air released from soil and groundwater
- Incidental ingestion of surface and subsurface soil
- External radiation exposure from surface and subsurface soil (radionuclides only)
- Dermal contact with surface and subsurface soil.

Given that subsurface soils are hypothesized to be exposed during construction activities, radionuclides in subsurface soil could be a source for external radiation exposures for this receptor group. External radiation exposure via immersion is also a complete, but negligible, exposure pathway for this receptor group.

2.2.4 Trespasser

~~The trespasser is assumed to have an exposure of intermediate length. For the purposes of this assessment, a trespasser has been assumed to be an older child (7-12 years old) that illegally enters the Site.~~

~~Soil exposure for this receptor group is limited to surface soils. Inhalation of vapors as well as dust generated by wind erosion and construction activities also may occur.~~

¹ Pathway will be evaluated based on soil gas data and supplemented by perimeter air monitoring if appropriate.

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Potentially complete exposure pathways for the trespasser are:

- Inhalation of outdoor dust
- Inhalation of vapors in ambient air released from soil and groundwater
- Incidental ingestion of surface soil
- External radiation exposure from surface soil (radionuclides only)
- Dermal contact with soil.

External radiation exposure via immersion is an additional complete, but negligible, exposure pathway.

2.2.5 Offsite Resident

Offsite residents could be exposed to SRCs present as vapors or to wind-blown dust released from the Site and dispersed to residential areas. For this scenario, the resident is expected to spend a portion of their time outdoors, and a portion of their time inside the residence.

Offsite residents may contact exposure media via the following exposure pathways:

- Inhalation of dust transported from the Site
- Inhalation of vapors in air released from soil and groundwater at the Site.

Offsite residents also can be exposed to chemicals in outdoor surface soil onto which dust from the Site has been transported, or to indoor dust that has been tracked inside from outside soils. The risk assessments will include a qualitative discussion of residential risks from exposures to site dust transported offsite to residential surface soils via this exposure pathway. The discussion will be based on risk results for onsite receptors exposed to surface soils, together with information regarding relative soil concentrations (on- and offsite) and exposure characteristics (e.g., duration, frequency). In addition offsite residents may be exposed to volatile chemicals released from offsite groundwater via inhalation from a vapor intrusion pathway. The offsite resident vapor intrusion pathway is being addressed as part of an all (BMI Complex and Common Areas) Companies investigation.

No radiation exposures to offsite residents will be evaluated in the risk assessments.

3 DATA EVALUATION

Analytical data collected as part of past and future site investigations will be the source of the SRC data evaluated in the risk assessments. This section describes the types of data that may be used for the risk assessments as well as the proposed procedures to 1) evaluate and select data for use in each risk assessment, 2) process analytical sample results to support use in each risk assessment, and 3) select specific SRCs for quantitative evaluation in each risk assessment.

3.1 DATA TYPES

The following types of data ~~will~~ may be evaluated in the risk assessments, as relevant and available:

- Soil data – all SRCs
- Groundwater data – volatile SRCs
- Soil vapor data - volatile SRCs.
- ~~Flux chamber data – volatile SRCs.~~

Soil data would be used to evaluate direct contact exposures (i.e., ingestion, dermal contact, and inhalation of dust) and potential impacts to groundwater from leaching. Soil vapor data would be used to evaluate inhalation exposures to volatile chemicals that could migrate into indoor or ambient air. Groundwater and soil data could be used as a secondary line of evidence in support of the evaluation of the soil vapor data. (e.g., will be used to define source characteristics in the case that vapor transport is modeled. The groundwater data that would ~~ill~~ be used in such an application modeling exercise would ~~ill~~ be that collected from the alluvial aquifer (i.e. Shallow Zone) and fine-grained Upper Muddy Creek Formation. This groundwater is closest to the surface and therefore best represents the potential source of groundwater chemicals available for vapor transport to the surface, which is the only groundwater-related pathway with a potentially completed exposure pathway. As mentioned earlier, direct consumptive use of groundwater will not be evaluated in the risk assessment. The quality of all groundwater (shallow and deep) will, however, be evaluated separately as part of the remedial alternatives assessment.

~~For modeling purposes, volatile SRCs will be defined as those categorized as such by EPA's Draft Guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway From Groundwater and Soils (Subsurface Vapor Intrusion Guidance) (2002b). SRCs identified as volatile by EPA are listed in Appendix B. If EPA (2002b) has not reviewed a particular SRC for volatility (2002b), NDEP's determination of chemical volatility that is presented in its table of basic comparison levels (BCLs) (NDEP 2009a) will be used. If measured soil vapor concentrations (e.g., from soil vapor~~

~~probes or soil vapor flux chamber sampling) are used to assess vapor exposures, all detected chemicals will be evaluated.~~

3.2 DATA REVIEW AND SELECTION

Available analytical data will be reviewed to determine its suitability for use in each risk assessment. EPA guidance for data usability in risk assessment (USEPA 1992a,b) and NDEP procedures outlined in guidance issued for assessing data usability for environmental investigations at the BMI Complex and Common Areas (NDEP 2008d) will guide the data assessments. Data usability evaluations will be completed prior to the risk assessments, and will be documented in reports following specifications outlined by NDEP (2008d). The risk assessments will include a summary of the findings of the data usability evaluation. The implications of issues raised in the usability evaluation will be discussed in the uncertainty section of the risk assessment.

3.3 DATA PROCESSING

Following the data usability evaluation, data deemed of sufficient quality to support the risk assessment will be compiled in a database to support the exposure and risk calculations. Relevant sampling data for the Site may include detected and non-detected values, duplicate samples, and split samples. The treatment of these different data types will follow EPA (1989, 1992a,b) and NDEP guidance (2008d,e,f) and is discussed below.

3.3.1 Detected Analytes

Laboratory results can be broadly classified as detects or non-detects. Detected results reflect cases in which a measurable quantity of a constituent was determined and reported by the laboratory. Detected results may have a qualifier assigned by the laboratory, or during the data validation process. As part of the data usability evaluation, all qualifiers assigned to detected data will be reviewed and treated in accordance with EPA guidance (USEPA 1989, 1992a,b). Detected data that are deemed appropriate for use in the risk assessment by the data usability evaluation will be used at the full reported value.

3.3.2 Non-Detects

Cases where analytical parameters are not detected above some measurement threshold are defined as non-detections. Non-detected results are qualified as such by the laboratory and an associated quantitation limit is provided. Non-detected values can also carry other qualifiers assigned during the analysis or validation process. As part of the data usability evaluation, the qualifiers assigned to all non-detected values will be reviewed using EPA guidance (USEPA

1989, 1992a,b). All non-detected results that are considered appropriate for use in the risk assessment will be included in the database.

For non-detected results the sample quantitation limit (SQL)³ will be reported for all analytes with the exceptions of radionuclides and asbestos. For radionuclides the value reported by the laboratory, which may be less than the minimum detectable activity (MDA)⁴, will be reported. For asbestos, the reported analytical sensitivity for the non-detected sample will be presented.

Summary statistics characterizing both detected concentrations, and the quantitation limits specified above for non-detected results, will be provided in a form consistent with NDEP guidance (NDEP 2008e).

3.3.3 Duplicate and Split Samples

Duplicate samples and split samples are commonly included as part of data collection efforts for assessing environmental contamination. A field duplicate is a distinct sample collected from the same point in time and space as the first sample, or as near to the same time and place as possible. A field split sample is derived from a sample homogenized in the field; the homogenized sample is split into two samples, each of which is analyzed separately. The second sample is assigned the label of field split and is considered the quality control (QC) sample (NDEP 2008f).

Following NDEP recommendations (NDEP 2008f) the treatment of duplicate samples will depend on the variance of the QC sample and the site sample results. Variance is not a factor for consideration in the treatment of split samples. For the treatment of duplicates, sample results will be summarized to determine whether the variance between duplicate samples and site samples is similar. If appropriate to the data (e.g., sufficient sample size), statistical tests will be used to evaluate if variance in the duplicate samples are similar or different from the site samples. Following the assessment of variance, duplicate, and split samples will be treated for use in the risk assessment as follows:

Duplicates with variance similar to site samples –

- Samples will be treated independently. All results will be carried forward in the quantitative characterization of Site SRCs.

All splits, and duplicates with a variance that differs from site samples –

³SQLs are sample-specific detection limits. They are usually an adjustment from the method detection limit (MDL) and reflect sample-specific actions, such as dilution or use of smaller aliquot sizes, and take into account sample characteristics, sample preparation, and analytical adjustments.

⁴The MDA is the lowest level of activity in a given sample that is statistically distinguishable from a sample with no activity, at the 2-sigma confidence interval. MDAs for radionuclide analysis take into account sample volume, chemical recovery, instrument detection efficiency and background, and sample counting duration.

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- The result of the first sample will be carried forth in the quantitative characterization of Site SRCs. The second QC sample will not be carried forward in a quantitative manner.

Uncertainties associated with the choice of the first sample will be tracked in the risk assessment and discussed in the uncertainty section as relevant.

3.4 SELECTION OF COPCS FOR EVALUATION IN THE RISK ASSESSMENT

More than 300 chemicals and analytical parameters have been identified as SRCs for the Site. To focus the risk assessment on those SRCs that are most important to defining potential human health risks at any given site assessment area, a series of screening steps will be applied to the data to select the particular SRCs to be considered in the risk evaluation. The SRCs selected for evaluation in the risk assessments are termed COPCs.

For purposes of the risk assessments, all analytes have been grouped initially by chemical class. The SRC group classifications presented in Hargis (2008), and presented in Appendix AB, will be used to characterize Site SRCs with the addition of radionuclides.

The following SRCs/SRC Groups will not be selected as COPCs for the risk assessments.

- **General and indicator chemicals.** This group of general analytical parameters (e.g., alkalinity, chloride, pH, sodium, sulfate) was used at the Site primarily to characterize general site conditions (e.g., total inorganic and total organic carbon) or as indicators of the potential presence of other SRCs (e.g., pH as an indicator for acid SRCs, ions for several of the salts). The potential toxicity and risks from this group of SRCs will not be evaluated.
- **Inorganics.** This SRC group as defined in Hargis (2008) is comprised of fluoride, iodide, nitrate, and total carbon, and has been used at the Site primarily to understand general chemical conditions. These SRCs will be used to understand conditions at the Site but will not be separately evaluated for toxicity or risk.
- **Total petroleum hydrocarbons (TPH).** This is another type of a general indicator SRC group (in this case, for petroleum products). Because toxicity is dependent upon the individual constituents that comprise the TPH mixture, the potential toxicity or risks associated with TPH exposure will be evaluated for the constituent SRCs as reported in the database. The potential toxicity and risks from TPH as a whole will not be evaluated separately.
- **Tentatively Identified Compounds (TICs).** TICs will not be selected as COPCs for quantitative evaluation in the risk assessment because of the uncertainty associated with

the identity of these compounds. These data will be evaluated qualitatively; however, and the potential risk implications discussed in the risk assessment.

The remaining SRCs will be further evaluated for selection as COPCs for inclusion in the risk assessments. The primary criteria to be used to select COPCs are a comparison to naturally occurring (background) levels and a comparison to risk-based levels. These steps are discussed in more detail below. For some site assessment areas, a frequency of detection screen may additionally be used to select COPCs, if SRCs are detected infrequently in any given area. This screen would not be used in any site assessment area without prior approval by NDEP, however.

3.4.1 Background Comparison

NDEP and EPA guidance allows for the elimination of constituents from further evaluation if detected levels are not elevated above naturally occurring levels (NDEP 2009b; USEPA 1989). Because metals and radionuclides occur naturally in the environment, concentrations of these constituents will be compared to background concentrations. Metals and radionuclides that are present at the Site at concentrations that are similar to regional background concentrations will not be selected as COPCs.

The background dataset to be used for the background/onsite comparisons will be selected as part of the data usability evaluation. This selection will consider representativeness, comparability to onsite data, and statistical power/sample size of available background datasets. The selection and justification of the background data to be used for onsite comparisons will be included in the data usability evaluations and in the risk assessment reports.

As recommended by NDEP in past communications with the Companies, comparison of onsite and background data will be conducted via hypothesis testing using EnviroGiSdT Software developed by Neptune and Company, Inc. (Neptune 2008a). As outlined in the software's users' manual (Neptune and Company, Inc. 2008b), four two-sample hypothesis tests are conducted as part of background comparisons: the t-test, the Wilcoxon Rank Sum test using the Gehan ranking scheme, the Quantile test, and the Slippage test. Because considering the results of four tests in combination increases the overall false rejection rate for the entire procedure, an adjusted significance level aimed at producing an overall false rejection rate of 0.05 will be adopted for each test. Following NDEP guidance (2009c) a default adjusted rate of 0.025 will be used unless specific limitations on sample size or unusual data characteristics warrant that more specific values be developed. Such values would be developed following NDEP guidance (NDEP 2009c). Specific values used will be included in the HHRA documents. Results from statistical tests, consideration of their robustness and limitations, and graphical displays of the data will be used to determine whether onsite concentrations of metals and radionuclides exceed background concentrations.

In addition to direct comparisons with background data as described above, in cases in which a sufficiently robust data set is available, radionuclide data will additionally be evaluated by analysis of secular equilibrium (SE), following guidance prepared by NDEP (2009b). The presence or absence of SE for onsite data can be used to characterize the source of radionuclides. SE exists when the quantity of a radioactive isotope remains constant because its production rate is equal to its decay rate; under natural background conditions approximate SE is expected. In the case that onsite radionuclide data do not exhibit SE there is an indication of radionuclide-specific contamination (NDEP 2009d).

Natural chemical and physical processes may cause some deviations from SE, and only approximate or quasi-SE can be expected even under the best field and ideal testing conditions. In order to accommodate small differences, equivalence testing, which allows some flexibility in terms of the statistical hypothesis tested, will be employed. The equivalence testing approach will follow the protocols set forth in NDEP guidance (2009d). Standard background comparisons, described above, and when employed the analysis of SE will be considered together in determining whether onsite radionuclides differ from background.

3.4.2 Risk-Based Screening

Soil SRCs that remain after the above screening step will be further screened by comparing to risk-based concentrations. No risk-based screening will be conducted for ~~groundwater, soil vapor, or surface emission isolation flux chamber data;~~ instead, all volatile SRCs that are detected (as determined using screening described in Section 3.1) will be evaluated if detected.

The risk-based concentrations to be used in the screening for SRCs in soil are BCLs developed by NDEP for chemicals and radionuclides (NDEP 2009a,e), BCLs for chemicals are based on direct contact exposure pathways (ingestion, inhalation, and dermal absorption). Radionuclide BCLs are based on ingestion, inhalation, and external radiation. The BCLs correspond to a target excess cancer risk of one in one million (1×10^{-6}), or a noncancer hazard quotient (HQ) of 1. BCLs developed for commercial/industrial settings will be used.

Soil SRCs that remain after the comparison to background levels will be evaluated by comparing the maximum detected concentration to one-tenth the value of the BCL for industrial/commercial land use scenarios (NDEP 2009a,e). Per NDEP guidance, the exceptions to this are lead, which will be compared directly to the commercial/industrial BCL of 800 mg/kg (NDEP 2008g), and titanium, which might be compared to a concentration limit that is lower than one-tenth of the BCL if it is present in substantial amounts in a form other than titanium metal or titanium oxide (NDEP 2008h).

Any organic SRC passing the initial screening steps that has a maximum detected concentration that exceeds the risk-based screening evaluation discussed above will be selected as a COPC for risk assessment. Similarly, any metal SRC or radionuclide that exceeds the risk-based screening

and exceeds regional background levels and/or shows deviations from SE, will be selected as a COPC.

The detection of amphibole or chrysotile fibers will be used to screen asbestos for the quantitative risk evaluation.

4 EXPOSURE ASSESSMENT

The magnitude of exposure for any given receptor is a function of the amount of the constituent in the exposure medium and the frequency, intensity, and duration of contact with that medium. This section presents an overview of the equations and default assumptions that will be used to calculate potential exposures as part of the risk assessments to be conducted at the Site. In cases in which site-specific information on receptor populations or exposure patterns is available site-specific assumptions will be incorporated into the risk assessments in consultation with NDEP.

4.1 GENERAL APPROACHES TO EXPOSURE CALCULATIONS

For non-radiological constituents, oral and dermal exposures are expressed in terms of intake (i.e., mg chemical per kg body weight [BW] per day – mg/kg-day), whereas inhalation exposure is expressed in terms of an exposure concentration (EC) in air (i.e., $\mu\text{g}/\text{m}^3$, f/cm^3 [fibers/ cm^3]). These different expressions of exposure are used to match the toxicity criteria that are available to calculate risks for each type of exposure. For radionuclides, exposure is expressed as total dose in terms of picocuries (pCi).

The general approaches for quantifying exposures for chemicals and radionuclides are discussed below. Appendix C provides the pathway-specific equations and default parameter values. The approaches for quantifying exposures to asbestos are unique and discussed separately later in this section.

4.1.1 Chemical and Asbestos Exposure

Exposure to chemicals and asbestos² for each scenario will be calculated using site-specific concentrations of constituents and receptor- and scenario-specific exposure assumptions.

The following equation is a general form of the equation used to estimate intake for oral and dermal exposures:

$$\text{Intake (mg/kg} \cdot \text{day)} = \frac{\text{CR} \times \text{C} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}} \quad \text{Eq. 4-1 (USEPA 1989)}$$

where,

CR = contact rate (e.g., mg/day)
C = contaminant exposure point concentration (e.g., mg/kg)

²In line with NDEP guidance (2009f) only inhalation of asbestos following suspension of fibers from soil will be evaluated.

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CF	=	conversion factor (e.g., 10 ⁻⁶ kg/mg)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
BW	=	body weight (kg)
AT ^a	=	averaging time (days).

Intake will be expressed in various forms, depending on the risks that it will be used to assess. Average daily dose (ADD) and lifetime average daily dose (LADD) will be calculated and used as measures of exposure from oral and dermal routes, for characterizing noncarcinogenic and carcinogenic effects, respectively.

The EC is a function of a constituent's concentration in air measured at the exposure point and scenario-specific parameters, such as ED and EF. The following equation is a general form of the equation used to estimate the EC:

$$EC (\mu\text{g}/\text{m}^3, \text{f}/\text{cm}^3) = \frac{C_{\text{air}} \times ET \times EF \times ED}{AT} \quad \text{Eq. 4-2 (adapted from USEPA 2009a; NDEP 2009f)}$$

where,

C_{air}	=	contaminant concentration in air ($\mu\text{g}/\text{m}^3, \text{f}/\text{cm}^3$)
ET	=	exposure time (hours/day)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
AT ^a	=	averaging time (hours).

4.1.2 Radionuclide Exposure

Unlike chemicals and asbestos, radionuclide exposure is typically expressed in units of activity per unit of the exposure medium, rather than mass per unit. Exposure to radionuclides may result from internal and external exposure pathways.

Internal exposure is expressed for completed pathways using the following equation:

$$\text{Dose (pCi)} = C \times CR \times EF \times ED \quad \text{Eq. 4-3 (adapted from USEPA 2009b)}$$

where Dose is the dose due to internal exposure, and the remainder of the variables are the same as Equation 4-1 above, except that "C" is the concentration term for soil or air expressed in

^{a,b} When evaluating cancer risk, the averaging time (AT) is equal to a lifetime of 70 years. When evaluating noncancer hazard, the AT is equal to the total exposure duration ED.

units of pCi/g, or pCi/m³, respectively, and "CR" is the contact rate expressed in the relevant units (i.e., g/day, m³/day) for that medium. The body mass and averaging time (AT) exposure factors are not relevant for radionuclides.

For some radionuclides, exposure via certain internal pathways (e.g., oral, dermal, or inhalation) may be insignificant (USEPA 2000). For instance, as reflected by their small dermal absorption and dermal permeability constants, dermal absorption of radionuclides is not an important pathway (USEPA 2000). The inhalation of particulates from dust represents significant exposure for only a few radionuclides (USEPA 2000). Quantitative exposure assessments will only be completed for significant pathways. The selection of pathways for quantitative evaluation will depend upon the radionuclide constituents that are present in and near each site assessment area and will be discussed in the individual risk assessment reports.

The external dose for radionuclide exposure will be calculated using the following equation:

$$\text{Dose (pCi - yr/g)} = C_{\text{soil}} \times [EF/CF_{\text{DY}}] \times ED \times ACF \times [ET_{\text{fo}} + (ET_{\text{fi}} \times GSF)] \quad \text{Eq. 4-4}$$

(adapted from
USEPA 2009b)

where,

C_{soil}	=	exposure concentration term for soil (pCi/g)
EF	=	exposure frequency (days/year)
CF_{DY}	=	conversion factor (days/year)
ED	=	exposure duration (years)
ACF	=	area correction factor (unitless)
ET_{fo}	=	fraction of time spent outdoors (unitless)
ET_{fi}	=	fraction of time spent indoors (unitless)
GSF	=	gamma shielding factor (unitless).

The EF and ED are the same as described above for calculating internal exposures to non-radiological and radiological constituents. As described in the context of internal exposures to radioactive constituents above, "C" is the concentration term for soil expressed in units of pCi/g.

The EPA model for external radiation assumes that an individual is continually exposed to a non-depleting radiological source that is effectively an infinite slab. The concept of an infinite slab means that the thickness of the contaminated zone and its aerial extent are so large that it behaves as if it were infinite in its physical dimensions. Source areas contaminated to a depth greater than 15 cm with an aerial extent greater than 1,000 m² will create a radiation field comparable to an infinite slab (USEPA 2000). The area correction factor (ACF) adjusts for smaller source areas. EPA has derived ACFs for various source area sizes, ranging from 10 to

10,000 m² (USEPA 2009b). These will be used to assess radiological risks at various site assessment areas at the Site.

The gamma shielding factor (GSF) is a factor that accounts for the shielding effect provided by buildings during times of indoor occupancy or by other site features. The fraction of time spent exposed in outdoor and indoor environments is described by ET_{i0} , ET_{i1} , and ET_{i2} , respectively.

4.1.3 Range of Exposure Assumptions

The variables/exposure factors shown in the exposure algorithms above vary depending on the receptor population being evaluated. Each receptor population will be characterized by a number of assumptions regarding the frequency of contact with potentially contaminated media, duration of exposure, and other parameters unique to the receptor population.

EPA (1992c) guidance for Superfund sites recommends that discusses two types of exposure estimates that may be calculated in a HHRA; the reasonable maximum exposure (RME), and the central tendency exposure (CTE). The reasonable maximum exposure (RME) is defined as the highest exposure that could reasonably be expected to occur for a given exposure pathway at a site. The RME is intended to account for both uncertainty of the contaminant concentration and variability in exposure parameters. The CTE is designed to reflect EPA also recommends that an average estimate of exposure, termed "central tendency exposure" (CTE), be presented in the risk assessment. Both RME and CTE estimates will be calculated for the risk assessments. The single exception is for lead. The ALM is sensitive to upper end values, and specifies the use of central tendency soil lead concentrations (USEPA 2003a); therefore, in the case that lead is brought forth as a COPC in the risk assessment, only CTE estimates will be calculated for the chemical⁹. CTE estimates for other SRCs may additionally be calculated and presented in the uncertainty analysis as a means to provide context to the RME evaluation.

The specific equation and assumptions used to estimate exposure varies, depending on the exposure route being evaluated. Appendix C presents a complete set of exposure equations along with the specific exposure assumptions that will be used for contact rate, ET , EF , and ED ; body weight BW ; and AT for each pathway and receptor group. It additionally presents exposure factors specific to radionuclide exposures including ET_{i0} , ET_{i1} , and GSF . In cases in which site-specific information on receptor populations or exposure patterns is available, site-specific exposure factors will be incorporated into the risk assessments.

General assumptions that are applicable to exposure estimates are discussed in Section 4.2 below. In addition to exposure assumptions, COPC concentration in the exposure medium at

⁹In the case that lead is brought forth as a COPC in the risk assessment, only CTE estimates will be calculated. The adult lead methodology (ALM) is sensitive to upper end values, and specifies the use of central tendency soil lead concentrations (USEPA 2003a).

the point of contact are required for evaluating risks. Section 4.3 describes the approaches used to estimate exposure point concentrations (EPCs).

4.2 GENERAL INTAKE ASSUMPTIONS

Exposure assumptions for ED, EF, ET, BW, and AT are discussed below. EPA guidance was used as the basis of these values, if available.

4.2.1 Exposure Duration

The ED is the length of time during which someone may be exposed to a particular medium via a specific exposure pathway. The ED varies depending on the population being evaluated. Both chronic and subchronic exposures will be assessed at the Site, depending upon the receptors evaluated. EPA (2009a) defines chronic exposures as repeated exposures that occur over 7 years¹⁰ or more, and subchronic exposures as repeated exposures that occur over a period greater than a month and less than 7 years.

For a typical indoor or outdoor occupational worker, chronic exposures are evaluated. EPA (2002a) recommends a RME ED of 25 years. This value is based on U.S. Census data and represents an upper bound estimate for the length of time a person works at the same location. The average, or CTE, value for occupational ED is assumed to be 7 years, which is the median occupational tenure of the working population (USEPA 1997a).

Construction workers are expected to work on limited-term projects, such as building construction, and are assessed for subchronic exposures. If multiple construction projects occur on the Site, it is assumed that different workers will participate in each project. EPA recommends an ED of 1 year for construction workers (USEPA 2002a). For this risk assessment, based on best professional judgment, a value of 6 months is proposed as the CTE value. Site-specific values will be substituted for these defaults when available and in consultation with NDEP.

~~Trespassers also will be evaluated for subchronic exposures. The trespasser scenario to be used for the Site assumes that an older child from the surrounding community accesses the Site without permission. EPA guidance on the duration of exposure for trespassers is not available. Given the assumption that the trespasser is age 7-12, a RME and CTE ED of 6 years, and 3 years respectively, are proposed for this scenario at the Site, based on best professional judgment.~~

~~RME and CTE EDs for the offsite resident are 30 years and 9 years, respectively. These values represent the 95th and 50th percentile values for years lived in the same house (USEPA 1997a). It is assumed that 6 and 2 years of the 30 and 9 year periods respectively, occur as a child. The~~

¹⁰Seven years is one-tenth of an EPA-assumed standard lifetime of 70 years.

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decision to assume 2 years of the 9-year CTE ED as the portion occurring during childhood is based on best professional judgment; the value is approximately equal to the ratio of 24 adult years to 6 child years, recommended by EPA and applied for the RME scenario.

4.2.2 Exposure Frequency

EF describes how many days someone may have contact with the exposure media of interest in a typical 1-year period.

EPA recommends an RME EF of 250 days/year for indoor workers and 225 days/year for outdoor workers (USEPA 2002a). These values will be adopted for the default RME and CTE cases for these receptor groups. EPA recommends an EF of 250 days/year for construction workers (USEPA 2002a). This value will be used as the default RME and CTE value. Site-specific values will be used in lieu of defaults when available.

Guidance is not available for the number of days that trespassers could be assumed to enter a site. Considering the sparse rainfall that occurs in the area, it is possible that the trespasser might access the Site throughout the year. Based on best professional judgment it is conservatively assumed that the trespasser accesses the Site an average of 1 day/week throughout the year, and an EF of 50 days/year is recommended for the RME and CTE value.

An EF of 350 days/year is assumed for offsite residents, as recommended by EPA (1991).

4.2.3 Exposure Time

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The ET is the amount of time each day which someone may be exposed to a particular medium via a specific exposure pathway.

ET is assumed to be 8 hours/day for the indoor worker, outdoor worker, and construction worker. The entire 8 hour period is assumed to be spent indoors for the indoor worker; while the entire 8 hour period is assumed to be spent outdoors for the outdoor and construction workers.

4.2.3.4 Body Weight

A value of 70 kg (154 lbs) represents the BW for all adult receptors, based on average male and female adult BWs (USEPA 1991). This value will be used for all RME and CTE worker scenarios, and offsite adult residents. A BW of 15 kg is assumed for the offsite child resident (USEPA 1991). These values will be used for CTE and RME scenarios. The trespasser is assumed to be an individual between 7 and 12 years of age. A value of 35 kg (77 lbs), based on average male and female BW for this age category, will be used for CTE and RME scenarios.

(USEPA 1991). This parameter is not included in dose estimation for radionuclides (USEPA 1989).

4.2.44.2.5 Averaging Time

The AT is the period over which an exposure is averaged. The ATs for evaluating carcinogenic and noncarcinogenic effects are different, and are expressed in different units dependent on the exposure route being evaluated. For evaluating carcinogenic effects, chemical intakes are averaged over a 70-year lifetime (25,550 days; 613,200 hours) to be consistent with the method by which cancer slope factors (CSF) and inhalation unit risks (IURs) are derived.

When evaluating noncarcinogenic effects, chemical intakes are averaged over the ED (USEPA 1989). Therefore, for noncarcinogenic effects, the ED is converted to days or hours and is used as the AT_{nc}. For example, the RME AT for the outdoor occupational and indoor worker is 25 years, or 9,125 days, or 219,000 hours; the RME for trespassers is 6 years, 2,190 days, or 52,560 hours; and the RME AT for the nearby offsite residents (adult and child component) is 30 years, 10,950 days, or 262,800 hours.

This parameter is not included in dose estimation for radionuclides (USEPA 1989).

4.3 EXPOSURE POINT CONCENTRATIONS

EPCs will be estimated using measured concentrations of chemicals and radionuclides in environmental media alone or in combination with fate and transport models. Methods for deriving EPCs in soil, airborne particulates, and ambient and indoor air vapors are described below.

4.3.1 Exposure Point Concentrations in Soil

Soil EPCs will be calculated to estimate direct contact exposure for onsite workers. The soil EPCs could also be used as inputs to emission models used for deriving airborne concentrations of SRCs released into the atmosphere as particles or vapors.

EPCs from soil in an exposure area will be derived using data results from soil samples taken within the site assessment source or waste management areas. Representative EPCs will be based on the potential exposure depth interval for each receptor. For receptors exposed to surface soil (e.g., for indoor workers, outdoor workers, trespassers), two EPCs will be calculated. For the first, data from the top 6 in. of soil will be used. For the second, a vertical average from the surface to 10 ft bgs will be used. The second EPC assumes a redevelopment scenario in which soil from the surface to 10 ft bgs is reworked and brought to the surface (i.e., 0-6 in. bgs). For receptors exposed to deeper soils (e.g., construction workers) data from the surface to 10 ft bgs will be used.

When developing the soil EPCs, the exposure areas will be combined to the greatest extent possible to make the largest decision units that can be justified for the source/site assessment area. Accordingly the modality of the data will be evaluated and areas of localized elevated concentrations will be evaluated as a separate decision unit, if necessary.

To estimate exposures that are representative of upper end exposures, EPA (1992c) recommends using the 95th upper confidence limit (UCL) of the arithmetic mean concentration. As recommended in past communications with NDEP, 95% UCLs will be estimated using EnviroGiSdT Software (Neptune and Company, Inc. 2008a). EnviroGiSdT provides three methods for computing the UCL; the Student's t- UCL and two bootstrap UCL methods. For each COPC the sample size, frequency of detection, and data distribution will be evaluated in order to select the appropriate method for computing a UCL. The EnviroGiSdT Software's default setting uses one-half the SQL/MDA, or reported value for radionuclides for non-detected results when computing the 95% UCL. If the substitution of one-half of the SQL/MDA, or reported result for radionuclides for non-detects appears to be driving the risks, alternative substitution methods for non-detects may be explored within the uncertainty evaluation.

Further refinement of the EPCs will be considered based on the HHRA results estimated using the 95% UCL analysis. For example, more refined EPCs can be derived using area-weighted or spatial statistics using Thiessen polygons. Any refinement to the EPC calculation method will be discussed with NDEP prior to implementation in the HHRA.

In the cases that lead is brought forth as a COPC in the risk assessment, the arithmetic mean concentration of lead in soil will be adopted as the EPC for estimating risk. The Adult-Lead Methodology (ALM_L), which will be used to characterize risks from exposure to lead, is sensitive to upper end values, and specifies the use of central tendency soil lead concentrations (USEPA 2003a).

Results of statistical analyses conducted to characterize the distribution of the data and the recommended UCL will be provided in the risk assessment.

4.3.2 Airborne Particulates

Airborne particulate concentrations will be calculated for dust emissions sources within a given source/site assessment area. For the purpose of this RAWP, airborne particulates will include nonvolatile chemicals, radionuclides, and asbestos. The emissions and dispersion modeling described in this section will be applied to all airborne particulates evaluated in the risk assessments. However, there are unique analytical data handling procedures used to develop the asbestos concentration to be used in the emissions models. These unique asbestos procedures are detailed at the end of this section on airborne particulates.

4.3.2.1 Emissions and Dispersion Modeling for Chemicals and Radionuclides

There are two primary sources of dust emissions at the Site: wind erosion, and soil disturbances associated with construction activities. For source areas where construction scenarios are not assumed to occur, wind erosion emissions are the only concern. For the purpose of this discussion such non-construction scenarios dust emissions from wind erosion alone are defined as the non-construction emissions. The airborne particulate concentrations will be calculated separately for dust emissions from wind erosion and from construction-related activities. Dust emissions from construction activities are assumed to occur for a limited period (i.e., no more than 1 year) whereas emissions from wind erosion can occur throughout the assumed exposure period for a receptor. If construction activities are evaluated for a source site assessment area, a time-weighted airborne particulate concentration will be calculated for all receptors, except the construction worker, to reflect the combined emissions from short-term construction activities and long-term wind erosion. For construction workers, the airborne particulate concentration for the risk assessment will be based only on the dust emissions during construction.

For most SRCs, the incidental ingestion and dermal absorption exposure pathways to be quantified in the site risk assessment will result in higher potential health risks than the inhalation pathways. Therefore a tiered modeling approach that progresses from a simplified and upper-bound assessment to a refined and more accurate estimate of potential health risks is proposed for evaluating inhalation exposures related to airborne particulates released from the source site assessment area being addressed. The first tier for estimating airborne particulate concentrations will be based on the simplified site-specific PEF methods modeling provided in EPA's *Soil Screening Guidance* (USEPA 2002a). If the inhalation pathway risks based on the simple site-specific method drive the overall risks to the site assessment area, then more refined and less conservative tiers will be used. The proposed methodology for the more refined analysis will be provided to NDEP for approval as an interim deliverable for the relevant site assessment area. Procedures for estimating non-construction dust emissions and construction emissions for the first tier evaluation are described briefly below. Appendix D provides the more complete details on the dust emission modeling proposed for the first tier.

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Non-Construction Dust Emissions

The non-construction dust emission scenario for a site assessment area will be exposure to wind erosion. A particulate emission factor (PEF) equation for wind erosion provided by EPA (2002a) will be used for estimating the chemical concentration in air associated with the surface soil concentration of the source. The soil concentrations for COPCs will be based on the 95% UCL for soils 0-6 in. bgs as described in Section 4.3.1. The area used in the PEF equation will reflect the size of the exposure source area represented by the 95% UCL soil concentration. In cases where modality is observed in the soil data, more than one wind erosion PEF may be required to address the entire source site assessment area. The fraction of vegetative cover will consider the land cover of the area being assessed; the value will be specified in the risk

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~~assessment for the given source area be set at zero in the first tier analysis, which optimizes the potential for wind erosion emissions from the surface soils.~~

An integral part of the PEF equation is the dispersion factor which provides an estimate of the dilution that occurs during transport from the emission source to the point of exposure once dust is released into the atmosphere. The dispersion factor is linked to the PEF to calculate the airborne particulate concentration. The EPA (2002a) dispersion factor used in the wind erosion PEF equation assumes that the receptor is located either at the edge, or in the center, of the emission source. ~~Offsite residents are assumed to be located at the downwind edge of the wind erosion source area. Construction workers for the simple first tier screening analysis, all workers are assumed to be located at the center of the source area. Onsite workers not involved in construction activities and trespassers are assumed to be post construction receptors within the exposure area. Thus they only experience dust emissions from non construction activities. The exposure is assumed to occur at the center of the source area. The PEF model generates dispersion factor equation provides estimates of the ambient air dust concentrations. For receptors that spend all or some of their day indoors (i.e., indoor workers and offsite residents, respectively), an dilution attenuation factor will be used to scale the ambient air dust concentrations to indoor air dust concentrations.~~

A sensitivity analysis will be conducted for the PEF model inputs to evaluate their impact on uncertainty in the risk estimates. Complete listings of the PEF equations and input values for the non-construction emissions are presented in Appendix D.

Construction Dust Emissions

If construction activities are anticipated to occur within for a site assessment source area then relevant PEF equations from EPA (2002a) will be applied to estimate associated airborne concentrations for construction workers and offsite residents. Onsite workers not involved in construction activities and trespassers are assumed to enter begin this the exposure areas with the decision unit post construction. EPA (2002a) has identified vehicle traffic as the most significant contributor to fugitive dust emissions during construction activities. Dust emissions for construction activities will be based in part on assumed vehicle traffic over unpaved surface soil. In addition, dust emissions from various construction activities (i.e., excavation, dozing, grading, and tilling) will also be calculated. The total outdoor ambient air dust concentration for construction activities will be estimated based on the combined contributions from wind erosion, vehicle traffic, and construction activities.

The soil disturbance area to be modeled ~~in the for the construction activity~~ PEF construction equations will be dependent on the size and characteristics of the assumed construction activities for the HHRA. site assessment area being addressed. A primary characteristic is the

soil concentrations used in this modeling, which will be based on the 95% UCL for the source area soil 0-10 ft bgs. As discussed in Section 4.3.1, the 95% UCL calculation will include an evaluation of modality to identify the largest justifiable decision unit within the source a-site assessment area. The soil disturbance area used in the construction activity PEFs will be reflective of the decision-unit exposure area represented by the soil 95% UCL value. ~~In some cases this may require more than one construction PEF be calculated to address a given site assessment area.~~

The air dispersion factor used in the construction activity PEFs depends upon the location of the receptor relative to the dust emission sources. Construction workers will be assumed to be located at the center of the emission source for the duration of the exposure. ~~Residential receptors are assumed to be located at the downwind edge of the emission source. Because residential receptors spend some portion of their day indoors, a dilution factor will be used to scale the ambient air dust concentrations to indoor air dust concentrations when calculating daily exposures.~~

A sensitivity analysis will be conducted for the PEF model inputs to evaluate their impact on uncertainty in the risk estimates. A complete listing of the PEF equations and input values for the construction dust emissions is presented in Appendix D.

4.3.2.2 Asbestos Airborne Exposure Point Concentrations

Asbestos concentrations in site soils have been characterized using an elutriator method that reports the number of asbestos structures detected per mass of respirable dust (Berman and Kolk 2000). The intent of this method is to provide analytical asbestos measurements that can be directly combined with standard dust emission and dispersion models to predict airborne asbestos concentrations associated with soil disturbances that would release respirable dust. This asbestos measurement methodology was employed as part of an NDEP-approved sampling and analysis plan for site soils (PES 2006). This methodology has also been employed for NDEP approved risk assessment activities at adjacent sites (ERM 2007) and is specified for use in the recent NDEP technical guidance for calculating asbestos risks from soils (NDEP 2009f).

The asbestos sampling results from the elutriator method are reported as structures per gram of respirable dust¹¹. The emissions and dispersion modeling discussed in Section 4.3.2.1 describes the approach for estimating the respirable dust concentration in air resulting from the various soils disturbing activities anticipated at the Site and is consistent with the NDEP (2009f) guidance for calculating asbestos risks from soils. The product of the airborne respirable dust concentration and the asbestos elutriator results yields an estimate of the airborne asbestos concentration that can be used in calculating potential human health risks as described in

¹¹ Concentrations are based on fibers observed in a sample multiplied by the analytical sensitivity of the measurement. In the case that more than one asbestos sample is collected than the pooled analytical sensitivity is used.

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Sections 5 and 6 of this RAWP. Following NDEP guidance (2009f) EPCs will be calculated by multiplying the UCL for the number of fibers observed by the analytical sensitivity (in the case that only a single sample is available) or pooled analytical sensitivity (in the case that multiple samples are available). The uncertainty associated with assumptions used for deriving the EPC will be addressed by discussing the variability in the sample results and the risk implications of using other inputs to characterize asbestos concentrations in soil.

4.3.3 Vapor Assessment

Inhalation exposures for vapors released from soils and groundwater will be evaluated for all worker and resident populations. Several types of Data data applicable to vapor assessment could may be available (e.g., surface emission isolation flux chamber data, soil gas data, soil data, and groundwater data); however, only soil gas data is being considered for the quantitative risk assessments¹². ~~come from several different investigative techniques. Each technique has inherent uncertainties in predicting EPCs and associated inhalation risks. The use of multiple lines of evidence to assess the vapor pathway is considered the best way to reduce uncertainty in the assessment (ITRC 2007; USEPA 2008b). Accordingly, more than one type of data may be available for the vapor assessment at a site assessment area. The comparability of the available data types will be assessed in the data usability evaluation conducted for the site assessment area prior to the risk assessment. The remainder of this section describes generally how soil gas data will from each anticipated vapor investigation technique could be used to develop EPCs for the risk assessments. Details of the modeling approach, including model input parameter values, will be developed in consultation with NDEP for approval prior to completion of the HHRA. Ultimately, whatever investigation technique is used will be based on an NDEP approved SOP that describes the modeling and risk assessment procedures as needed.~~

4.3.3.1 Surface Emission Isolation Flux Chamber Data

~~Surface emission isolation flux chamber (flux chamber) data may be collected at the Site for use in evaluating inhalation risks from volatiles released from soil and/or groundwater. Flux chamber data is obtained by placing an enclosure directly on the surface to be monitored and collecting vapors as they enter the chamber. EPA has produced technical guidance for measurement of gaseous emissions using flux chambers (Kienbusch 1986) which will serve as a primary reference for this technique. Flux chamber testing has been conducted and used for vapor assessment at adjacent sites in the BMI Complex based on a collection procedure approved by NDEP (SOP 16, BRC 2008). This RAWP assumes that any flux chamber data available for vapor assessment at the Site will be collected in a manner approved by NDEP.~~

¹² Other data types may be considered for comparative purposes in the data usability evaluation conducted for the source/site assessment area or as a prior to the risk assessment secondary line of evidence discussed in the uncertainty section of the HHRA.

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The results of the flux chamber testing are presented as a chemical-specific flux rate in units of mass over area and time (e.g., $\mu\text{g}/\text{m}^2 \cdot \text{min}$). For use in the risk assessment, the chemical-specific flux rate must be converted to an air concentration. This conversion is made by linking the flux rate to a dispersion model that predicts the dilution of the vapors in the exposure environment relevant to the receptor population being evaluated. The chemical-specific flux rate adopted in the modeling will be the maximum flux rate of all data collected within the site assessment area. The 95% UCL of all data collected within the site assessment area for a chemical will also be reviewed as a means of addressing the uncertainty in the risk estimates based on the maximum flux rate.

The dispersion model used to develop EPCs from flux chamber data depends on the receptor location. For indoor worker exposures, flux rate data would be linked to an indoor air model to estimate indoor air concentrations. The ASTM (2004) provides an equation that combines the flux rate with parameters that balances the infiltration rate of the vapors into the indoor air and the volume and ventilation rate in the building to predict a steady-state air concentration. Input parameters for the indoor air model will be developed in accordance with NDEP approved methodologies (i.e., SOP 16, BRC 2008). For exposures assumed to occur outdoors in the ambient air (i.e., outdoor workers, construction workers, residents, trespassers) the default approach would be to link the flux rate data with a dilution factor developed by EPA (1996a) based on dispersion modeling that reflected annual average wind flow and mixing conditions for the Las Vegas, NV area. Because of the conservative nature of this default dilution factor, more refined site-specific modeling may be employed using EPA recommended air dispersion models. However, indoor air exposures generally drive inhalation risks relative to ambient air exposures for the same vapor source. Therefore, refinement of the ambient air modeling may not be required. Details on the equations used to calculate ambient and indoor air concentrations based on flux chamber data are provided in Appendix E.

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4.3.3.2 Soil Gas Data

Soil gas data used for the risk assessment will be obtained via an NDEP approved methodology and standard operating procedures (SOPs). There are a wide variety of soil gas sampling methods available; however, they can be divided into either active or passive methods. For the purpose of this RAWP it is assumed that only active soil gas sampling will not be used in calculating a quantitative estimate of inhalation risk because of the difficulties in converting the passive soil gas resulting measurements to a soil gas concentration. Active soil gas sampling consists of driving a probe into the soil and extracting a soil gas sample for laboratory analysis. The results of active soil gas sampling are reported as a concentration in units of mass over volume (e.g., mg/m^3). This soil vapor gas concentration must then be scaled to a representative air EPC for use in the risk assessment. EPCs would be required for ambient and indoor air may be required in order to evaluate the full range of receptors and exposure pathways identified for the Site. Emissions and dispersion modeling will be conducted to scale the soil gas vapor concentrations to the appropriate inhalation EPCs. This modeling will be

conducted separately for the indoor and ambient air exposures because of the differences in the infiltration rates and dilution that occur for soil gas vapors entering ambient versus indoor air. Indoor and ambient air EPCs are discussed below.

4.3.3.1 Indoor Air Exposure Point Concentrations

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For indoor air concentrations, the soil vapor gas concentration will be scaled through the use of attenuation factors. Indoor air exposures at the Site will be in commercial or industrial buildings. Default attenuation factors are available from the California Environmental Protection Agency (Cal/EPA;) (2005) for commercial buildings. The default attenuation factors would will be used with the maximum detected soil vapor concentration in the site assessment area to provide an initial screening of the potential inhalation health risks. For chemicals detected in soil gas that present an elevated health risk based on the initial screening, a more refined and site-specific approach based on the Johnson and Ettinger (I&E) vapor intrusion model would will be executed.

The ~~Johnson and Ettinger (I&E)~~ vapor intrusion model has been used by EPA (2002b) for developing attenuation factors for soil gas infiltration into residential-indoor air. The I&E model predicts the rate of transport of volatile chemicals through the vadose zone and into indoor air. The transport through the vadose zone is a response to the concentration gradient modeled using Fick's First law. The diffusion in soil is described by an effective diffusion coefficient that is based on chemical-specific diffusivity values and soil porosity. At the interface of the vadose zone and building foundation, the ~~Johnson and Ettinger~~ model uses an approximation of the convective flux to estimate the rate at which the vapors would be drawn into the indoor air. The infiltration rate of vapors from the soil is balanced with the exfiltration rate of gases from the above-ground portion of the building to estimate the steady-state indoor air concentration.

Several versions of the ~~Johnson and Ettinger~~ model are available from EPA (2004b) depending on the nature of the source being modeled. The spreadsheet model developed for use with soil gas will be applied for this evaluation. Inputs will be a mixture of chemical- and site-specific values along with recommended defaults ~~from EPA (2004b)~~. The chemical-specific inputs are comprised of the soil gas concentration and various chemical properties (e.g., diffusivity and Henry's Law constant). The input soil gas concentrations will be generated from the source area soil vapor sampling. The chemical properties will be the default values included withfor the I&E model. Site-specific parameters will be used when available for the soil and building properties required for the I&E model. Default parameters values from ASTM (2000) for commercial buildings will be used when site-specific information is unavailable. The model predicts a chemical specific attenuation factor and associated indoor air concentration as a function of the input values, including soil gas concentration. The 95% UCL on the mean soil gas concentration for the site assessment area will be used as the input for the model calculations. If the lateral dimensions of the soil gas sampling locations for a site assessment

area are larger than the assumed building footprint, then sampling locations will be grouped into smaller units for calculation of 95% UCL values and used in the model to better represent the possible source area for the assumed building. Conversely, if the available soil gas data are not sufficient to construct a defensible 95% UCL within the assumed building footprint, then the maximum concentration will be used. Additionally, soil gas sampling results may be available from a variety of depths bgs. In most cases the shallowest soil gas concentration would be most relevant for modeling infiltration into indoor air. However, the full range of soil vapor data that passes through the data validation for the site assessment area will be considered in the risk assessment. Additional details on the Johnson and Ettinger model and the input parameters can be found in Appendix E.

4.3.3.2 Ambient Air Exposure Point Concentrations

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Based on the results of the indoor air risk assessment it may not be necessary to quantitatively evaluate ambient air exposures EPCs in ambient air associated with soil vapor concentrations as indoor air concentrations will be orders-of-magnitude higher than ambient air concentrations. If necessary, the ambient air concentrations will be determined using a will also be estimated using an emissions and dispersion modeling approach. A steady-state Fickian diffusion model will be used to predict the flux of vapors through the soil and into ambient air. The vapor flux emissions model is based on an effective diffusion coefficient in soil, the soil gas concentration, and the distance the soil gas vapor must travel to reach the soil surface. The effective diffusion coefficient in soil is calculated from the chemical-specific air diffusivity values and soil porosity values. The chemical-specific diffusivity values and soil porosity values will be consistent with the values used in the J&E modeling for the indoor air concentrations. The soil gas sampling for the source area will be used to generate values of the soil gas concentration and travel distance in the vadose zone. input soil vapor concentration will be the 95% UCL soil gas concentration for the site assessment area. The range of vertical soil gas samples available for use in the risk assessment will be reviewed to determine the most relevant data to retain in the calculation of the 95% UCL.

The ambient air concentrations for vapors released from soil to ambient air will be estimated using the dispersion factor presented by EPA (1996a). The dispersion factor proposed for use in the risk assessment is based on meteorological data collected from Las Vegas, NV. The EPA (1996a) provides a range of dispersion factors depending on the size of the source area being evaluated. The available dispersion factor that is based on a source size that most closely matches the site assessment area being considered in a risk assessment will be selected. Details of the dispersion factor equation are provided in Appendix E.

4.3.3.3 Soil Data

Soil sampling data collected at the Site will be available for consideration in the vapor assessment. However, the soil vapor concentration for each chemical must be estimated using

partitioning equations based on physicochemical parameters and soil conditions. This extrapolation requirement makes the use of soil data in vapor assessment less preferable to the direct measurement techniques discussed above (i.e., flux chamber and soil gas). Soil data may still be useful as a line of evidence to augment other direct measurement techniques for assessing soil vapor. If the data usability analysis for a site assessment area indicates that soil data are available, the following discussion summarizes how it could be used in the vapor assessment.

For outdoor workers, residents and construction workers, the soil data could be used to estimate exposures based on the soil volatilization model provided in the EPA *Soil Screening Guidance* (USEPA 1996a). For indoor workers the soil data could be used to estimate exposure based on the Johnson and Ettinger model of vapor intrusion. Each of the volatilization models is described in the following text.

For outdoor workers, residents and construction workers, the inhalation exposure to volatile chemicals released from soil will be conducted using a volatilization factor (VF) approach. Like the PEF, the VF is composed of an emission component and a dispersion component. The volatilization component is based on a time-dependent equilibrium model that assumes an infinite source of contamination. An average flux rate would be calculated by integrating the time-dependent model over the exposure period assumed for each receptor. The AT used in the model will match the ED for all receptors with the exception of subchronic exposures assumed for construction workers (i.e., less than 7 years). For subchronic exposures the AT will be set to 6 years to avoid overestimation of the emissions that would occur by assuming a shorter AT. Modeling with shorter ATs would in effect reflect a fresh spill rather than the older residual sources of contamination at the Site. Site-specific values collected using approved NDEP methodologies will be used for the soil characteristics related to porosity and density to the extent possible. Chemical-specific soil concentrations used in the modeling would be based on the 95% UCL for the upper portion of the vadose zone in the site assessment area. Appendix E provides a summary of the model equations and input values.

The dispersion factor portion of the VF model for outdoor exposures is based on air modeling conducted for a range of source sizes and climatic regions throughout the United States. We propose to use the dispersion factor developed by EPA (1996a) using meteorological data for Las Vegas, NV. In addition, we will modify the dispersion factor to reflect the portion of the site assessment area which is assumed to represent the source extent for the emissions modeling.

The flux rate of chemicals from the soil can be increased due to soils handling activities related to construction. EPA (2002a) has determined that the conservative nature of the infinite source model used to model volatilization from soil, which assumes that volatile contaminants are present at the soil surface, should be protective of offsite residents, even for periods of construction activities. A sensitivity analysis will be conducted to determine the potential

increase in exposure and risk estimates based on the potential increase in the flux rate during construction activities. The sensitivity analysis will also compare the model predictions to other volatilization-related studies conducted within the immediate vicinity of the Site as a means to evaluate the uncertainty in the risk estimates.

For onsite indoor workers the inhalation exposure related to release of volatile organic chemicals in soil will be evaluated using the EPA's Johnson and Ettinger model (USEPA 2004b). The model to be used for the risk assessment is contained in spreadsheets available from EPA. For this application the version that deals with soil sources will be used. The Johnson and Ettinger models available from EPA contain defaults for all of the physical parameter values needed to conduct the modeling. We propose to use site specific parameters for physical properties required to describe soil and groundwater properties (e.g., porosity, vadose zone temperature, depth of soil contamination). The default building characteristics contained in the EPA Johnson and Ettinger models will be used in this evaluation unless site specific information is available. These include parameters such as vapor flow rate into the building, the building lateral footprint, indoor mixing height, and air exchange rate.

The input soil concentration for the COPCs evaluated in the vapor intrusion model will be based on the 95% UCL soil concentrations found in the upper vadose zone for the assumed building footprint for the site assessment area. If a 95% UCL cannot be calculated for the building footprint, the maximum value will be used. A sensitivity analysis will be conducted for the model inputs to evaluate the uncertainty in the risk estimates. A detailed discussion of the model and the input parameters, are presented in Appendix E.

4.3.3.4 Groundwater Data

Groundwater data are also available for the Site and could be used as a line of evidence in the vapor assessment. Inhalation exposures to volatile chemicals in onsite groundwater could be evaluated for all worker and resident populations. For outdoor workers, residents and construction workers, the exposures will be based on the groundwater volatilization model provided in the EPA *Soil Screening Guidance* (USEPA 1996a). For indoor workers the exposure will be based on the Johnson and Ettinger model of vapor intrusion. Each of the volatilization models is described in the following text.

For outdoor workers, offsite residents and construction workers, inhalation exposures to volatile chemicals released from groundwater are assumed to occur in ambient air. A VF approach will be used to characterize this exposure. The volatilization model is a steady-state equilibrium model that describes the transport of vapors through the vadose zone and into ambient air. The model assumes that groundwater is the source of the vapors in the vadose zone and not the soil column. The movement of the vapor is calculated based on Fick's First law. The diffusion is in response to the concentration gradient across the vadose zone and can be described by an effective diffusion coefficient. The total effective diffusion coefficient will be

calculated based on a two-component approach, as used in the soil vapor intrusion modeling via the Johnson and Ettinger model. The first component described is the effective diffusion across the capillary zone, and the second component reflects movement in the vadose zone.

The dispersion factor for groundwater volatilization to ambient air will be based on a dilution factor presented by EPA (1996a). The dilution factor was based on the modeling conducted using the Industrial Source Complex air dispersion model. The air dispersion model was run for a range of square area sources from 0.5 acres to 30 acres in size. A unit flux rate (i.e., $1 \text{ g/m}^2\text{-sec}$) was used in the model to generate normalized ambient air concentrations at the center of the source area (i.e., kg/m^3 per $\text{g/m}^2\text{-sec}$). The normalized air concentrations will be used with site-specific volatilization flux rates to yield the associated ambient air concentrations. Normalized ambient air concentrations were calculated for 29 locations throughout the United States, including Las Vegas, NV. The model results for Las Vegas, NV will be used in this risk assessment. The dilution factor selected for use in the risk assessment will be adjusted to reflect the area of the relevant groundwater source at the Site. The volatilization and dispersion factors will be combined to develop the VF for the groundwater to ambient air pathway. Details of the equations and inputs to be used for exposure pathway are presented in Appendix E.

For onsite indoor workers the infiltration of volatile chemicals from groundwater into indoor air will be evaluated using EPA's Johnson and Ettinger model (USEPA-2004b). The model predicts the rate of transport of volatile chemicals through the vadose zone and into indoor air. The transport through the vadose zone is modeled as a response to the concentration gradient using Fick's First law. The diffusion is described by an effective diffusion coefficient that is based on chemical-specific diffusivity values and soil porosity. At the interface of the vadose zone and building foundation, the Johnson and Ettinger model uses an approximation of the convective flux to estimate the rate at which the vapors would be drawn into the indoor air. The infiltration rate of vapors from the soil is balanced with the exfiltration rate of gases from the above-ground portion of the building to estimate the steady-state indoor air concentration.

The model to be used for the risk assessment is contained in spreadsheets available from EPA. For this application the version that deals with groundwater sources will be used. The Johnson and Ettinger models available from EPA contain defaults for all of the physical parameter values needed to conduct the modeling. We propose to use site-specific parameters for physical properties required to describe soil and groundwater properties (e.g., porosity, vadose zone temperature, depth to groundwater). The default building characteristics contained in the EPA Johnson and Ettinger models will be used in this evaluation unless site-specific values are available. This includes parameters such as vapor flow rate into the building, the building lateral footprint, indoor mixing height, and air exchange rate.

The input groundwater concentration for the COPCs evaluated in the vapor intrusion model will be based on either the 95% UCL or the maximum for concentrations found within the relevant building footprint for the site assessment area. A sensitivity analysis will be conducted

for the model inputs to evaluate the uncertainty in the risk estimates. This analysis will include consideration of localized areas of elevated groundwater concentrations relative to the 95% UCL based on all data within the site assessment area. A complete listing of the inputs values for the model parameters, except for chemical specific parameters, is presented in Appendix E.

5 TOXICITY ASSESSMENT

The purpose of a toxicity assessment is to summarize health effects that may be associated with exposure to the constituents included in the risk assessment and to identify doses that may be associated with those effects. The focus of the toxicity assessment will be on effects associated with repeated long-term exposures and on effects that could be associated with the chemical and radionuclide concentrations and pathways of exposure that are relevant for this Site. Toxicity values developed based on dose-response assessments for these relevant adverse effects will be identified. These toxicity values are numerical expressions of dose and response, and vary based on factors such as the route of exposure (e.g., oral or inhalation) and duration of exposure (e.g., subchronic, chronic).

In assessing the potential toxicity of chemicals and radionuclides, duration of exposure is an important factor because the exposure levels that can cause toxic effects are usually lower when exposures continue for a longer period of time. For example, with continuous exposure to a chemical for many years (typically referred to as chronic exposure), much lower concentrations (and resulting doses) of a chemical could be associated with toxic effects, compared with concentrations that would be identified as causing toxic effects in a person who is exposed to a chemical for only one day (referred to as an acute exposure). Intermediate duration exposures (referred to as subchronic exposures) are more likely to suggest toxic effects at intermediate concentrations. The risk assessments will evaluate risks associated with scenarios involving subchronic and chronic exposures to COPCs on and around the Site; acute exposures will not be evaluated quantitatively.

The following section describes the procedures that will be used to identify and assess toxicity information. Additional discussion is provided for the approach used to assess the toxicity of asbestos and mixtures of dioxins/furans and carcinogenic polycyclic aromatic hydrocarbons (PAHs).

5.1 METHODS FOR TOXICITY ASSESSMENT

Standard procedures, per EPA (1989 and others) will be followed to identify and assess toxicity factors and other relevant toxicity information, such as the weight-of-evidence (WOE) category for carcinogenic potential. As recommended in the EPA memorandum, *Human Health Toxicity Values in Superfund Risk Assessments* (USEPA 2003b), the primary sources that will be consulted for toxicity values are, in order of priority, EPA's Integrated Risk Information System (IRIS; USEPA 2007a-2010) and EPA's provisional peer reviewed toxicity values (PPRTVs) from the National Center for Environmental Assessment/Superfund Health Risk Technical Support Center. If neither IRIS toxicity values nor PPRTVs are available, then toxicity values will be obtained from other documented sources, such as EPA's Health Effects Assessment Summary Tables (HEAST; USEPA 1997b), the Agency for Toxic Substances and Disease Registry (ATSDR)

minimal risk levels (MRLs; ATSDR 2007), and Oak Ridge National Laboratory's (ORNL) Risk Assessment Information System (ORNL-RAIS; USDOE 2007). Toxicity values appropriate to the relevant exposure routes (e.g., oral, inhalation) and exposure times (e.g., subchronic, chronic) determined for the risk assessment will be collected from these sources.

In addition to these sources, human health toxicity criteria developed by Integral Consulting (Integral) for five organic acid SRCs will be used. The toxicity criteria developed by Integral for diethyl phosphorodithioic acid (DEPT) and dimethyl phosphorodithioic acid (DMPT) were submitted and approved with modification by NDEP in 2007 (Integral 2006; NDEP 2007). The toxicity criteria developed for 4-chlorobenzene sulfonic acid (pCBSA), benzenesulphonic acid (BSA), and phthalic acid were submitted to NDEP in November 2007 (Integral 2007) and approved by NDEP in 2008 (NDEP 2008i,j). The final NDEP-approved values will be used in the risk assessment. Following NDEP guidance (NDEP 2009g), the noncarcinogenic toxicity criterion for dichlorobenzil will be based on the toxicity criterion for 4,4'-dichlorobenzophenone (DCBP), adjusted with additional uncertainty factors to account for the likely greater environmental persistence of dichlorobenzil compared to the surrogate, and for database deficiencies. Additionally, in line with NDEP guidance (NDEP 2006c) pyrene will be used as a toxicological surrogate for noncancer toxicity endpoints for PAHs where no noncancer toxicity criterion are available from EPA or the alternative sources listed above. As recommended by NDEP, the noncarcinogenic toxicity criterion for pyrene will be adopted for the following PAHs: benzo(a)anthracene, benzo(a)pyrene (BaP), benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-c,d)pyrene, acenaphthylene, benzo(g,h,i)perylene, and phenanthrene.

Route-to-route extrapolation assumes that once a chemical is absorbed into the bloodstream, the health effects are similar regardless of whether the route of exposure is oral, dermal, or inhalation. This assumption may be valid for some chemicals with pharmacokinetic characteristics that are similar regardless of the route of administration; however, for many chemicals, factors such as absorption, metabolism, distribution, and elimination vary by exposure route, leading to substantial differences in toxicity. Typically, EPA recommends using route-to-route extrapolation to assess risks from absorbed dose following dermal exposures. These recommendations will be followed here and are discussed in the following sections.

EPA (2009a) explicitly warns against extrapolating oral toxicity criteria to inhalation values because the amount of the chemical that reaches the target site through the inhalation pathway is not a simple function of known parameters (i.e., BW, inhalation rate), but rather a complicated set of factors including the physiochemical characteristics of the inhaled contaminant and human physiologic parameters. Therefore, consistent with EPA (2009a) guidance, route-to-route extrapolations will not be conducted to assess inhalation exposures for most chemicals. The only exceptions to this are cases in which EPA has published inhalation toxicity values that were generated using route-to-route extrapolations. Consistent with EPA (2009a) guidance, these values will be used in the risk assessment without adjustment.

In the case that toxicity criteria are not available for a COPC specific to the exposure route being evaluated a quantitative evaluation of risks associated with exposure to the COPC will not be completed. Uncertainties associated with the exclusion of these COPCs from the quantitative risk evaluation will be discussed in the uncertainty section, as relevant.

The following two subsections describe the toxicity values used to assess noncancer and carcinogenic effects of chemicals including radioactive constituents.

5.1.1 Noncancer Effects from Chemical Exposures

The potential for noncancer health effects from chronic exposures (i.e., greater than 7 years) will be evaluated by comparing the estimated daily intake with a reference dose (RfD) for oral exposure routes, and a reference concentration (RfC) for inhalation exposure routes. Chronic toxicity values represent average daily exposure levels at which no adverse health effects are expected to occur with chronic exposures. Subchronic RfDs/RfCs represent average daily exposure levels at which no adverse health effects are expected to occur with subchronic exposures of less than 7 years, as would be the case for the construction worker and trespasser scenarios to be evaluated for the site. RfDs/RfCs reflect the underlying assumption that systemic toxicity occurs as a result of processes that have a threshold.

The RfDs/RfCs for many noncarcinogenic effects are derived based on laboratory animal studies or epidemiological studies in humans. In such studies, the RfD/RfC is typically calculated by identifying the highest concentration or dose that does not cause observable adverse effects (the no-observed-adverse-effects level [or-NOAEL]) in the study subject. If a NOAEL cannot be identified from the study, a lowest-observed-adverse-effects level (LOAEL) may be used. This dose or concentration is then divided by uncertainty factors to calculate an RfD/RfC.

Uncertainty factors are applied to account for limitations of the underlying data and are intended to ensure that the toxicity value calculated based on the data will be unlikely to result in adverse health effects in exposed human populations. For example, an uncertainty factor of 10 is used to account for interspecies differences (if animal studies were used as the basis for the calculation), and another factor of 10 is used to address the potential that human subpopulations such as children or the elderly may have increased sensitivity to the chemical's adverse effects. Thus, variations in the strength of the underlying data are reflected in the uncertainty factors used to calculate the toxicity values and in the low, medium, or high confidence ratings assigned to those values (USEPA 201097a).

For cases in which toxicity values are not available for the specific time-frame, or exposure route being evaluated, in some instances existing values for other time-frames or routes may be used. For example, EPA states that in cases in which a reference value for a desired duration period (e.g., subchronic) is not available, a reference value based on the next longer duration of

exposure may be used as a conservative estimate that would be protective for the shorter-term ED (USEPA 2009a). This procedure will be adopted for the risk assessments.

RfDs are not available for assessing the dermal exposure route. Oral toxicity values are typically used instead. Because oral toxicity values are usually derived from administered doses, while dermal exposure estimates are expressed as absorbed doses, the oral toxicity values must be adjusted to reflect absorbed dose. This adjustment is accomplished by multiplying the oral RfD by a chemical-specific oral absorption rate. The chemical-specific oral absorption rate is an expression of the fraction of contaminant absorbed in the gastrointestinal (GI) tract in the critical toxicity study. This procedure will be used in the risk assessment. GI absorption values (ABS_{GI}) will be obtained from EPA's RAGS Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) (USEPA 2004a), NDEP guidance (NDEP 2008k) and the ORNL RAIS (ORNL 2007). Following EPA guidance (2004a) toxicity criteria for chemicals with an ABS_{GI} less than 50-percent % will be adjusted.

A summary of toxicity for each COPC will include the chronic and subchronic RfD or RfC, as well as the target organ of toxicity and uncertainty factors used in deriving the RfD/RfC. Uncertainties in the toxicity values will additionally be described.

5.1.2 Carcinogenic Effects from Chemical Exposures

To assess carcinogenic health effects, CSFs are used for oral and dermal exposures, while IURs are used for inhalation exposures. CSFs and IURs are upper-bound estimates of the carcinogenic potency of chemicals. They are used to estimate the incremental risk of developing cancer, corresponding to a lifetime of exposure at the levels described in the exposure assessment. In standard risk assessment procedures, estimates of carcinogenic potency reflect the conservative assumption that no threshold exists for carcinogenic effects (i.e., that any exposure to a carcinogenic chemical will contribute an incremental amount to an individual's overall risk of developing cancer).

Another component of assessing carcinogenic health effects is a qualitative evaluation of the extent to which a chemical is a human carcinogen. For many chemicals listed in IRIS, this evaluation was conducted by EPA using a classification system for WOE determination.¹³ A chemical is assigned a WOE classification based on data obtained from both human and animal studies. Chemicals for which EPA considers adequate human data indicating carcinogenicity are available are categorized as "known human carcinogens" (WOE class A), while other chemicals with various levels of supporting data may be classified as "probable human carcinogens" (WOE class B1 or B2), or "possible human carcinogens" (WOE class C). Where

¹³ The WOE categories described in the final *Guidelines for Carcinogen Risk Assessment* (USEPA 2005) as "standard hazard descriptors" differ from and may eventually supersede those used in IRIS (USEPA 2007a,2010). These descriptors include "carcinogenic to humans," "likely to be carcinogenic to humans," "suggestive evidence of carcinogenic potential," "inadequate information to assess carcinogenic potential," and "not likely to be carcinogenic to humans."

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EPA considers that data are inadequate for determining carcinogenicity, the chemical is "not classifiable as to human carcinogenicity" (WOE class D). When studies provide evidence of noncarcinogenicity, a chemical is assigned a WOE class E (USEPA 2007a/2010).

As described for noncarcinogens, toxicity values measuring carcinogenic potency are not readily available for the dermal exposure route. Following EPA guidance, oral CSFs for chemicals with ABS_{GI} less than 50 percent will be adjusted to determine dermal CSFs. ABS_{GI} will be obtained from EPA's RAGS Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) (2004a), NDEP guidance (2008k), and the ORNL RAIS (ORNL 2007).

A summary of toxicity for each COPC will include the qualitative WOE classification and the CSF or URF. Uncertainties in the incremental risk values will additionally be described.

5.1.3 Effects from Radionuclides

Biological effects associated with exposure to ionizing radiation in the environment may include carcinogenicity, mutagenicity, and teratogenicity. EPA (2001) has determined that cancer risk is the most significant health effect potentially associated with exposure to radionuclides¹⁴. EPA classifies all radionuclides as WOE Class A, based on their property of emitting ionizing radiation and on the WOE provided by epidemiological studies of radiogenic cancers in humans (USEPA 2001, 2009b).

CSFs for radionuclides are available from HEAST for specific ingestion, inhalation, and external exposures (USEPA 2001, 2009b). The CSFs are derived using models that take into account age- and gender-dependence of radionuclide intake, metabolism, dosimetry, radiogenic risk, and competing causes of death. The model averages the risk over the lifetime of the exposed individual. Consequently the slope factors are not expressed as a function of BW and time.

The resultant CSFs represent central estimates of age-averaged, excess lifetime cancer incidence per unit of activity of a given radionuclide inhaled or ingested, for internal exposure, or per unit time-integrated activity concentration in air or soil for external exposure for an average member of the reference population¹⁵. The CSFs may be used to estimate the lifetime cancer incidence risk attributable to a given radionuclide exposure for an average member of the population, but are not appropriate for assessing the risk to a single individual of a particular age or gender. In addition to the age-averaged values, for the soil ingestion pathway, an adult only CSF is available from HEAST.

¹⁴ The only exception to this is uranium, which presents both noncarcinogenic chemical hazard and carcinogenic radiological risks. In line with EPA guidance (USEPA 1996b) in the case that uranium is selected as a COPC for a risk assessment, both types of risk will be evaluated. Noncarcinogenic health effects will be evaluated as for other noncarcinogenic chemicals using toxicity criteria specific to uranium.

¹⁵ Current values were calculated using characteristics, mortality statistics, and baseline cancer rates from the 1980s U.S. population

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All radionuclides undergo a decay process in which the parent radionuclide is transformed in atomic number, mass, or excitation state. In some cases the resulting decay products are radioactive, and may undergo further decay. Each of these decay products may have different physical and chemical properties which affect their environmental fate and transport, as well as different toxic characteristics and potencies. Because each is unique in its action and toxicity, consideration of all of the decay products is a key element in the risk assessment process. The radiation dose estimates used to calculate the radionuclide CSFs explicitly consider the production of radioactive decay products within the body following ingestion or inhalation; however, only intake or external exposure to the single radionuclide is considered. For certain radionuclides with decay products where contributions of dose and risk from radioactive decay products may be significant, EPA has derived CSFs which incorporate the contribution of short-term decay chain products (i.e., less than 1 year half-life) to the total risk. The resultant CSFs are higher than those which consider the parent radionuclide only, because they additionally consider the risk contribution from the short-lived decay products. The calculation of the CSF from these decay chains assumes the presence of SE.

The CSFs from HEAST will be used to evaluate risks to populations with completed exposures at the Site. The adult only CSF for soil ingestion will be utilized for scenarios where exposure occurs within adulthood only (e.g., worker populations). For all other receptor populations and completed exposure pathways, the age-averaged CSFs will be used. Given that the difference between the age and gender-averaged risk coefficients and the adult-only risk coefficients are slight, the use of the age-averaged values are considered appropriate for evaluating risks to these populations. For the radionuclides for which it is available, the CSF which includes the contribution of short-term decay products will be selected. Any significant uncertainties resulting from the use of an age adjusted CSF, or CSFs which incorporate, or do not incorporate decay products will be discussed in the risk assessments.

5.1.4 Effects from Lead

Adverse health effects associated with exposure to lead include, but are not limited to, neurotoxicity, developmental delays, and reproductive impairment (USEPA 2010). No RfD or RfC is available from EPA for lead, and given the current knowledge regarding background body burdens, lead pharmacokinetics, and low exposure levels associated with some health effects, EPA has determined that it is not appropriate to develop reference levels for lead (USEPA 2010). Given the lack of an RfD and RfC, the method for characterizing risk from lead exposure differs from that utilized for most noncarcinogenic agents. This methodology is presented in Section 6.4.

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5.1.45.1.5 Effects from Asbestos

Asbestos risks will be assessed in line with the approaches specified in NDEP's (2009f) *Technical Guidance for the Calculation of Asbestos-Related Risk in Soils for the BMI Complex and Common Areas*. The approach relies on exposure-response coefficients that describe the toxicity of different fiber lengths and types of asbestos. These risk coefficients, are adopted from the draft, *Technical Support Documents for a Protocol to Assess Asbestos Related Risk* (USEPA 2003c), are discussed below.

The majority of available information indicates that lung cancer and mesothelioma are the most important risks associated with low levels of asbestos (NDEP 2009f, USEPA 2003c). Types and aspect ratios (relative length versus diameter) of asbestos fibers differ, and are known to affect the potency of the material; therefore, deriving conclusions regarding the health effects related to asbestos exposure is complex. In the EPA draft document (USEPA 2003c) studies from environments with asbestos dusts of differing characteristics were reviewed to evaluate asbestos related risks. EPA developed an optimal exposure index, which best reconciles the published literature. The index assigns equal potency to fibers longer than 10 μm and thinner than 0.4 μm and assigns no potency to fibers of other dimensions. The optimal exposure index also assigns unique exposure-response coefficients for chrysotile and amphibole fibers for the endpoints of mesothelioma and lung cancer. Optimum dose response coefficients, based on the body of available data will be assumed for this risk assessment. The coefficients are presented in Appendix EF.

5.2 APPROACHES FOR CHEMICAL MIXTURES

For some groups of chemicals, such as polychlorinated dibenzo-p-dioxins (PCDDs)/polychlorinated dibenzofurans (PCDFs)/PCBs and PAHs, information on the toxic potency of individual constituents of the group are expressed in relative terms¹⁶. The approaches for evaluating PCDDs/PCDFs and PAHs are described below.

5.2.1 Toxicity Equivalency Approach for PCDDs/PCDFs/PCBs

Dioxins and furans (PCDDs and PCDFs) are two groups of structurally similar, tricyclic, almost planar, organic compounds that exhibit similar physical and chemical properties. There are 75 dioxins and 135 furans, called congeners, which are differentiated by their number and position of chlorine atoms. Researchers in the early 1980s concluded that a subset of PCDDs, PCDFs, and PCB congeners shared a common mechanism of action and induced comparable biological and toxic responses (USEPA 2003d). However, the potency of the different congeners varies considerably.

¹⁶ Other chemical mixtures have toxicity criteria that represent the potency of the entire mixture (i.e., various Aroclors). These mixtures will be treated in line with the protocols described in Section 5.1.

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Seventeen PCDD and PCDF congeners (7 PCDDs, 10 PCDFs) exhibit what is termed "dioxin-like" toxicity. These 17 congeners have chlorine atoms present in the 2, 3, 7, and 8 positions on the ring structure of the molecule and are more toxic than other congeners with fewer chlorine atoms or with chlorine atoms in different positions on the ring structure. The congener 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) is the most widely studied and has been found to exhibit the most potent toxic response. Similarly, 12 coplanar PCB congeners have been shown to exhibit dioxin-like toxicity and are grouped with the 17 dioxin/furan congeners that exhibit toxicity similar to 2,3,7,8-TCDD (USEPA 2007; Van den Berg et al. 1998).

Human health risk estimates for exposures to PCDDs/PCDFs traditionally require conversion of concentrations of individual dioxin and furan congeners to their 2,3,7,8-TCDD toxic equivalent (TEQ) concentration using congener-specific toxic equivalency factors (TEFs). The 2,3,7,8-TCDD TEQ concentration for each sample is calculated by multiplying concentrations of individual congeners by their congener-specific TEFs, and summing the results for all congeners as shown in Equation 5-1, below. The 2,3,7,8-TCDD TEQ concentration is assumed to express the total potency of the mixture of PCDDs/PCDFs in a sample to exert the toxicity of 2,3,7,8-TCDD.

$$TEQ = \Sigma(C_1 \times TEF_1) + (C_2 \times TEF_2) + \dots(C_n \times TEF_n) \quad \text{Eq. 5-1}$$

where,

C = congener specific concentration (e.g., mg/kg)
TEF = congener specific TEF (unitless).

For assessment of human health risks, TEFs developed by the World Health Organization (WHO) (Van den Berg et al. 1998) and adopted by NDEP for deriving BCLs (NDEP 2009e) will be to calculate TEQs. These TEFs are the most widely accepted equivalency factors and are typically expressed as "WHO98 TEFs". Table 5-1 presents the TEFs that will be used in the risk assessment.

Risk from TEQ concentrations are calculated similarly to that from other COPCs by combining calculated exposure with a risk-based criteria.

5.2.2 Relative Potency Approach for PAHs

The cancer potencies of individual carcinogenic PAH chemicals are expressed relative to the cancer potency of BaP. This procedure involves applying chemical-specific relative potency factors (RPFs) to the CSF for BaP, resulting in a CSF adjusted for the toxicity of each PAH relative to BaP. Table 5-1 presents the RPFs provided by EPA (1993) that will be used in the risk assessment if PAHs are selected as a COPC.

Risks associated with PAHs will be evaluated in a compound specific manner using toxicity criteria based on the RPFs outlined above. In order to retain the ability to more fully understand the contributions of various PAHs to estimates of risk, the individual PAHs for a given sample will not be summed in an *a priori* manner. However, to avoid reducing the effects of multiple PAHs that may act via a similar mode of action, in the case that any single carcinogenic PAH is selected as a COPC, the full suite of carcinogenic PAHs will be evaluated using one half $\frac{1}{2}$ the SQL for non-detects. This could in certain situations lead to risks that are dominated by non-detect values. If this occurs, the uncertainty associated with this approach will be discussed in the risk report.

Despite the wide use of RPFs in health risk assessments at Superfund and Resource Conservation and Recovery Act (RCRA) sites to express the toxicity of carcinogenic PAHs in relation to the toxicity of BaP, numerous limitations of its use have been identified. These limitations contribute to uncertainty in the estimation of risks for the Site. The uncertainties associated with this approach will be discussed in the risk assessment.

6 RISK CHARACTERIZATION

The goal of risk characterization is to present and interpret the key findings of the risk assessment, along with their limitations and uncertainties, for use in risk management decision making. In the process of risk characterization, quantitative estimates of exposure and toxicity are compared to yield estimates of potential health risk. Risks for noncancer and cancer effects are estimated separately because of differences in calculation methods.

With the exception of lead, risks associated with exposure to multiple non-carcinogens will be considered cumulatively. Similarly, risks associated with exposure to multiple chemical carcinogens will be added. The methods for combining risk estimates to non-carcinogens and chemical carcinogens for a given exposure pathway is discussed below. Cancer risks from chemical, radionuclide, and asbestos will be calculated and presented separately.

As presented in Section 3.4.1, if statistical analyses indicate that a particular SRC is within background soil levels, then the SRC will not be identified as a COPC and to be quantified in the HHRA. Background risks for COPCs may be calculated separately and discussed in the uncertainty evaluation to provide context to the HHRA results.

This section describes the methods that will be used for quantifying and interpreting risks and for characterizing uncertainties associated with the risk estimates.

6.1 NONCANCER RISKS FROM CHEMICAL EXPOSURES

Health risks other than cancer are characterized as the increased likelihood that an individual will suffer adverse health effects as a result of chemical exposure. To evaluate noncancer risks, the ratio of the exposure term (i.e., average daily intake or EC) to the corresponding noncarcinogenic toxicity reference value (i.e., RfD or RfC) is calculated. It is most appropriate to apply reference values that correspond with the duration of exposure assumed for a specific receptor (e.g., where ED is less than 7 years, a subchronic RfD or RfC is ideally used). This ratio is referred to as the HQ. If the calculated value of the HQ is less than or equal to 1, no adverse health effects are expected. If the calculated value of the HQ is greater than 1, then further risk evaluation is needed.

The HQ is calculated for oral and dermal exposure pathways using the following equation:

$$HQ(\text{unitless}) = \frac{ADD}{RfD}$$

Eq. 6-1 (adapted from
USEPA 1989)

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where,

ADD¹⁷ = average daily dose of the chemical via the specified exposure route (mg/kg-day)
RfD = reference dose (mg/kg-day).

The HQ is calculated for the inhalation exposure pathway using the following equation:

$$HQ(\text{unitless}) = \frac{EC}{RfC} \quad \text{Eq. 6-2 (adapted from USEPA 2009a)}$$

where,

EC = exposure concentration ($\mu\text{g}/\text{m}^3$)
RfC = reference concentration ($\mu\text{g}/\text{m}^3$).

To evaluate the effect of exposure to multiple chemicals that act on the body in a similar manner, the HQs for each exposure pathway for individual chemicals are typically summed to determine a noncancer hazard index (HI) using the following formula:

$$HI(\text{unitless}) = HQ_1 + HQ_2 + \dots + HQ_i \quad \text{Eq. 6-3 (adapted from USEPA 1989)}$$

where,

HQ = hazard quotient for specified exposure pathway (unitless).

HI for multiple chemicals are generally not summed if the reference doses for the chemicals are based on effects on different target organs. This is because the noncancer health risks associated with chemicals that affect different target organs are not likely to be additive. For this reason, in the case that the total HI exceeds 1 for all COPCs combined, a more refined analysis based on target organ may be conducted.

6.2 CANCER RISKS FROM CHEMICAL EXPOSURES

The cancer risk estimates derived using standard risk assessment methods are characterized as the incremental probability that an individual will develop cancer during his or her lifetime due

¹⁷ For exposure via dermal contact, the ADD is referred to as the dermally absorbed dose (DAD); however, for simplicity, intakes are referred to as the ADD for all exposure routes.

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to exposure to SRCs resulting from the specific exposure scenarios that are going to be evaluated. The term “incremental” reflects the fact that the calculated risk associated with site-related exposure is in addition to the background risk of cancer experienced by all individuals in the course of daily life.

Excess incremental lifetime cancer risks are calculated as the product of the exposure term (i.e., lifetime average daily intake or EC) and the expression of the carcinogenic potency of chemicals (i.e., CSF or IUR).

Excess incremental lifetime cancer risk from oral and dermal exposures is calculated using the following equation:

$$\text{Cancer Risk}(\text{unitless}) = \text{LADD} \times \text{CSF} \quad \text{Eq. 6-4 (adapted from USEPA 1989)}$$

where,

LADD = lifetime average daily dose of the chemical via the specified exposure route (mg/kg-day)
CSF = cancer slope factor (kg-day/mg).

Excess incremental lifetime cancer risk from inhalation exposures is calculated using the following equation:

$$\text{Cancer Risk}(\text{unitless}) = \text{EC} \times \text{IUR} \quad \text{Eq. 6-5 (adapted from USEPA 2009a)}$$

where,

EC = exposure concentration ($\mu\text{g}/\text{m}^3$)
IUR = inhalation unit risk ($\text{m}^3/\mu\text{g}$).

6.3 RADIONUCLIDE RISKS

Cancer risks resulting from intakes of radionuclides are calculated in a similar manner to cancer risks for chemicals. The primary difference in the characterization is that equations used to characterize risks from radionuclides rely on intake parameters, and risk coefficients, expressed in units of activity.

For internal exposure excess cancer risk will be calculated as:

$$\text{Cancer Risk}(\text{unitless}) = \text{Dose} \times \text{CSF} \quad \text{Eq. 6-6 (adapted from USEPA 1996b)}$$

USEPA 1996b)

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where,

Dose = total dose of a radionuclide via the specified exposure route
(pCi)
CSF = cancer slope factor (pCi⁻¹).

For external exposure excess cancer risk will be calculated as:

$$\text{Cancer Risk (unitless)} = EET \times CSF \quad \text{Eq. 6-7 (adapted from USEPA 1996b)}$$

where,

EET = external exposure term for a radionuclide (pCi - year/g)
CSF = cancer slope factor (g/pCi - year).

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6.4 LEAD RISKS

In the case that lead analytical results exceed the NDEP BCL of 800 mg/kg, the ALM will be used to estimate risks associated with lead exposure. The ALM predicts the blood lead level in an adult with a site-related lead exposure by summing the "baseline" blood lead level (PbB₀) (i.e., that which would occur in the absence of any site-related exposures) with the increment in blood lead concentration that is expected as a result of increased exposure due to contact with lead-contaminated soil at the Site (USEPA 2003a). According to EPA (2003a), protection of the fetus is the most health-sensitive endpoint for adults. In-line with assessing this endpoint the ALM includes a module to predict fetal blood lead levels. In the case that the ALM is applied, following EPA guidance (2003a), central estimates of exposure will be used. An arithmetic mean concentration will be used for the EPC in the model. Baseline blood lead concentrations and geometric standard deviations of blood lead for the ALM will be obtained from U.S. population data presented in the National Health and Nutrition Examination Survey (NHANES) III. A target risk level of no more than a five percent probability that a fetus exposed to lead will exceed a blood lead level of 10 µg/dL will be applied as the risk threshold.

6.5 ASBESTOS RISKS

Risks associated with asbestos will be evaluated using NDEP (2009f) assessment methodology. This methodology details procedures to calculate the risk of additional deaths from lung cancer and mesothelioma from inhalation exposures to asbestos and is discussed in detail below.

NDEP guidance adopts the approaches recommended in EPA's draft protocol (USEPA 2003c) for evaluating asbestos-related cancer risk. Under the approach risk is estimated as the product of a risk coefficient and a mathematical function that depends on the level of exposure, the duration of exposure, and time. Estimates of additional deaths attributable to asbestos from lung cancer, from mesothelioma, and from both combined, are based on the optimum risk coefficients, described in Section 5.1.4. Lifetime asbestos induced risk of both lung cancer and mesothelioma differ between males and females, and smokers and non-smokers, and individual risk coefficients have been derived for each of these sub-populations. Risk estimates for each subgroup are combined with population statistics to determine a population averaged risk.

Asbestos-related risk (ARR) will be calculated as:

$$ARR \text{ (unitless)} = EC \times URF \quad \text{Eq. 6-8 (adapted from NDEP 2009f)}$$

where,

EC = exposure concentration (f/cm³)
URF = unit risk factor (cm³/f).

and,

$$URF = \frac{10^{-5}}{0.0001} \times R = \frac{1}{10} \times R \quad \text{Eq. 6-9 (NDEP 2009f)}$$

where,

R = estimated additional deaths from lung cancer or mesothelioma per 100,000 persons from continuous, lifetime exposure to an asbestos concentration of 0.0001 f/cm³ (for fibrous structures longer than 10 μm and thinner than 0.4 μm) as determined using transmission electron microscopy (TEM) methods.

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and,

$$R = 0.5 \times [0.786 \times (NSM + NSF) + 0.214 \times (SM + SF)] \quad \text{Eq. 6-10 (NDEP 2009f)}$$

where,

- NSM = corresponding risk for male non-smokers
- NSF = corresponding risk for female non-smokers
- SM = corresponding risk for male smokers
- SF = corresponding risk for female smokers.

The numerator value (10^{-9}) and denominator value (0.0001) in equation 6-9 allow for an adjustment for the units embedded within the risk coefficients in equation 6-10 which refer to risk per 100,000 persons for exposure to an asbestos air concentration of 0.0001 f/cm³ to be made.

Risks of additional deaths by sub-population to be used for the risk calculations are included in Appendix EF. In line with NDEP guidance, in order to be protective of exposure to second hand smoke, the same R value will be used for child receptors in the offsite residential scenario. The combined risks of lung cancer and mesothelioma will be calculated.

6.6 DATA QUALITY ASSESSMENT

A data quality assessment (DOA) is an analysis performed at the completion of a risk assessment in order to determine if a sufficient amount of data were available to support the risk-based decisions evaluated. A DOA of the sampling data used in the HHRA will be presented in the risk assessment report. The sample size calculations will be conducted for the risk driving COPCs. The formula used for the sample size calculation is based on a non-parametric test (the Wilcoxon signed rank test) and on simulation studies performed by the Pacific Northwest National Laboratories (PNNL 2009) that formed the basis for an approximate formula that is based on the normal distribution. Essentially, the formula is the one that would be used if a normal-base test were being performed, but an adjustment is made (multiply by 1.16) to account for the intent to perform a non-parametric test. The formula is as follows:

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$$n = 1.16 \left[\frac{s^2}{\Delta^2} \right] (z_{1-\alpha} + z_{1-\beta(\mu)})^2 + 0.5z_{1-\alpha}^2 \quad \text{Eq. (equation 6-11)}$$

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(PNNL 2009)

where,

- n = number of samples
- s = estimated standard deviation of concentrations/fibers/activities
- Δ = the difference between the threshold value stated in the null hypothesis and the point at which β is specified.
- α = significance level or Type I error tolerance
- $\beta(\mu)$ = Type II error tolerance
- z = a quantile from the standard normal distribution.

For the selected risk drivers, inputs for the calculations will include an estimate of the variance form the measured data, a desired significance level, and desired power of the test that must be specified at a concentration of interest (which determines the tolerable difference from the threshold value), typically the NDEP BCL. The calculations will cover a range of Type I and Type II error tolerances, and the point at which the Type II error is specified. Accordingly, various combinations of input values will be used, including: values of α of 5%, 10% and 15%; values of β of 15%, 20% and 25%; and, a gray region of width 10%, 20% and 30% of the threshold level.

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The results of the DQA will be used to support the uncertainty evaluation, described below.

6.66.7 _____ UNCERTAINTY EVALUATION

The final element of the risk assessments will be an assessment of the uncertainty in the estimated noncarcinogenic and carcinogenic risks. Uncertainty is inherent in many aspects of the risk assessment process, and generally arises from a lack of knowledge of 1) site conditions and future site use, 2) toxicity and dose-response for COPCs, 3) the extent to which an individual may be exposed to COPCs, and/or 4) the representativeness of modeled EPCs. This lack of knowledge means that assumptions must be made based on information presented in the scientific literature or professional judgment. In general, such assumptions will be made in a manner that intentionally biases the process towards health protection.

Uncertainties in the risk will be identified and addressed qualitatively in general, although some quantitative measures of uncertainty (e.g., probabilistic analyses using Monte Carlo analysis) may be provided. Descriptions of the uncertainty inherent in analytical data and toxicity and exposure parameters used to characterize risks will be provided in the risk assessment reports. The uncertainty analyses will conclude with a discussion of the overall impact of uncertainty in the risk assessment on the risk characterization for the site assessment area.

6.76.8 _____ PRESENTATION AND INTERPRETATION OF FINDINGS

The risk assessments results will be presented in tabular format and include key supporting information used to calculate the risks. Key pathways and COPCs that drive risk estimates will be identified and discussed. Reports will include discussions of the results in the context of their implications for risk management actions at the site assessment area. Key uncertainties or

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data gaps and their influence on risk management decisions also will be discussed. Risk assessment reports will include the following:

- Background - description of the site assessment or waste management area being addressed including relevant history; relevant geographical information.
- Exposure scenarios - description of receptor groups and pathways for which risks will be characterized.
- Data evaluation - description of data sources selected for use in the risk assessment; details of data treatment.
- COPC selection - description of methodology for selecting COPCs; list of COPCs that will be evaluated.
- Exposure assessment - presentation of exposure parameters and media-specific EPCs; presentation of methodology for calculating exposures; resulting exposures.
- Toxicity assessment - presentation of noncarcinogenic and carcinogenic toxicity criteria; discussion of human health effects associated with risk-driving COPCs.
- Risk characterization - presentation of methodologies for characterizing risks; calculated noncarcinogenic and carcinogenic risks.
- Uncertainties - qualitative and quantitative assessments of key uncertainties and data gaps; a description of the impacts of uncertainties on resulting risk estimates.
- Conclusions.
- References.

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