6.0 RISK ASSESSMENT METHODOLOGY

This Risk Assessment (RA) was undertaken in order to evaluate potential impacts to both human health and the environment from the existing chemical contamination at two SWMUs within TEAD-S. The RA was conducted in accordance with *USEPA Risk Assessment Guidance for Superfund* (USEPA 1989b and USEPA 1989c) and in compliance with the State of Utah Corrective Action Cleanup Standards Policy (R315-101). The methodology is divided into two sections: Baseline Human Health Risk Assessment (Section 6.1) and Ecological Assessment (Section 6.2). These sections describe the general methodology used to evaluate the potential risk to human health and the environment from exposure to site-related chemicals within the nine sites, in groundwater at SWMU 13, and within the two sites at SWMU 17. The results of the RA are presented separately for each site in Sections 7.0 and 8.0 of this report. These results will be used as a decision-making tool for the RFI process, leading to the selection and subsequent implementation of corrective measures for each release site. Recommendations for corrective measures will be based on the closure equivalency requirements of Section R315-101-6 of the Utah Hazardous Waste Rules. The last paragraph of Section 1.2 describes the risked-based criteria used to develop all recommendations.

The two SWMUs and their respective sites and areas of potential concern investigated as part of this risk assessment are as follows:

- SWMU 13
  - Fuel Spill Site
  - Underground Storage Tank Site
  - 3X Yard
  - Boiler Blowdown Discharge Site
  - Drainage Ditch Site
  - Chemical Unload Site
  - Pavement Perimeter Site
  - Sodium Hydroxide Spill Site
  - Wastewater Lagoons Site
  - Groundwater

- SWMU 17
  - Fuel Spill Site
  - Drum Storage Site

6.1 BASELINE HUMAN HEALTH RISK ASSESSMENT

The purpose of the Baseline Human Health Risk Assessment is to estimate potential cancer risks and potential chemical hazards to receptor populations under current and possible future land use conditions at both SWMUs. There are five major steps in the human health risk assessment process. The initial step of the assessment, which is identification of chemicals of potential concern (COPCs), involves the evaluation of the analytical data obtained from field

investigations at the 11 sites within SWMUs 13 and 17 at TEAD-S. Evaluations include the examination of quality assurance/quality control (QA/QC) parameters, the relative levels of potential health risk posed by detected compounds, and a comparison against background values (for metals and anions). After COPCs are identified, an exposure assessment is performed to examine the type, timing, and magnitude of exposures to the COPCs at each site. The primary tasks in this assessment include identification and characterization of potential receptor populations, identification of potentially complete chemical exposure pathways, and quantitative estimates of potential chemical intakes. Based on USEPA guidance, conservative and health-protective exposure parameters were selected for evaluating potential chemical exposure. The toxicity assessment section then documents the known toxicological properties of the chemicals detected within each site and lists the USEPA health criteria for each substance. Toxicity information was obtained primarily from the USEPA's Integrated Risk Information System (IRIS) (USEPA 1994) and the Health Effects Assessment Summary Tables (HEAST) (USEPA 1992a). A risk characterization was conducted that integrates the chemical toxicity information and the exposure assessment results to produce quantitative estimates of potential health risks and hazards at each site. As a final step, an uncertainty analysis was performed to provide qualitative evaluations of the sources of uncertainty associated with the risk estimates. The results from this human health risk assessment will assist in determining what, if any, corrective action is necessary at any of the sites in SWMUs 13 and 17.

This methodology section follows the format of the five key components of the risk assessment process. Section 6.1.1 describes the screening process used to identify the COPCs at each release site. Potential receptor populations and complete exposure pathways are identified in Sections 6.1.2.1 and 6.1.2.2, respectively. The methodologies used to estimate exposure point concentrations (EPCs) and chemical intakes are described in Sections 6.1.2.3 and 6.1.2.4, respectively. Section 6.1.3 describes the toxicity assessment, followed by the risk characterization in Section 6.1.4. Finally, the potential sources of uncertainty in the risk assessment process are discussed in Section 6.1.5. The COPCs, potential exposure pathways, and risk estimates derived from the use of these methodologies are discussed individually for each site in Sections 7.0 and 8.0 of this report.

6.1.1 Methodology for Identifying Chemicals of Potential Concern

The results of previous and Phase II RFI field investigations at each release site were evaluated to identify the COPCs. The sampling techniques used during the Phase II RFI field investigations are discussed in Section 4.0 of this report. The results of these investigations are discussed individually for each SWMU in Sections 7.0 and 8.0. The process used to identify the COPCs included an evaluation of the analytical methods, sample quantitation and detection limits, coded data, field and laboratory QA/QC, and a screening process that compares the concentration of inorganic compounds measured in samples from each release site against established background values.

6.1.1.1 *Analytical Methods*

All samples collected during the Phase II RFI were analyzed by Arthur D. Little of Cambridge, Massachusetts, or DataChem Laboratories of Salt Lake City, Utah, using USAEC or USEPA analytical methodologies, and following the USATHAMA Quality Assurance Program (USATHAMA 1990).

Soil-, groundwater-, and surface-water-sample analyses performed for this RFI included VOCs, SVOCs, metals, nitroaromatic compounds (explosives), agent breakdown products, TPHC, pH, anions, alkalinity, total organic carbon (TOC), and radioactivity (gross alpha, gross beta, and total uranium). The particular suite of analyses to which samples from each site were subjected was based on historical information and the results of previous field investigations. The analytical method(s) selected for each suite was selected on the basis of providing data that meet regulatory requirements for limits of detection and reporting. This allows comparison of Phase II RFI data with regulatory standards.

6.1.1.2 *Chemical Data Screening Against QC Sample Results*

Prior to applying the screening process for removing common laboratory contaminants from a data set, the sample results were subjected to QA/QC checks against protocol specified by the USAEC QAP (USATHAMA 1990). This program focuses on ensuring precision, accuracy, representativeness, comparability, and completeness in the reported data. The methodology for determining data quality is described in Section 4.0 of this report. A screening methodology recommended by the USEPA for removing common laboratory contaminants from a data set was then utilized. Acetone, 2-butanone (methyl ethyl ketone), methylene chloride, toluene, and the phthalate esters are considered by the USEPA to be common laboratory contaminants. The methodology assumes that for these chemicals, if a reported concentration is less than 10 times the maximum amount detected in any QC blank sample, that site data should be considered laboratory contamination and not carried further through the human health risk assessment. Chemicals not generally considered common laboratory contaminants were assumed to be artifacts if the reported concentration in a site sample was less than five times the maximum amount detected in any blank. The data for all the sites can be found in Appendix D of this report.

6.1.1.3 *Chemical Screening Against Background*

The selection process for the COPCs included a screening process that compares the concentration of inorganic chemicals detected in field samples from each site against established background concentrations (for metals and anions). The approach used to estimate background concentrations for these chemicals is described in Section 5.0 of this report. Inorganics that were detected in site soil and groundwater samples at levels below the corresponding background values for these media were considered to be present at the site at natural concentrations and, thus, not considered COPCs in the human health risk assessment.

6.1.2 Exposure Assessment

An exposure assessment was conducted to estimate the type, timing, and magnitude of exposures that receptors may experience because of contact with the COPCs. The primary goal of this task was to quantify complete exposure pathways for the 11 sites within SWMUs 13 and 17 under current and potential future land use scenarios. This was achieved by identifying potentially exposed receptor populations, identifying potential chemical exposure pathways, and quantitatively evaluating the levels of chemical exposure associated with each potential exposure pathway. The qualitative portions of this task are described in Sections 6.1.2.1 and 6.1.2.2, while the quantitative exposure assessment is presented in Sections 6.1.2.3 and 6.1.2.4.

6.1.2.1 *Identification of Potentially Exposed Receptor Populations*

The following section describes the receptor populations that may potentially become exposed to site-related chemicals under both current and potential future land use scenarios.

6.1.2.1.1 ***Current Land Use***. Adult populations potentially exposed to the on-site contamination at the 11 sites under current land use consist of the on-site workers. Public access to the CAMDS (SWMU 13) and the DF/MC (SWMU 17) is controlled, thereby precluding public exposure. Armed guards patrol the area on foot within the double fence surrounding the CAMDS facility and by vehicle outside the fences, and are stationed at the TEAD-S entrance.

Off-site residents (i.e., adults and children) were regarded as a potentially exposed receptor population. The area surrounding TEAD-S is predominantly undeveloped. It is a sparsely settled, rural area that consists of open space and cultivated land that is used primarily for grazing and agricultural purposes. A few small communities exist within a 5-to-10-mile radius of TEAD-S: Clover and St. John are approximately 2 miles northwest; Stockton is 10 miles to the north; Ophir is approximately 4 miles northeast; and Faust is about 5 miles south. All of these towns, except Stockton, have populations less than 40. Stockton's 1990 population was 426. The nearest residence to the CAMDS facility is approximately 5 miles to the northwest, on a road off Highway 199 near the communities of Clover and St. John. The nearest residence to SWMU 17 is approximately 1 mile east near the community of West Mercur.

6.1.2.1.2 ***Future Land Use***. At all release sites, a future construction worker scenario was added to evaluate the impact from exposure to site-related chemicals in subsurface soil during potential future excavations within the site. In some instances, a construction worker could be exposed to subsurface soils that contain elevated levels of site-related chemicals. The estimated risk to this receptor will be included at those sites where contamination was identified in subsurface samples (below 1 foot bgs).

On-site land use at SWMUs 13 and 17 could change in the future although it is unlikely. Development of both SWMUs 13 and 17, and the rest of TEAD-S as a residential area or other public use area is considered highly unlikely given the low-population density in the area and the unavailability of adjacent, non-Army land for public development. Furthermore, the Army has no current plans to "excess" any land of the TEAD-S and, considering the mission of TEAD-S, it is unlikely that the installation would close. If, in the future, the Army elects to release TEAD-S property for unrestricted use, a Base Realignment and Closure (BRAC) site investigation and risk assessment would be required before any such release. However, for the baseline human health risk assessment conducted for each site, future residential land use was chosen as an additional conservative exposure scenario. Although a residential future land use scenario was selected, decisions for near-term remedial action alternatives should be made primarily on the basis of current land use scenarios.

6.1.2.2 *Identification of Potential Exposure Pathways*

Potential human-exposure pathways were identified for current and potential future land use scenarios. An exposure pathway describes the course that a chemical takes from a source to an exposed individual. In order for an individual to be exposed to a chemical, the following four factors contributing to a complete exposure pathway must be identified: (1) a source of COPCs, (2) an impacted medium such as soil, (3) an exposure or contact point with the impacted medium such as soil contact while working, and (4) an exposure route for chemical intake by a receptor, such as incidental soil ingestion. The occurrence of these four factors leading to potentially complete exposure pathways is examined in Sections 6.1.2.2.1 and 6.1.2.2.2.

A conceptual site model was developed for each site to illustrate the potential exposure pathways under current and future land use conditions. The conceptual site model is included individually for each site in Sections 7.0 and 8.0 of this report. In the conceptual site model diagram, receptors potentially exposed in each complete pathway are indicated by shaded blocks. Two types of shading are utilized: slanted-lines indicate complete pathways due to site-specific contamination; stippling designates complete pathways due to chemicals originating over the entire SWMU or both SWMUs. Blocks with no shading indicate incomplete pathways (i.e., having one or more of their components missing). It should be noted that features such as access restrictions vary between release sites and, thus, the exposure pathways that appear complete on the conceptual site models may differ between release sites. Also, it is important to note that each conceptual site model reflects complete exposure pathways based on the presence and availability of a source of site-specific, SWMU-specific, or depot-specific chemicals. Sections 7.0 and 8.0 include a description of each site's justifications leading to complete and incomplete exposure pathways.

6.1.2.2.1 ***Current Land Use Exposure Pathways.*** In general, the following describes the exposure pathways that are complete within each environmental medium for the 11 sites. Further discussion of site-specific features leading to complete and incomplete exposure pathways are provided in Sections 7.0 and 8.0 of this report.

Soil/Sediment

Under the current land use scenario, exposure to site-specific chemicals in soil or sediment can occur through dermal contact and incidental ingestion. The soil dermal contact and ingestion pathways apply to on-site worker exposure at all sites.

Groundwater

Exposure to SWMU-wide chemicals in groundwater can potentially occur through the ingestion, dermal contact, and inhalation pathways. Use of groundwater as a potable water supply for domestic purposes can entail the direct consumption of chemicals in drinking water, as well as dermal contact with chemicals and inhalation of VOCs during showering or bathing. The current source of potable water at the TEAD-S consists of two water supply wells located several miles northeast of SWMUs 13 and 17. These wells are located upgradient to the groundwater flow direction and, thus, are not subject to site-related chemicals. The groundwater exposure pathway was therefore considered incomplete for the on-site worker. Potential exposure to the off-site resident was evaluated with the use of modeling of current groundwater monitoring sample results (representing all of the monitoring wells at SWMU 13).

Surface Water

Surface water was encountered at the Wastewater Lagoons, Boiler Blowdown Discharge Site, and the Drainage Ditch Site. The lagoons are fenced, and access to them is controlled. Therefore, the only pathway evaluated for this medium at this location is potential exposure of on-site workers and off-site residents to inhalation of VOC emissions dispersed from surface water. The surface water at the other two site locations was also assumed to be inaccessible to on-site workers and off-site residents and, thus, exposure due to dermal contact and ingestion of site-related chemicals in the surface water was not evaluated. However, inhalation of VOC emissions from surface water at the Boiler Blowdown Discharge Site by on-site workers and off-site residents was evaluated.

Air

Under the current land use scenario, the inhalation pathway was assumed complete for the on-site worker and the off-site resident. For the worker, this pathway was assumed complete at all 11 sites regardless of whether a site-specific source of chemicals was present. The current on-site worker was assumed to be exposed to SWMU-wide chemicals in air, while only site-specific surface soil data was used to model the incremental air pathway impacts to off-site residents due to each individual site (i.e., a site-specific evaluation). The conceptual site

models for the Fuel Spill and UST Sites indicate this pathway is incomplete because there is no source of site-specific chemicals in the surficial soil. However, exposure from this pathway was evaluated since any on-site receptor at these locations can inhale airborne chemicals from other site locations within SWMU 13. Volatilization of VOCs from soil and fugitive dust emissions was the release mechanism considered for organic and inorganic site-related chemicals, respectively.

6.1.2.2.2 *Future Land Use Exposure Pathways.* In general, this section describes the exposure pathways that could be complete within each environmental medium for future on-site residents. Site-specific features leading to complete and incomplete exposure pathways are provided in Sections 7.0 and 8.0 of this report.

Soil/Sediment

Under a future residential scenario, exposure to site-related chemicals in soil or sediments can take place through dermal contact and incidental ingestion. Both dermal exposure and ingestion pathways with respect to site-specific contamination are considered to be complete for potential future on-site residents and/or construction workers at all sites.

Groundwater

Exposure to SWMU-wide chemicals in groundwater at SWMU 13 by potential future on-site residents and construction workers within the 11 sites can potentially occur via ingestion, dermal absorption, and/or vapor inhalation. These receptors may ingest drinking water derived from future on-site wells, contact chemicals in groundwater, and inhale VOCs during showering or bathing. However, this pathway was only evaluated for potential future on-site residents at the nine sites within SWMU 13. Evaluation of the exposure by the construction worker is not necessary since the exposure by the on-site resident represents a more conservative assessment.

Exposure to SWMU-wide chemicals was the approach selected for the groundwater pathway. Future on-site residents were assumed to be potentially exposed to groundwater contaminants measured in monitoring wells located across SWMU 13.

Surface Water

Under future use conditions, water releases into former ponds and lagoons used for industrial wastewater discharge are not expected to occur. As a result, future surface water is not expected to exist and, thus, exposure to surface water was not evaluated. However, inhalation of fugitive dust emissions due to wind erosion, incidental ingestion, and dermal contact with the remaining contaminated sediments were evaluated.

Air

For the future-use scenario, the inhalation pathway was assumed to be potentially complete for the potential future on-site resident and/or construction worker at all sites. Volatilization of VOCs from soil and fugitive dust emissions due to wind erosion was the release mechanism considered for organic and inorganic site-related chemicals in soil, respectively.

The future on-site residents were assumed to be exposed to SWMU-wide chemicals in air, while only site-specific soil data were used to model air pathway impacts to the construction worker (i.e., a site-specific evaluation).

Homegrown Vegetables, Beef, and Milk

Exposure to site-related chemicals in soil can potentially occur via consumption of homegrown vegetables, beef, and milk by potential future on-site residents and, thus, these pathways were considered complete for all sites. At the Fuel Spill Site, the UST Site, and the Sodium Hydroxide Spill Site, exposure from vegetable consumption was not evaluated since site-specific chemicals were measured in subsurface soil only and are not available for plant uptake. However, exposure from consumption of beef and milk was evaluated quantitatively since it was based on exposure point concentrations derived as depot-wide average values over TEAD-S. Therefore, future residents are assumed to be exposed to depot-wide chemicals in beef and milk at all locations despite the fact that site-specific chemicals were not measured in surface soil at some of these sites.

6.1.2.2.3 ***Summary of Exposure Pathways.*** Human-exposure pathways considered to be complete for this human health risk assessment are as follows:

- Dermal contact with and incidental ingestion of site-specific soils or sediment by current on-site workers, potential future on-site residents, and construction workers
- Inhalation of volatiles and fugitive dust from soils by current on-site workers, off-site residents, potential future on-site residents, and construction workers
- Dermal contact with and ingestion of SWMU-wide groundwater (migrating downgradient of SWMU 13) by off-site residents from off-site domestic wells
- Inhalation of volatiles while showering or bathing by off-site and potential future on-site residents
- Dermal contact with and ingestion of SWMU-wide groundwater from on-site wells by potential future on-site residents at SWMU 13
- Ingestion of homegrown vegetables, beef, and milk by potential future on-site residents
- Inhalation of VOC emissions from surface water by on-site workers and off-site residents

6.1.2.3 *Derivation of Exposure Point Concentrations*

Estimation of chemical intake rates for each complete exposure pathway are discussed in greater detail in Section 6.1.2.4. For this baseline human health risk assessment, chemical intakes were estimated using a combination of average and high-end values for the various intake variables or exposure factors. Combining average and high-end values within an exposure pathway resulted in an estimate of the reasonable maximum exposure (RME) for that pathway.

The analytical results of the field investigations conducted in 1991 and 1993 were used to estimate the concentration term in each intake equation. Because of the uncertainty associated with any estimate of the exposure concentration, the 95 percent upper confidence limit (UCL) on the arithmetic average concentration (upper bound value) for each environmental medium was used for this value in the intake equation for each complete pathway. In data sets where some sample results were reported below the CRL (non-detects), these were included in the calculation of the arithmetic mean as one-half the CRL value. In cases where a CRL was not available, one-half the method or instrument detection limit was used. In cases where this upper bound value exceeded the maximum measured concentration, the maximum measured concentration was utilized as the estimated exposure concentration. In instances where one-half of the detection limit for non-detects was higher than the maximum reported concentration, this non-detect value was deleted from the exposure point concentration calculation because of the high uncertainty associated with this value.

The following sections describe the general approach to estimating exposure point concentrations for each exposure medium under both land use scenarios. The actual estimated exposure point concentrations in surface soil, subsurface soil, surface water, groundwater, and air at each site are presented in Sections 7.0 and 8.0 of this report. The general approaches for deriving these values in groundwater and air are described in Sections 6.1.2.3.2 and 6.1.2.3.3, respectively. The measured concentrations of site-specific chemicals were assumed to remain constant for the risk evaluation of the potential future land use scenario (i.e, that no degradation or attenuation of chemicals would occur—a conservative assumption).

6.1.2.3.1 ***Chemical Fate and Transport.*** Organic and inorganic chemicals may migrate from the identified sources to other environmental media. Generally, these compounds vary in their capacity to migrate between environmental media according to their physicochemical properties. Several of these properties—water solubility, vapor pressure, Henry's Law constant, octanol/water partition coefficient (K_{ow}), organic carbon partition coefficient (K_{oc}), and bioconcentration factor—indicate the potential mobility of organic chemicals. The physicochemical properties of these analytes detected within the 11 release sites are summarized in Table 6-1. The mobility of organic chemicals in the environment that have small water solubilities (< 10 mg/L), small vapor pressures ($< 1 \times 10^{-6}$ mm Hg), large K_{ow} values ($> 1{,}000$), and large K_{oc} values ($> 1{,}000$) may be limited since they exhibit a tendency to adsorb strongly to soil and be resistant to leaching. These chemicals typically do not hydrolyze, photolyze, or volatilize; they also tend to biodegrade slowly and bioaccumulate.

Table 6-1. Summary of Physicochemical Properties of Organic Chemicals of Potential Concern

Contaminant	CAS No.	Water Solubility (mg/L)[a]	Vapor Pressure (mm Hg)[a]	Henry's Law Coeff. (atm-m^3/mole)[a]	Log K_{ow} (unitless)	K_{oc} (unitless)	Bioconc. Factor (BCF - fish) (unitless)
Explosives							
1,3-Dinitrobenzene[b]	99-65-0	469	8.15E-04	2.75E-07	1.49	2.18	8.51
2,4-Dinitrotoluene	121-14-2	300	1.4E-04	8.79E-08	1.98	282	19
2,6-Dinitrotoluene	606-20-2	ND	3.5E-04[c]	8.79E-08[d]	172	204	5,225 for algal biomass. Estimate based on Log K_{ow} of 12[c].
HMX[e]	2691-41-0	2.6	9.0E-16	ND	0.13	130	0.5
RDX	121-82-4	Insoluble	4.1E-09	2.6E-11	0.87	42-167	24.8
Nitrobenzene	98-95-3	1,780	1 (@ 44.4°C)	2.2E-05	1.85	1.27-370	6
Tetryl[b]	479-45-8	0.02 wt%	< 1	1.89E-03	2.04	2.37	54
1,3,5-Trinitrobenzene	99-35-4	340	3.2E-06	3.08E-9	1.10	104-178	5-23
2,4,6-Trinitrotoluene	118-96-7	130	2.0E-04	4.57E-7	1.60	35-1,900	210-2,030
Agent Breakdown Products							
Isopropyl Methyl Phosphonic Acid (IMPA)	1832-54-8	Soluble[f]	Relatively Nonvolatile[f]	ND	ND	ND	ND
Fluoroacetic Acid[b]	62-74-8	ND	ND	ND	ND	ND	ND

Table 6-1. Summary of Physicochemical Properties of Organic Chemicals of Potential Concern (continued)

Contaminant	CAS No.	Water Solubility (mg/L)[a]	Vapor Pressure (mm Hg)[a]	Henry's Law Coeff. (atm-m^3/mole)[a]	Log K_{ow} (unitless)	K_{oc} (unitless)	Bioconc. Factor (BCF - fish) (unitless)
Semi-Volatile Organic Compounds							
Acenaphthylene	208-96-8	3.93	9.12E-04	1.13E-05	4.07	950-3,315	129-575
Acenaphthene	83-32-9	Insoluble	10 (@ 131°C)	1.55E-04	3.92	2,065-3,230	387
Anthracene	120-12-7	1.29	1.95E-04	2.7E-03	4.45	26,000	1,029
Bromacil[b]	314-40-9	815	8.0E-04	ND	ND	ND	ND
Bis(2-ethylhexyl)phthalate	117-81-7	0.285	1.32 (@ 200°C)	0.612[g]	5.17	10,000-100,000	100-10,000
2-Chloronaphthalene[b]	91-58-7	6.74	1.7E-02	6.12E-04	4.07	3.93	2,050
Dimethylnaphthalenes[h]	28804-88-8	25.8	ND	2.6E-04	3.87	8,500	30-160
Fluorene	86-73-7	Insoluble	10 (@ 146°C)	2.1E-04	4.38	3.7	ND
Methylnaphthalenes	1321-94-4	25.8	ND	2.6E-04	3.87	8,500	30-160
2-Methylnaphthalenes	91-57-6	24.6	0.068	5.2E-04	3.86	8,500	23,500
4-Methylphenol	106-44-5	25,000 (@ 50°C)	1 (@ 53°C)	9.6E-07	1.94	5-50	18
N-nitrosodiphenylamine	86-30-6	40	0.1	6.4E-04	3.13	1,200	217
Naphthalene	91-20-3	30	0.075	ND	3.01	420-4,100	40-1,000
3-Nitrotoluene[b]	99-08-1	0.498	1.5E-01	5.41E-05	2.45	2.16	16
Palmitic Acid	57-10-3	Insoluble	ND	ND	ND	ND	ND
Phenanthrene	85-01-8	1.29	6.8E-04	1.24E-04	4.57	22,900	ND

Table 6-1. Summary of Physicochemical Properties of Organic Chemicals of Potential Concern (continued)

Contaminant	CAS No.	Water Solubility (mg/L)[a]	Vapor Pressure (mm Hg)[a]	Henry's Law Coeff. (atm-m^3/mole)[a]	Log K_{ow} (unitless)	K_{oc} (unitless)	Bioconc. Factor (BCF - fish) (unitless)
TPH	NA	ND	ND	ND	ND	ND	ND
Trimethylnaphthalenes[b]	NA	25.8	ND	2.6E-04	3.87	8,500	30-160
Volatile Organic Compounds							
Acetone	67-64-1	insoluble	266	4.28E-05	0.24	0.37	0.69
Benzene	71-43-2	180,000	95.2	5.3E-03	2.13	98	24
Bromodichloromethane[b]	75-27-4	4,700	50	2.12E-04	2.10	1.79	5.2-23.4
Chloroform	67-66-3	9,300	160	7.27E-03	1.95	44	6
Chloromethane[b]	74-87-3	7,400	3,789	8.82E-02	0.91	1.40	Negligible
Dibromochloromethane[b]	124-48-1	0.004	76	8.7E-04	2.08	1.92	5
1,2-Dichlorobenzene	95-50-1	145-149	1.47	0.0012	3.38	280	270-560
1,4-Dichlorobenzene[b]	106-46-7	49	0.7	1.5E-03	3.62	2.20	Negligible
1,1-Dichloroethylene[b]	75-35-4	6,400	591	1.5E-02	1.48	1.81	ND
1,2-Dimethylbenzene/ O-Xylene	95-47-6	Insoluble	760 (@ 144.4°C)	5.35E-03	3.12	2.14	132
1,3-Dimethylbenzene/ M-Xylene	108-38-3	Insoluble	760 (@ -47.5°C)	3.14E-01	3.20	ND	2.2
Ethylbenzene	100-41-4	1.4	10	8.44E-03	3.15	871	1.9
Methyl-n-butyl ketone/ 2-hexanone	591-78-6	174,000	12	3.39E-05	1.38	134	6
Methylene chloride	75-09-2	ND	400	ND	1.25	48	5

Table 6-1. Summary of Physicochemical Properties of Organic Chemicals of Potential Concern (continued)

Contaminant	CAS No.	Water Solubility (mg/L)[a]	Vapor Pressure (mm Hg)[a]	Henry's Law Coeff. (atm-m^3/mole)[a]	Log K_{ow} (unitless)	K_{oc} (unitless)	Bioconc. Factor (BCF - fish) (unitless)
Methylisobutyl ketone	108-10-1	20,400	15.7	9.4E-05	1.19	19-106	2-5
Monochlorobenzene[c]	108-90-7	448	11.8	3.56E-03	2.18	ND	10-100
1,1,2,2-Tetrachloroethane	79-34-5	2,962	6.1	4.55E-04	2.39	79	8-10
Toluene	108-88-3	535[c]	28.4[c]	330[c]	2.73	37-178	90
1,1,1-Trichloroethane	71-55-6	4,400	127	8.0E-03	2.49	81-89	9
1,1,2-Trichloroethane[b]	79-00-5	4,500	19	0.09E-04	2.18	1.75	< 10
Trichloroethylene[b]	79-01-6	1,100	57.8	9.9E-03	3.30	2.15	17-39
Xylene	1330-20-7	Insoluble	ND	3.2E-01	3.20	48-68	2.14-2.20

Notes.—All values were compiled from HSDB (1993) unless otherwise noted. [The National Library of Medicine, Toxicology Data Network, Hazardous Substances Data Bank Database, accessed March, 1994.] ND denotes a lack of data.

[a]Values for water solubility, vapor pressure, and Henry's law constants were measured at 20° to 25°C.

[b]Values compiled from Montgomery, John H., 1991. Groundwater Chemicals Field Guide: Lewis Publishers, Chelsea, Michigan.

[c]Values compiled from Howard, 1990.

[d]Value for 2,4-Dinitrotoluene used in lieu of data for 2,6-Dinitrotoluene.

[e]Values from Layton et al., 1987.

[f]Quantitiative data not available. Qualitative information from USEPA, 1992b.

[g]Values compiled from Montgomery, Weston 1991, and Rust E&I 1993.

[h]Value for methylnaphthalene used in lieu of dimethylnaphthalene and trimethylnaphthalene specific data.

Table 6-2. Fate and Transport for the Organic Chemicals of Potential Concern

Contaminant	Soil	Water	Air	Biological Systems
Explosives				
1,3-Dinitrobenzene	Adsorption to clay expected but only weakly to other soils; leaching may occur. Volatilization from surficial soil may occur.	Biodegradation and slow volatilization may occur.	Reacts photochemically with hydroxyl radicals (half life of 14.15 hours). Direct photolysis may occur.	Bioconcentration and hydrolysis not expected.
2,4-Dinitrotoluene (Howard 1990)	Slightly mobile in soil. Not susceptible to hydrolysis and photolysis should not be significant.	Slight tendency to adsorb to soil/sediment and biota. Photolysis (surface water) is probably the most significant removal process.	Not Volatile.	May biodegrade in both aerobic and anaerobic zones of soil. Not expected to bioconcentrate in aquatic systems.
2,6-Dinitrotoluene (Howard 1990)	Slightly mobile in soil. Not susceptible to hydrolysis and photolysis should not be significant.	Slight tendency to adsorb to soil/sediment and biota. Photolysis (surface water) is probably the most significant removal process.	Not Volatile.	Not expected to bioconcentrate in aquatic systems.
HMX (Rust E&I 1993)	NA	Photolysis is the dominant transformation process in surface water.	NA	May biodegrade under aerobic and anaerobic conditions.
RDX (Rust E&I 1993)	Moderate to high mobility in soils. Not expected to hydrolyze.	Mobile in water. Not expected to hydrolyze.	Direct photochemical degradation occurs if RDX is released to surface water (half-life of 1.5 hrs.). Direct photodegradation will also occur.	Biodegradation is not an important fate process, but anaerobic degradation in soil can occur. Bioaccumulation in aquatic organisms should not be significant.

Table 6-2. Fate and Transport for the Organic Chemicals of Potential Concern (continued)

Contaminant	Soil	Water	Air	Biological Systems
Nitrobenzene (Howard 1990)	Mobile in soil and subject to leaching.	Mobile in water.	Not volatile. Photolyses in the atmosphere.	Some studies indicate the biodegradation is an important removal process. Not expected to bioconcentrate.
Tetryl	Susceptible to slow hydrolysis in acidic and neutral soils; however, in highly alkaline soils, hydrolysis may be rapid. Moderate leaching expected.	May degrade by hydrolysis and photolysis (half life of 33 to 305 days).	Exist primarily in the particulate phase.	Potential for bioconcentration negligible.
1,3,5-Trinitrobenzene (Rust E&I 1993)	Expected to be mobile in soil and subject to leaching.	May photolyze.	May exist partly in vapor phase and partly adsorbed to particulate matter.	May biodegrade. Not expected to bioaccumulate.
2,4,6-Trinitrotoluene (Rust E&I 1993)	Expected to be mobile in soil and subject to leaching.	Not expected to hydrolyze.	Exists largely in the vapor phase, based on vapor pressure. Slow removal through reaction with hydroxyl radicals (half-life 110 days).	Biodegrades slowly under aerobic conditions. Not expected to bioaccumulate.
Agent Breakdown Products				
Isopropyl Methyl Phosphonic Acid (IMPA) (USEPA, 1992c)	Available information is limited. Chemically very stable (relatively little degradation likely to occur). Potentially binds to cationic soil surfaces.	Environmentally persistent due to its chemical stability and relative resistance to degradation.	NA	Available information is limited. Vascular plants probably unable to metabolize IMPA.
Fluoroacetic Acid	NA[a]	NA	NA	NA

Table 6-2. Fate and Transport for the Organic Chemicals of Potential Concern (continued)

Contaminant	Soil	Water	Air	Biological Systems
Semi-Volatile Organics				
Polynuclear Aromatic Hydrocarbons: Acenaphthene Acenaphthylene Anthracene Phenanthrene	Based on chemical characteristics, expected to adsorb to soil and be resistant to leaching.	Not soluble. Not expected to hydrolyze or photolyze.	Not volatile. May adsorb to airborne particulates; removal through wet and dry deposition.	Expected to be resistant to biodegradation. Based on physical properties, may bioconcentrate.
Bromacil	NA	NA	NA	NA
Bis(2-ethylhexyl)phthalate (Rust E&I 1993)	Strong tendency to adsorb to soil/sediment. Resistant to leaching.	Low water solubility. Expected to partition to solids such as sediment.	Not volatile. May become airborne through adsorption to particulates; removed by precipitation.	May biodegrade under aerobic conditions. Tends to bioconcentrate in aquatic organisms.
2-Chloronaphthalene	NA	NA	NA	NA
Dimethylnaphthalenes[b]	Slightly mobile to immobile in soil. Hydrolysis not significant but may undergo direct photolysis.	May partition form the water column to organic matter contained in sediments and suspended solids.	Exists largely in the vapor phase and will react with photochemically produced hydroxyl radicals (half-life of 5.1 hrs.).	Should biodegrade in the environment. Has potential to bioconcentrate in aquatic systems.
Fluorene	NA	NA	NA	NA
Methylnaphthalenes	Slightly mobile to immobile in soil. Hydrolysis not significant.	Direct photolysis half-life of 54-71 hours in water. May partition from the water column to organic matter contained in sediments and suspended solids.	Exists largely in the vapor phase and will react with photochemically produced hydroxyl radicals (half-life of 7.4 hrs.) and ozone (half-life of 28.7 to 88.2 days).	Should biodegrade where micro-organisms have acclimated to PAHs and at a moderate rate in unacclimated soils and aquatic systems. Bioconcentration not significant.

Table 6-2. Fate and Transport for the Organic Chemicals of Potential Concern (continued)

Contaminant	Soil	Water	Air	Biological Systems
2-Methylnaphthalenes	Immobile in soil. Photolysis likely to occur on sunlit soil surfaces.	May partition to organic matter contained in sediments and suspended solids.	Exists in vapor phase. Reactions with hydroxyl radicals (half-life of 7.4 hrs.) and oxides (half-life of 28.7 days).	Biodegrades rapidly where micro-organisms have acclimated to PAHs and at a moderate rate in unacclimated soil and aquatic systems.
4-Methylphenol	Poorly adsorbed to soil and should leach extensively.	Not subject to sorption and volatilization.	Reacts photochemically during day (half-life of 10 hrs.) and react nitrate radicals at night (half-life of 4 minutes).	Biodegradation is expected in water. Bioconcentration in fish is not expected.
N-nitrosodiphenylamine	Not expected to rapidly migrate or be persistent.	Moderate tendency to partition to sediments, suspended organic matter and biota.	An atmospheric half-life at 7 hours is estimated based upon reaction with hydroxyl radicals.	Biodegradation is affected by the organic carbon level in soils.
Naphthalene	Adsorbed moderately to soil and undergoes biodegradation.	Subject to volatilization, photolysis, adsorption, and biodegradation.	Rapidly photodegrades (half-life of 3-8 hrs.).	Bioconcentration occurs to a moderate extent in aquatic organisms.
3-Nitrotoluene	May leach and degrade by direct photolysis.	Susceptible to aerobic and anaerobic biodegradation. Dominant removal processes are biodegradation, photolysis, and volatilization (half life of 2.7 days).	May degrade by direct photolysis or by reaction with photochemically produced hydroxyl radicals (half life of 16.9 days).	Bioconcentration is not significant.
Palmitic Acid	NA	NA	NA	NA
TPH	NA	NA	NA	NA

Table 6-2. Fate and Transport for the Organic Chemicals of Potential Concern (continued)

Contaminant	Soil	Water	Air	Biological Systems
Trimethylnaphthalenes[c]	Slightly mobile to immobile in soil. Hydrolysis not significant.	Direct photolysis half-life of 54-71 hours in water. May partition from the water column to organic matter contained in sediments and suspended solids.	Exists largely in the vapor phase and will react with photochemically produced hydroxyl radicals (half-life of 7.4 hrs.) and ozone (half-life of 28.7 to 88.2 days).	Should biodegrade where micro-organisms have acclimated to PAHs and at a moderate rate in unacclimated soils and aquatic systems. Bioconcentration not significant.
Volatile Organic Compounds				
Acetone (Howard 1990)	Subject to evaporation and biodegradation.	Subject to biodegradation.	Degrades by reaction with photochemically produced hydroxyl radicals (half life of 22 days to 1 day).	Bioconcentration is not significant.
Benzene (Howard 1990)	Highly mobile in soil and subject to leaching.	Soluble in water. Not expected to hydrolyze.	Highly volatile. Not subject to direct photolysis. Removal by reaction with atmospheric hydroxyl radicals (half-life 4 hours to 13 days).	Can biodegrade under aerobic conditions in soil and water. Not expected to bioconcentrate.
Bromodichloromethane	Highly mobile. Removal through volatilization and leaching.	Removal through volatilization and anaerobic biodegradation.	Degrades in troposphere through reaction with hydroxyl radicals (half life of 6.6 months).	Bioconcentration not significant.
Chloroform (Howard 1990)	Poor absorbance to soil. Subject to leaching. Not expected to hydrolyze.	Soluble in water. Not expected to hydrolyze.	Volatilizes readily. Slow removal by reaction with hydroxyl radicals (half-life 80 days). May be removed by precipitation.	Biodegradation data are conflicting; some data indicate that biodegradation can occur under aerobic and anaerobic conditions. Not expected to bioaccumulate.

Table 6-2. Fate and Transport for the Organic Chemicals of Potential Concern (continued)

Contaminant	Soil	Water	Air	Biological Systems
Chloromethane	Removal through volatilization and leaching.	Rapid removal through volatilization.	Degrades in troposphere through reaction with hydroxyl radicals.	Bioconcentration is not significant.
Dibromochloromethane	NA	NA	NA	NA
1,2-Dichlorobenzene	Tendency to be moderately to tightly adsorbed. Leaching is possible.	High affinity for lipophilic materials. Low aqueous solubility. Tendency to adsorb to sediments.	Exists in vapor phase and will react photochemically produced hydroxyl radicals (half-life of 24 days).	Biodegrades under aerobic conditions. Tends to bioaccumulate.
1,4-Dichlorobenzene	Moderately to highly adsorbed. Not susceptible to hydrolysis, oxidation, or direct photolysis.	Expected to adsorb to sediments.	Exists in vapor phase and will react with hydroxyl radicals at an estimated half-life rate of 30 days.	Aerobic biodegradation in water may be possible.
1,1-Dichloroethylene	Removal through evaporation and leaching. Biodegradation expected to be low.	Removal through evaporation.	Readily biodegrades by photo-oxidation (half life of 2 to 11 hours).	Will not bioconcentrate.
1,2-Dimethylbenzene/O-xylene	Moderately mobile. May leach and biodegrade.	Volatilization is primary removal process.	Photochemically degrades by reaction with hydroxyl radicals (half life of 1.5 to 15 hours).	Bioconcentration is not significant.
1,3-Dimethylbenzene/M-xylene	Moderately mobile in soil and may leach into groundwater.	Subject to volatilization and some adsorption to sediment may occur.	May photochemically degrade by reaction with hydroxyl radicals (half-life of 1 to 10 hours).	Bioconcentration not expected to be significant.
Ethylbenzene (Howard 1990)	Adsorption to soil is moderate. Susceptible to leaching, especially from soil with a low organic carbon content. Does not hydrolyze. Slow biodegradation is likely.	Biodegradation is likely. Some adsorption to soil/sediment may occur. Photolysis and hydrolysis are not significant.	Readily volatilizes. Not expected to photolyze. Removal by reaction with hydroxyl radicals (half-life 0.5 hour to 2 days).	Biodegrades fairly rapidly by activated sludge. Resistant to anaerobic biodegradation. Does not bioaccumulate.
Methyl-n-butyl ketone/ 2-hexanone	Highly mobile. Capable of rapid biodegradation.	Removal through volatilization.	Photochemically degrades by reaction with hydroxyl radicals (half life of 2 days).	Bioconcentration not significant.

Table 6-2. Fate and Transport for the Organic Chemicals of Potential Concern (continued)

Contaminant	Soil	Water	Air	Biological Systems
Methylene chloride	Subject to evaporation and may partially leach into groundwater.	Subject to evaporation and direct photolysis. Adsorption to sediment not significant.	Degrade by reaction with photochemically produced hydroxyl radicals (half-life of a few months).	Biodegradation possible in natural waters. Bioconcentration is not significant.
Methylisobutyl ketone	Subject to direct photolysis on soil surface, volatilization, or aerobic biodegradation.	Subject to volatilization and direct photolysis. Hydrolysis is not significant.	Subject to direct photolysis and reaction with hydroxyl radicals (half-life of 16 to 17 hours).	Bioaccumulation in aquatic organisms is not significant.
Monochlorobenzene[(d)]	Subject to vaporization and slow biodegradation. Mobile in sandy soil. May leach into groundwater.	Subject to vaporization and slow biodegradation. Primary loss due to evaporation.	Reaction with hydroxyl radicals is the dominant removal mechanism with the formation of chloronitrophenols (half-life of 9 days).	Little bioconcentration is expected into fish and food products.
1,1,2,2-Tetrachloroethane	Highly mobile in soils and may leach into groundwater. Slowly biodegrades.	Volatilization half-life in water estimated to be 6.3 hours to 3.5 days. Not expected to partition from the water column to organic matter contained in sediments and suspended solids. Under alkaline conditions, expected to hydrolyze.	Rapidly photodegraded in stratosphere.	Biodegradation under anaerobic conditions. Not expected to bioconcentrate into the food chain.
Toluene (Howard 1990)	Moderate to very high mobility in soil. Susceptible to leaching. Does not significantly hydrolyze.	Does not significantly hydrolyze, photolyze, or adsorb to sediment.	Highly volatile. Volatilization from surface soil and water is a significant fate process. Degrades by reaction with hydroxyls (half-life of 3 hours to 1 day). Will also wash out with rain. Not subject to direct photolysis.	Readily biodegradable. Biodegradation occurs both in soil and water. Bioaccumulation is not significant.

Table 6-2. Fate and Transport for the Organic Chemicals of Potential Concern (continued)

Contaminant	Soil	Water	Air	Biological Systems
1,1,1-Trichloroethane	Highly volatile and leachable.	Release to surface water will decrease in concentration almost entirely due to evaporation.	Will degrade very slowly by photo-oxidation in the troposphere and slowly diffuse to the stratosphere where photodegradation will be rapid.	Not expected to bioaccumulate.
1,1,2-Trichloroethane	Highly mobile and leachable.	Removal occurs through evaporation.	Photochemically degrades by reaction with hydroxyl radicals (half life of 24 to 50 days).	Bioconcentration not significant.
Trichloroethylene (Howard 1990)	Highly mobile in soil and susceptible to leaching. Hydrolysis is not an important process.	Does not significantly hydrolyze, photolyze, or adsorb to sediment.	Highly volatile. Volatilization from soil and water is a significant fate process. Degrades rapidly through reaction with hydroxyl radicals (resident time of about 5 days). Not subject to direct photolysis.	Resistant to biodegradation. Slow biodegradation is possible under aerobic and anaerobic conditions. Bioaccumulation is not significant.
Xylene	Mobile in soil and may leach into groundwater.	Dominant removal process is volatilization.	Photochemically degrade by reaction with hydroxyl radicals (half-life of 1-18 hours).	Biodegrades in both soil and groundwater. Bioconcentration is not expected to be significant.

Note.—All data were compiled from HSDB (1993) unless otherwise noted. [The National Library of Medicine, Toxicology Data Network, Hazardous Substances Data Bank Database, accessed March, 1994.]

[a]Data not available.

[b]Criteria for 1,6-dimethylnaphthalene used in lieu of information for dimethylnaphthalene.

[c]Criteria for methylnaphthalene used in lieu of information for trimethylnaphthalene.

[d]Criteria for chlorobenzene used in lieu of information for monochlorobenzene.

bulk density, saturated hydraulic conductivity, aquifer thickness, and organic carbon content) were consistent throughout the modeling effort. Parameters associated with the site hydrogeology (aquifer thickness, mixing zone depth, and hydraulic gradient) and contaminant of interest (initial concentration, distribution coefficient, normalized distribution coefficient, and biodegradation coefficient) varied according to the contaminant being modeled.

This simplified subsurface model consisting of only one saturated zone layer adds to the conservative approach of the model. Using coarser-grained sediments for the entire thickness of the aquifer zone allows for faster simulated transport of the contaminant as it migrates through the aquifer. When available, field data were incorporated into the conceptual model. In cases where such data were not available, parameters were conservatively estimated based on information contained in the Multimedia Exposure Assessment Model (MULTIMED) Manual (Salhotra et al. 1993)

Modeling Software

MULTIMED, developed as a technical and quantitative management tool to address the problem of the land disposal of chemicals, was selected as a basic tool for this modeling effort. It utilizes analytical and semi-analytical solution techniques to solve the mathematical equations describing water flow and contaminant transport. A one-dimensional module simulates flow in the vadose zone. The output from this module, water saturation as a function of depth, is used as input to the unsaturated zone transport module. The latter simulates one-dimensional (vertical) transport and includes the effects of longitudinal dispersion, linear adsorption, and first-order decay. Output from the unsaturated zone modules is used to couple the vadose zone with the semi-analytical saturated zone transport model. The latter includes one-dimensional uniform flow, three-dimensional dispersion, linear adsorption, first-order decay, and dilution due to infiltration from the vadose zone to the groundwater plume. MULTIMED also allows use of the groundwater module alone. In this case, the source is placed directly on the water table and the vadose zone thickness is set by the model to zero (Salhotra et al. 1993).

As one of the input variables, MULTIMED requires user-specified infiltration and recharge rates. The infiltration rate is defined as the rate at which leachate percolates into the aquifer system from a land disposal facility. The recharge rate is defined as the net amount of water that percolates directly into the aquifer system outside of the disposal facility. For the purpose of this model, it was assumed that infiltration and recharge rates were the same. The value for this parameter was estimated using another model, the Pesticide Root Zone Model (PRZM).

PRZM is a one-dimensional, dynamic, compartmental model that can be used to simulate chemical movement in unsaturated soil systems within and immediately below the plant root zone. It has two major components: hydrology and chemical transport. Only the hydrology component of the model was utilized in this modeling task. The hydrology component for calculating runoff and erosion is based on the Soil Conservation Service curve number

technique and Universal Soil Loss Equation. Evaporation is estimated either directly from pan evaporation data or based on an empirical formula. Evaporation is divided among evaporation from crop interception, evaporation from soil, and transpiration by the crop. Water movement is simulated by the use of generalized soil parameters, including field capacity, wilting point, and saturation water content (Mullins et al. 1993).

The PRZM model requires the use of meteorological data files, which are compiled by the National Oceanic and Atmospheric Administration and are available for all major meteorological stations in the U.S. Each file contains daily records of precipitation, Class A pan evaporation, temperature, wind speed, and solar radiation. Salt Lake City W24127.MET file was used for PRZM simulations in this project. The file contains data from 1948 through 1983. A Fortran program PREC2.EXE was written for this modeling project in order to analyze the contents of the W24127.MET file. The analysis revealed 92.08 days with precipitation and 40.08 cm of precipitation per year on average over the analyzed period of 1948 to 1983 for that station.

No vegetation and low surface runoff values were used for PRZM simulations in order to make the model predictions conservative. The result is a higher infiltration rate than the model would predict under less conservative assumptions (with vegetation present and more surface runoff allowed). Model predictions for daily recharge were stored in the file TIMES.OUT. A Fortran program, RECH2.EXE, was written for this modeling effort to analyze the contents of that file. It was determined from the PRZM output that there are 15.86 days with recharge below root zone and 8.77 cm of infiltration per year on average.

The PRZM model does not take into account that over long periods of time without precipitation and recharge, hydraulic gradients in the vadose zone profile may be reversed and more water may be lost through evaporation (Scanlon 1994). Therefore, the average annual infiltration value predicted by the model (8.77 cm) may be considered very conservative. This higher infiltration value results in more potential for soil contaminants to leach to groundwater.

The GWM-2 spreadsheet was developed as a support tool used in the modeling process. An example of the spreadsheet printout and description are included in Appendix H.

Description of Modeling Steps

The flow chart contained in Appendix H demonstrates steps applied in modeling contaminants that have been detected in groundwater around the CAMDS facility. Although extensive amounts of surface and subsurface soil contamination data were collected, no vadose zone modeling was conducted for this site. This decision to eliminate the vadose zone modeling step was based on the results of modeling conducted for TEAD-N (Rust E&I 1994). It was concluded that under site conditions (a vadose zone only 2 meters thick with inactive contaminant-generating sites), a spill occurring 25 years ago would have already migrated through the vadose zone and reached the shallowest water-bearing unit.

All contaminants detected in groundwater, except metals and anions, were subject to modeling, regardless of whether maximum concentrations were above or below MCLs. For metals and anions, only those constituents elevated above background levels were modeled. In the first step, the maximum concentration for each contaminant was determined and the plume area was estimated. In the next step, the spreadsheet GWM-2 was used to estimate source parameters for input into MULTIMED. MULTIMED was then applied to simulate a contaminant plume comparable in size and concentration to the plume documented by groundwater data. In the final step, MULTIMED was used to simulate movement of the plume to the off-site receptor point where peak concentrations and times when the peak concentration reaches the receptor were estimated.

Selection of Contaminants of Concern for Modeling and Calculation of Representative Contaminant Concentrations

A chemical screening process was completed prior to modeling the migration of contaminants through the subsurface. This screening process and methodology is described in Section 6.1.1 of this report. For metals and anions, only those constituents elevated above background levels were modeled. The chemicals lacking USEPA health criteria/toxicity values (e.g., uranium, bromacil, IMPA, bromide, chloride, and sulfate) were not modeled.

Model Set-Up and Simulations

The lists of all MULTIMED input parameters for all simulations are included in Appendix H. The following parameters were adjusted for each simulation for a series of simulations:

- Area of waste disposal unit (for each contaminant at each site)
- Duration of pulse (for each contaminant at each site)
- Initial concentration at source (for each contaminant at each site)
- Normalized distribution coefficient, K_{oc} (for each organic contaminant)
- Distribution coefficient, K_d (for each contaminant)
- Biodegradation coefficient (for degradable contaminants only)

To configure the contaminant source, it was necessary to simulate the maximum detected contaminant concentration in groundwater and confirm that the delivery of contaminant mass is approximately equal to the estimated mass present in the detected plume. The duration of a source contaminant pulse was set to no more than 25 years, while the area of the waste disposal unit varied for each contaminant. This parameter estimation was based on the number of wells that contained detectable concentrations surrounding the CAMDS facility. The rest of the MULTIMED input parameters were held constant for all of the model simulations. The following is a list of some of the important parameters and applied values:

- Percent organic matter: 0.05
- Bulk density of aquifer material: 1.51 g/cc

- Hydraulic conductivity of aquifer: 7,760 m/yr (21.6 m/d)
- Aquifer thickness: 6 m

Parameters associated with the aquifer zone, when applicable, were based on field data presented in this report. The temperature of the aquifer and the pH were based on well development data, while the aquifer thickness and hydraulic gradients were calculated using groundwater data presented in this report and previous investigations.

The majority of the MULTIMED chemical module parameters were inactive or set to 0. Only three parameters required input: the normalized distribution coefficient, K_{oc}; distribution coefficient, K_d; and biodegradation coefficient. The K_{oc}, K_d, and biodegradation coefficients used are provided in Appendix H, and their sources are listed in Section 10.0, References. Soil organic carbon content and K_d values were selected to result in minimized retardation of the contaminants. The longest half-lives listed were selected as most conservative in estimating degradation (to make a degradable contaminant last the longest). Assignment of parameter values in this fashion results in conservative model output for the health risk assessment.

Modeling Results

A total of 30 contaminants were analyzed and modeled in 60 MULTIMED saturated zone simulations conducted for SWMU 13. This modeling consisted of initially modeling the existing plume as documented by groundwater sample analysis and, then, simulating the plume movement from its on-site position to an off-site receptor point. The off-site receptor point was conservatively chosen as the point along the TEAD-S boundary that was closest to SWMU 13 (in this case, 1,300 meters to the west-southwest). The results of modeling are presented in the report tables of Appendix H. These results are conservative for the following reasons:

(1) The PRZM-estimated recharge rate may be much larger than the actual rate as discussed previously in the subsection Modeling Software. For the higher recharge simulated in the model, faster contaminant travel times and greater contaminant mass fluxes to the aquifer are predicted as compared to likely natural conditions.

(2) Wide ranges of measured values are presented in the literature for the distribution coefficients, K_d (or K_{oc}). The lowest quoted values of K_d were used in this modeling task. The higher the K_d value, the larger is the retardation factor R and the slower the resulting movement of contaminant. It is possible that actual K_d values are much larger than those used in the model for many contaminants. In such a case, contaminant travel time would be several orders of magnitude longer than those predicted by the model. The model, therefore, predicted movement of contaminants at higher rates than may be expected. This assumption allows contaminants to travel faster through the vadose zone and aquifer and, thus, to reach receptor points more quickly.

(3) Solubility limits on contaminant concentrations in water are not imposed as a condition on calculated soil water or simulated saturated zone concentrations. The resulting modeled concentrations, therefore, may be higher than those likely to occur in the subsurface environment. For example, metals are frequently listed in literature as relatively insoluble under normal field conditions.

(4) The saturated zone modeling results for metals may be conservative because the model used groundwater contaminant concentrations as detected in unfiltered samples. Part or most of the detected mass may exist in a fixed state (it is not known to what extent laboratory analytical procedures had or had not detected fixed metal concentrations). Mass available to water partition would then be less than modeled. Therefore, the modeled contaminant concentrations in groundwater may be much higher than the concentrations likely to occur in a real aquifer.

During chemical migration from the source to the TEAD-S boundary, the plume disperses and contaminants degrade; thus, concentrations are reduced. Based on the modeling results, 8 of the 30 contaminants (acenaphthalene, anthracene, bis(2-ethylhexyl)phthalate, fluorene, naphthalene, phenanthrene, ethylbenzene, and toluene) degrade completely prior to reaching the off-site receptor point. Of the remaining contaminants, peak concentrations at the off-site receptor point ranged from 10^{-2} mg/L (for fluoride and nitrite/nitrate) to 10^{-17} mg/L (nitrobenzene and m-xylene); travel times for the peak concentration to reach the TEAD-S boundary range from 22 years (m-xylene) to 380,000 years (thallium).

Many of the modeled metals were detected at concentrations that fall within the range of typical, naturally occurring concentrations (Dragun 1988). Therefore, it is likely that some of these contaminants do not represent manmade pollutants. Several of the present and future simulated groundwater plumes overlap at the off-site receptor point, although maximum concentration for each plume will arrive at different times.

6.1.2.3.3 ***Air-Transport Modeling***. Fugitive dusts (PM_{10}) from wind erosion of soils and VOCs from site-related chemicals in soil were modeled for the 11 sites within both SWMU 13 and 17. Source areas for each site were determined based on the extent of contamination indicated by soil sampling.

PM_{10} sources included wind erosion of currently exposed site soils as well as future soils exposed because of excavation activities. Likewise, surface-soil VOC emissions were modeled to represent current exposures, while future scenarios included both surface and subsurface VOC concentrations to simulate excavated soils. For the Wastewater Lagoons Site and Boiler Blowdown Discharge Site, surface water VOC emissions were also modeled.

Emission Rate Estimations

The source of fugitive dusts at TEAD-S was assumed to be only wind erosion of soil. Open field emission rates were calculated based upon the methodology described in EPA/600/8-

85/002, *Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites* (Cowherd et al. 1985). A soil size mode of 0.1 mm was used, corresponding to the fine sand and silts present at the site. A roughness height of 2.0 cm was assumed, corresponding to flat, bare ground. An average wind speed of 3.9 meters per second (at 7 meters high) was used (based on Salt Lake City, Utah data). These calculations led to an erosional wind speed threshold of 3.75 meters per second and an average emission rate of 2.0×10^{-5} g/m^2/second.

For wind erosion of excavated soils, it was assumed that a trench (10 feet deep by 10 feet wide) was dug along the longest possible length at each site. It was then assumed that piles containing 2,000 cubic feet were placed on the surface every 20 feet. Piles were assumed to be 7.5 feet high (a 2:1 radius-to-height ratio). The resulting emission rates due to wind erosion of exposed piles were calculated using AP-42 emission factors (USEPA 1985). The calculated emission rates for the piles turned out to be very close to the open field rates, so open field rates were also used for all excavated soil areas. Excavation was assumed to occur over 1 month.

A vegetation cover of 50 percent was assumed for current and future open fields with no cover for excavated soils. This was based on photographs of the CAMDS facility. A field with 50 percent vegetation cover has half the emissions of bare soil.

VOC emission rates were determined using the following:

- EPA Chemdat7 program, using contamination rates found from on-site sampling
- Land Treatment option with default values (except that more conservative soil porosity values were chosen; no biodegradation was assumed)
- Disposal impoundment option for surface water VOC emissions
- Depth of 1 meter
- Temperature of 25 °C
- Hourly turnover in contaminated water (with no biological activity)

This produced very conservative values for surface water emissions as a continually new batch of contaminated water was available for VOC release with all the water volume being close to the surface.

Dispersion Modeling

The ISCST2 (Industrial Source Complex) dispersion model was used to simulate dispersion for the calculated PM_{10} and VOC emission rates. Square area sources were chosen to match each site area. Irregular sites were simulated with squares centered at the approximate area centroid of each site or part of a site, depending on the site shape. Some irregular sites required several squares.

For calculating current on-site worker exposure concentrations, an overall site annual average at a 5-foot breathing height was estimated from isopleths, which are plots of equal

concentration contours, created for each source. It was assumed that on-site workers change location over the course of a year. Therefore, this receptor would be exposed to an annual average concentration of each site-related chemical from each site within SWMU 13. Off-site resident concentrations were calculated for residents of St. John, located approximately 5 miles to the northwest of SWMUs. Similarly, this concentration at the off-site location was derived in such a manner that it represents the annual average contribution from each release site within both SWMUs. Exposure to future on-site residents from inhalation of chemicals was based on a chemical concentration derived using an approach similar to that used for the on-site worker, except that in this case the derived value for future on-site residents represents a maximum yearly concentration occurring anywhere within SWMU 13 or SWMU 17. Two values were derived for this receptor corresponding to different receptor heights: 5 feet for adults and 2 feet for toddlers. Future cattle exposures were evaluated assuming overall site averages at 2-foot heights. For construction workers, maximum monthly averages were calculated based on exposed soil areas due to trench digging. These trenches were 10 feet deep, 10 feet across, and dug across each site. Excavated soils were placed next to each trench.

The ISC model used 1991 meteorological data from Salt Lake City. Local meteorological data were reviewed and determined to be insufficient since a full year of monitoring data was not available. As a result, comparison of wind roses from on-site meteorological data and other nearby stations showed Salt Lake City to be the best match. Other ISC model assumptions included flat terrain and rural wind velocity profiles.

The model input parameters, the PM_{10} concentrations for various receptors, and the soil or surface-water VOC concentrations are included in Appendix H of this report. Actual exposure concentrations for each VOC were obtained by scaling the Chemdat7 emission rate for VOCs by the modeled emission rate and multiplying this by the ISC-calculated VOC air concentration. SVOC exposure concentrations were obtained by multiplying the SVOC soil concentration by the calculated PM_{10} air concentration.

6.1.2.3.4 *Summary of Exposure Point Concentrations for Current Land Use*

Soil

Site-specific exposure point concentrations were estimated for use in evaluating the dermal and ingestion exposure to contaminated soil by on-site workers. The value used originated from the sample results representing the top 6 inches of soil or the top 12 inches, if necessary.

Air

Exposure point concentrations due to volatilization of VOCs and fugitive dust emissions were estimated with the use of air emission rate and dispersion model calculations as described in Section 6.1.2.3.3. These calculations were based on the surface-soil chemical concentrations

within each site and meteorological data collected in Salt Lake City for a full year. The surface-soil concentrations used as modeling inputs were the upper-bound values representing the top 6 inches of soil or the top 12 inches, if necessary.

The on-site worker was assumed to be exposed to SWMU-wide (SWMU 13 or 17) chemicals in air; thus, the exposure point concentrations at each SWMU represent the sum of the annual average air concentration modeled at each site within the SWMU. This approach was selected because of the close proximity of the sites.

The air levels modeled for the off-site resident represent annual average concentrations from each individual site.

Groundwater

The groundwater pathway was considered incomplete for current on-site workers and, therefore, no exposure point concentrations were calculated. For the current off-site resident downgradient of SWMU 13, saturated zone computer modeling of SWMU-wide chemicals was performed as described in Section 6.1.2.3.2. The modeling results are included in Section 7.11 of this report.

Surface Water

Exposure point concentrations due to volatilization of VOCs from the surface water at the Wastewater Lagoons Site and the Boiler Blowdown Discharge Site were estimated with the use of air emission rate and dispersion model calculations as described above in Section 6.1.2.3.3. These calculations were based on the surface water chemical concentrations within each site and meteorological data collected in Salt Lake City for a full year.

6.1.2.3.5 *Summary of Exposure Point Concentrations for Future Land Use*

Soil/Sediment

Separate site-specific exposure point concentrations were estimated for use in evaluating the dermal and incidental ingestion exposure to contaminated soil and/or sediment by potential future on-site residents at SWMUs 13 and 17. For the future on-site resident, the values were the same as those developed for on-site workers under the current land use scenario. The exposure point concentrations used to evaluate the construction worker originated from the soil-sample results representing the 0-to-10-foot sample-depth interval.

Air

Exposure point concentrations due to volatilization of VOCs and fugitive dust emissions were estimated with the use of air emission rate and dispersion model calculations as described in Section 6.1.2.3.3. The surface-soil concentrations used as model inputs were 95 percent

UCL of the mean, or the maximum value (whichever was lower), from the top 6 inches of soil or the top 12 inches, if necessary, for the on-site resident, and subsurface soil for the construction worker.

The on-site resident was assumed to be exposed to SWMU-wide (SWMU 13 or 17) chemicals in air; thus, the exposure point concentrations at each SWMU represent the sum of the annual maximum air concentrations modeled at each site within the SWMU. This approach was selected because of the close proximity of sites.

The air levels modeled for the construction worker represent maximum monthly values that are site-specific.

Groundwater

SWMU-wide exposure point concentrations for groundwater ingestion and dermal contact by potential future on-site residents were estimated from current groundwater data representing all monitoring wells at SWMU 13. Inhalation of VOCs in groundwater by potential future on-site residents during showering or bathing was evaluated based on average shower stall air concentrations derived using the corresponding groundwater concentrations. The value used as the exposure point concentration was either the 95 percent UCL of the mean, or the maximum value (whichever was lower), as calculated from the site groundwater data.

Vegetable Consumption

Chemical contamination of future garden produce, as well as pasture grasses, can arise from aerial deposition of contaminated airborne particulates and via chemical uptake from contaminated soil. Under the proposed future land use scenario, however, vegetative land cover is expected to prevent significant wind erosion and to prevent the consequent generation of fugitive dust at concentrations likely to impact plant tissues. Therefore, only root uptake of analytes from contaminated surface soil into the edible portions of vegetables and crops was modeled. Root uptake of chemical contaminants into plant biota at each site (i.e., a site-specific evaluation) was calculated as follows:

(Equation 6-1)

$$CP = RUF \times CS \times DWF$$

where

CP	=	Contaminant level in vegetables (mg/kg)
RUF	=	Root uptake factor (unitless)
CS	=	Contaminant concentration in surface soil (mg/kg)
DWF	=	Dry-to-wet weight conversion factor (unitless)

Vegetation accumulation was calculated for the following crops:

- Potatoes
- Carrots
- Legumes
- Lettuce
- Tomatoes
- Grasses

Contaminant levels in vegetables are expressed on a wet-weight basis. Levels in cattle feed are expressed on a dry-weight basis since feed consumption rates are on a dry-weight basis.

The following are dry-to-wet conversion factors for various vegetables: potatoes, 0.25; tomatoes, 0.06; carrots, 0.12; legumes, 0.28; and lettuce, 0.05. The dry-weight root uptake factors for metals were taken from NRC (1992). A chemical partition approach was utilized to quantify RUF for organic chemicals. The following regression equation published by Travis and Arms (1988) for leafy vegetables was utilized to estimate dry weight root uptake factors for above-ground vegetation based on the K_{ow} value for that COPC:

(Equation 6-2)

$$\log RUF = 1.588 - 0.578 (\log K_{ow})$$

The concentration of organic chemicals in below-ground vegetation (i.e., root vegetables) can be estimated by a method developed by Briggs (1982):

(Equation 6-3)

$$CR = \frac{(CS)\ (RCF)\ (VG_{bg})}{Kd_s}$$

where

CR	=	Contaminant level in root vegetable (mg/kg fresh weight)
CS	=	Contaminant level in soil (mg/kg)
RCF	=	Root concentration factor, L soil water/kg plant tissue
VG_{bg}	=	Empirical correction factor, unitless
Kd_s	=	Soil/water partition coefficient, L soil water/kg soil

VG_{bg} is a factor introduced into the calculation of contaminant concentrations to reflect the reduced translocation of compounds in bulky, below-ground vegetables, such as carrots and potatoes. This factor is 0.01 for all chemicals (USEPA 1994). The Kd_s for organic compounds were estimated by multiplying the lowest Koc (see Table 6-1) by an assumed

fraction of organic carbon in surficial soils of 0.01. The RCF values for organics were calculated using the following regression equation from Topp et al. (1986):

(Equation 6-4)

$$\log RCF = 0.63 \log K_{ow} - 0.959$$

Beef Consumption

Beef cattle grazing on contaminated land may accumulate chemicals in their muscle tissue (including fat). Cattle were assumed to have equal access to all of TEAD-S, including all sites as well as uncontaminated areas. Beef and milk concentrations were first calculated for each site, assuming cattle graze only at a single site (other feed, such as hay or grain, assumed to originate off-site). Surface soil, forage, and air concentrations of chemicals were based on high end (upper-bound) soil concentrations for each site. Final beef and milk concentrations to which the future resident is exposed were calculated by multiplying the site-specific concentrations by a "proportion factor," which was derived by dividing the area of each site by the area of TEAD-S (approximately 8.6E+08 ft^2), then summing the proportions contributed by each site. It should be noted that the beef exposure point concentrations derived for this pathway represent average values used to evaluate exposure over all of TEAD-S (i.e., depot-wide values). Therefore, the future on-site resident at each site is exposed to site-specific chemicals in other media and also to those associated with beef due to all sites within SWMUs 13 and 17. Exposure to beef by future residents at the fuel spill site, the UST site, and the NaOH site was evaluated since these receptors are exposed to chemicals originating from other sites within SWMUs 13 and 17 despite that site-specific chemicals were not measured in the surface soil at these locations. The equation used to estimate the potential site-related chemical concentration in beef is:

(Equation 6-5)

$$CB = ICB \times BB$$

where

CB	=	Concentration of chemical in beef (mg/kg)
ICB	=	Daily chemical intake by beef cattle (mg/d)
BB	=	Biotransfer factor for beef (d/kg)

Daily chemical intake (ICB) by beef cattle was calculated as follows:

(Equation 6-6)

$$ICB = IH + IS + II$$

where

IH = Chemical intake from hay/grasses (3,080 mg/d, dry weight (NRC 1992))
IS = Chemical intake from soil (154 mg/d (NRC 1992))
II = Chemical intake from inhalation (100 m^3/d (University of Minnesota 1986))

Chemical intake from hay/grasses (IH) by cattle was calculated using the following general equation:

(Equation 6-7)

$$IH = CV \times CR \times F$$

where

CV = Chemical level in feed type (mg/kg)
CR = Cattle consumption rate of feed type on a dry-weight basis (kg/d)
F = Weight fraction of feed from contaminated area (unitless)

Chemical intake by cattle via soil ingestion (IS) was calculated as follows:

(Equation 6-8)

$$IS = CS \times SCR$$

where

CS = Chemical level in surface soil (mg/kg)
SCR = Cattle soil consumption rate = 0.19 kg/d (Fries 1987)

Chemical intake by cattle via inhalation (II) was calculated as follows:

(Equation 6-9)

$$II = CA \times IR$$

where

CA = Chemical concentration in air (mg/m^3)
IR = Inhalation rate = 20.0 m^3/d (beef animal (Mitchell 1990))

Beef biotransfer factors for metals were taken from Stevens (1992) and NRC (1992). Biotransfer factors for organic compounds were calculated using the following regression equation (Travis and Arms 1988):

(Equation 6-10)

$$\log BB = -7.6 + \log Kow$$

Milk Consumption

Dairy cattle may also be exposed to COPCs via inhalation of air and ingestion of contaminated soil and vegetation. These chemicals could then bioaccumulate in milk to a steady-state condition. The general equation used to calculate the potential concentration of chemicals in bovine milk (depot-wide evaluation) is:

(Equation 6-11)

$$CM = ICM \times BM$$

where

CM	=	Concentration of chemical in milk, representing an average depot-wide value (mg/kg)
ICM	=	Daily chemical intake by dairy cattle (mg/d)
BM	=	Biotransfer factor for milk (d/kg)

ICM for the dairy cow was calculated in the identical manner that ICB was calculated for the beef animal. The dairy cow was assumed to consume 8 kg/d of forage (dry-weight basis) and 0.4 kg soil a day. The dairy cow inhalation rate of 150 m^3/d was used. Milk biotransfer factors for metals were taken from Stevens (1991) and NRC (1992). Biotransfer factors for organic compounds were calculated using the following regression equation (Travis and Arms 1988):

(Equation 6-12)

$$\log BM = -8.1 + \log Kow$$

6.1.2.4 *Development of Chemical Intakes*

Chemical-specific intakes or chronic daily intakes (CDIs) were calculated for complete exposure pathways identified for current and potential future land use scenarios. Intakes are estimated as milligram of contaminant per kilogram body weight per day. The equations used to determine chemical intakes and the assumptions employed in the equations are

discussed below. Exposure parameters used in the intake equations to estimate the CDI include the chemical concentration in the exposure medium, exposure time, exposure frequency, exposure duration, USEPA-recommended intake rates, and media-specific exposure factors. Default exposure parameters recommended by the USEPA were used as the RME value unless site-specific values were available. Most of the exposure parameters used to estimate the CDI are summarized in Tables 6-3 and 6-4 for both current and potential future land use scenarios, respectively.

6.1.2.4.1 ***Current Land Use.*** Exposure pathways considered complete for the on-site worker include incidental soil ingestion, soil dermal contact, and inhalation of volatiles and fugitive dust. Exposure pathways considered complete for the off-site resident include inhalation of volatiles and fugitive dust emissions due to volatilization and wind erosion. Based on the results of the saturated zone modeling described in Section 7.11.3.1.2, exposures to this receptor from ingestion of and dermal contact with site-related chemicals in groundwater, and inhalation of volatiles from groundwater during showering and bathing were not evaluated. The sections below describe the approach used to develop CDIs for these complete pathways.

On-Site Worker-Incidental Ingestion of Soil

Incidental ingestion of soil particulates occurs by the accidental ingestion of particles present on the hands and by swallowing particles collected in the nasal passages. The level of exposure from incidental ingestion depends on the site-related chemical concentration in soil, the amount ingested, and the frequency and duration of exposure. The intake of chemicals through this pathway is estimated by:

(Equation 6-13)

$$Intake\ (mg/kg\text{-}day) = \frac{CS \times IR \times FI \times EF \times ED \times CF}{BW \times AT}$$

where

CS	=	Concentration of chemical in soil (mg/kg)
IR	=	Adult ingestion rate (60 mg/day)
FI	=	Fraction ingested from chemical source (1.0)
EF	=	Exposure frequency (23.1 d/year for SWMU 13, and 104 d/year for SWMU 17)
ED	=	Exposure duration (25 years)
CF	=	Conversion factor (10^{-6} kg/mg)
BW	=	Adult body weight (70 kg)
AT	=	Averaging time (25,550 days for carcinogens = 70-year lifetime x 365 days/year; 9,125 days for noncarcinogens = 25-year exposure duration x 365 days/year)

Table 6-3. Summary of Exposure Factors for Current Land Use Conditions

Exposure Pathway	Contact Rate	Surface Area (cm^2)	Exposure Frequency (days/years)	Exposure Time (hr/day)	Exposure Duration (years)	Source (unless noted)
On-Site Worker						
Soil Ingestion	60 mg/day	NA	23.1(104)	NA	25	USEPA, 1991
Soil Dermal Contact[a]	NA	4,590[b]	23.1(104)	NA	25	USEPA, 1991
Inhalation (part. & vapor)	2.5 m^3/hr	NA	23.1(104)[c]	10	25	USEPA, 1991
Off-Site Resident						
Inhalation (part. & vapor)						
Adult	0.8 m^3/hr[d]	NA	350	24	30	USEPA, 1991
Child	0.6 m^3/hr	NA	350	24	6	USEPA, 1991

Notes.—NA indicates not applicable. Body weight used is 70 kg for an adult and 15 kg for a child.

[a]Exposure from soil dermal contact is based on absorption factor of 0.001 for inorganics and 0.01 for organics, and an adherence factor of 1.0 mg/cm^2-event (USEPA 1989d).

[b]Value taken from USEPA, 1989d (pg. 4-10) for an individual wearing short-sleeved shirt, pants, and shoes. Exposed areas will be head, arms, and hands.

[c]These values based on a 10-hour workday, four days a week, for 52 weeks and, have been adjusted to reflect the exposure by the on-site worker at a single release site.

[d]These values taken from USEPA, 1989d, pg. 3-4 for a child at rest and performing light activities at other times, and an adult performing light duty, respectively.

Table 6-4. Summary of Exposure Factors for Future Land Use Conditions

Exposure Pathway	Contact Rate	Surface Area (cm^2)	Exposure Frequency (days/years)	Exposure Time (hr/day)	Exposure Duration (years)	Source
Construction Worker						
Soil Ingestion	480 mg/day	NA	30[a]	NA	5[a]	USEPA, 1989d, pg. 2-52
Soil Dermal Contact[b]	NA	4,590[c]	30	NA	5	USEPA, 1989d, pg. 4-15
Inhalation (part. & vapor)	2.5 m^3/hr	NA	30	10	5	USEPA, 1989d, pg. 3-4
On-Site Resident						
Soil Ingestion						
Adult	100 mg/day	NA	350	NA	30	USEPA, 1991
Child	200 mg/day	NA	350	NA	6	USEPA, 1991
Soil Dermal Contact						
Adult	NA	4,590	350	NA	30	USEPA, 1991
Child	NA	2,650	350	NA	6	USEPA, 1992b, pg. 8-10 & 8-12
Inhalation (part. & vapor)						
Adult	0.8 m^3/hr[d]	NA	350	24	30	USEPA, 1991
Child	0.6 m^3/hr	NA	350	24	6	USEPA, 1991
Groundwater Ingestion						
Adult	2 L/day	NA	350	NA	30	USEPA, 1991
Child	1 L/day	NA	350	NA	6	USEPA, 1989d, pg. 2-1
Groundwater Dermal Contact						
Adult	NA	20,000	350	0.16	30	USEPA, 1992b, pg. 8-20
Child	NA	6,640	350	0.16	6	USEPA, 1992b, pg. 8-20
Groundwater Vapor Inhalation						
Adult	0.8 m^3/hr	NA	350	0.16	30	USEPA, 1992, pg. 8-20
Child	0.6 m^3/hr	NA	350	0.16	6	USEPA, 1992, pg. 8-20
Homegrown Beef Consumption						
Adult	199 g/day[e]	NA	350	NA	30	USEPA, 1991
Child	88 g/day	NA	350	NA	6	USEPA, 1991

Table 6-4. Summary of Exposure Factors for Future Land Use Conditions (continued)

Exposure Pathway	Contact Rate	Surface Area (cm^2)	Exposure Frequency (days/years)	Exposure Time (hr/day)	Exposure Duration (years)	Source
Homegrown Vegetable Consumption						
Adult	719 g/day[f]	NA	350	NA	30	USEPA, 1991
Child	408 g/day	NA	350	NA	6	USEPA, 1991
Milk Consumption						
Adult	0.66 L/day[g]	NA	350	NA	30	USEPA, 1991
Child	0.81 L/day	NA	350	NA	6	USEPA, 1991

Notes.—NA indicates not applicable. Body weight used is 70 kg for an adult and 15 kg for a child.

[a]There is no guidance available for this value, but it is assumed that exposure of a construction worker to subsurface soil occurs for a total of 30 days/yr (days randomly distributed over the entire 12 month period), each year for a duration of 5 years.

[b]Exposure from soil dermal contact is based on absorption factor of 0.001 for inorganics and 0.01 for organics, and an adherence factor of 1.0 mg/cm^2-event.

[c]Value taken from USEPA, 1989d (pg. 4-10) for an individual wearing short-sleeved shirt, pants and shoes. Exposed areas will be head, arms and hands.

[d]These values taken from USEPA, 1989d, pg. 3-4 for a child at rest and performing light activities at other times, and an adult performing light duty, respectively.

[e]These values were taken from Pao (1982). The average percent of annual homegrown beef consumption (44%) recommended in USEPA, 1989d pg. 2-25 will be used to estimate CDI.

[f]Both values represent the total vegetable consumption. The consumption rates were taken from Pao (1982 and are as follows: potatoes—209 for adults and 123 for children; tomatoes—133 for adults and 67 for children; carrots—130 for adults and 85 for children; beans/peas—181 for adults and 104 for children; lettuce—66 for adults and 29 for children.

[g]Both values taken from Pao (1982).

The surface-soil EPCs representing the upper 6 or 12 inches of soil were used to estimate the CDI. The soil ingestion rate of 60 milligrams per day for the RME case is the standard default value recommended by USEPA (1991) for commercial/industrial land use (50 mg/d), adjusted for an exposure frequency of 208 days per year (d/yr). The fraction ingested from a chemical source is 1.0, calculated under the assumption that all soil ingested by industrial workers is derived from the contaminated sites in the industrial area. It should be noted that exposures to on-site workers were evaluated by assuming that a single worker visits all nine sites equally in each workday within SWMU 13, since all of these sites are in close proximity to each other. Similarly, at SWMU 17, it was assumed that a single worker spends parts of his 10-hour workday at both sites. Therefore, based on a 4-day work week (208 work days/year), a mathematical exposure frequency for each site was calculated to be 23.1 d/yr for the worker at SWMU 13 and 104 d/yr for the worker at SWMU 17. These site-specific exposure frequencies are the equivalent number of days per year that a worker would have to go to each site all day to receive the same exposure as he/she would get visiting all sites each day. This worker exposure scenario was adopted because it was felt that it more realistically describes how these exposures could occur within an active facility without compromising the USEPA requirement that the scenario evaluate a reasonable maximum exposure. A worker is assumed to remain at this facility for 25 years as a reasonable maximum upper duration (USEPA 1991). The standard default parameters of 70 kilograms (154 pounds) for adult body weight and 70 years for average life span are assumed (USEPA 1989b). The averaging time used for carcinogens is 365 days per year for a 70-year lifetime and for noncarcinogens is 365 days per year for the applicable exposure duration. The difference in averaging times relates to the different mechanisms of action for carcinogens and noncarcinogens, based on the assumption that a higher dose of a carcinogen received over a shorter period of time is equivalent to a corresponding lower dose spread over a lifetime (USEPA 1989b). The averaging time for noncarcinogens is for the duration of exposure, not for a lifetime.

On-Site Worker-Dermal Contact with Soil

Chemical exposure can occur when contaminated soils contact dermal surfaces and compounds are absorbed through the skin. The level of potential chemical exposure by dermal contact is a function of the soil chemical concentration, the area of the skin exposed, the amount of soil adhering to the skin, receptor body weight, and frequency and duration of exposure. The absorbed dose of contaminants through this pathway is estimated using the following equation:

(Equation 6-14)

$$Dose\ (mg/kg\text{-}day) = \frac{CS \times SA \times AF \times ABS \times EF \times ED \times CF}{BW \times AT}$$

where

CS = Concentration of chemical in soil (mg/kg)

SA = Skin surface area for contact (4,590 cm^2)
AF = Soil-to-skin adherence factor (1.0 mg/cm^2-event)
ABS = Adult skin absorption factor (0.001 for inorganics, 0.01 for organics USEPA Region IV recommendations)
EF = Exposure frequency (23.1 d/year for SWMU 13, and 104 d/year for SWMU 17)
ED = Exposure duration (25 years)
CF = Conversion factor (10^{-6} kg/mg)
BW = Adult body weight (70 kg)
AT = Averaging time (25,550 days for carcinogens = 70-year lifetime x 365 days/year; 9,125 days for noncarcinogens = 25-year exposure duration x 365 days/year)

The high-end values representative of the upper 6 or 12 inches of soil were used to estimate the absorbed doses for this pathway. Exposure factors used are based on estimates of soil-to-skin adherence and skin absorption as reported by USEPA (1989d). Exposed skin surface area is based on values for head, arms, and hands of adult males. Since average body weights are used in these equations, average surface areas were also included since these two parameters are related. Exposures to on-site workers were evaluated by assuming that a single worker visits all nine release units within SWMU 13. Similarly, at SWMU 17 it was assumed that a single worker spends his 10-hour workday between both release units. Therefore, exposure frequencies of 23.1 and 104 days per year per release unit were used for the worker at SWMU 13 and SWMU 17, respectively.

On-Site Worker-Inhalation of Particulates and Volatiles

Exposure associated with the inhalation of particulates and volatiles was estimated for workers using the equation:

(Equation 6-15)

$$Intake\ (mg/kg\text{-}day) = \frac{CA \times IR \times DF \times ET \times EF \times ED}{BW \times AT}$$

where

CA = Concentration of chemical in air (mg/m^3)
IR = Adult inhalation rate (2.5 m^3/hour)
DF = Deposition Fraction (1.0 for VOCs; 0.3 for particulate)
ET = Exposure time (10 hours/workday)
EF = Exposure frequency (23.1 d/year for SWMU 13 and 104 d/year for SWMU 17)
ED = Exposure duration (25 years)
BW = Adult body weight (70 kg)

AT = Averaging time (25,550 days for carcinogens = 70-year lifetime x 365 days/year; 9,125 days for noncarcinogens = 25-year exposure duration x 365 days/year)

The approach for estimating the annual average exposure point concentrations for site-related chemicals in the air over all of SWMU 13 and SWMU 17 is described in Section 6.1.2.3.3 (Air Transport Modeling). The standard default value of 2.5 m^3 per hour was used for worker inhalation (USEPA 1991). An exposure time of 10 hours per day was used based on the 10-hour workday at CAMDS facility. The recommended value of 0.3 for the deposition fraction of particulates to the deep lung with nose breathing was used (Giacomelli-Maltoni 1972). This value assumes uniform particulate size distribution of less than or equal to 10 microns in size (i.e., the respirable fraction). The deposition fraction of 0.3 was not used if the studies upon which the inhalation toxicity values are based were conducted using particulate-bound chemicals. VOCs were assumed to be 100 percent available for adsorption in the deep lung.

On-Site Worker-Inhalation of Volatiles

Chemical exposure from this pathway by the on-site worker was estimated using the same approach and exposure factors utilized for inhalation of fugitive dust emissions by the on-site worker.

Off-Site Resident-Inhalation of Volatiles and Fugitive Dust

Chemical exposure from this pathway by the off-site resident was estimated using the same approach and intake equation as for the on-site worker. The exposure by a child was added to the evaluation as well as slightly different exposure factors to account for specific activity patterns associated with this receptor population. The inhalation rates used were 0.8 and 0.6 m^3/hour for an adult and child, respectively; the exposure frequency and time used were 350 days/year and 24 hours/day; the exposure durations used were 30 and 6 years; and the averaging times used for noncarcinogenic effects were 10,950 and 2,190 days. The body weights were 70 and 15 kg (adult and child, respectively).

6.1.2.4.2 ***Future Land Use***. Exposure pathways considered complete for the construction worker are dermal contact with and incidental ingestion of contaminated subsurface soils and inhalation of volatiles and fugitive dust emissions. Exposure pathways considered complete for the potential future on-site resident are dermal contact with and incidental ingestion of contaminated surficial soils and sediment; inhalation of volatiles and fugitive dust emissions; inhalation of VOC emissions from groundwater; dermal contact with and ingestion of groundwater; and consumption of homegrown beef, vegetables, and milk. The approach for estimating the CDI for the construction worker from the above-mentioned pathways is described below. The approaches for estimating the CDI for consumption of homegrown beef, produce, and milk are also described below. The CDIs for the remaining pathways were estimated using the same equations as for the on-site worker or off-site resident with the use of the exposure factors in Tables 6-3 and 6-4.

On-Site Construction Worker-Dermal Contact with and Incidental Ingestion of Soil

Chemical exposure from these pathways to an on-site construction worker was estimated using the same intake equations as for the on-site worker. The 95 percent UCL of the mean, or the maximum detected value, whichever was lower, from the upper 10 feet of soil were used as the EPCs for both pathways. The activity pattern of a construction worker differed somewhat from that of an on-site worker and, thus, in some cases the exposure factors changed. This receptor was assumed to have a larger soil ingestion rate of 480 mg/day. The construction worker is estimated to work at the site for a total duration of 5 years and a frequency of 30 days/year in projects that involve disturbing site soils (e.g., new building construction, road building, and utility installation). The exposure frequency of 30 days per year is assumed to occur randomly over the entire 12-month period and not as a single period of 30 consecutive days. The averaging time used for noncarcinogenic effects was 1,825 days (365 days per year x 5 years). The dermal absorbed dose was based on an exposed skin-surface area of 4,590 cm^2.

On-Site Construction Worker-Inhalation of Volatiles and Fugitive Dust

Chemical exposure from this pathway by an on-site construction worker was estimated using the same intake equation utilized to estimate exposure to the on-site worker. The soil concentrations representative of the upper 10 feet of soil were used as the exposure point concentrations. The exposure was based on an elevated inhalation rate of 2.5 m^3/hour. The exposure duration and frequency used were 5 years and 30 days/year, respectively. The averaging time used was 1,825 days.

On-Site Resident Dermal Contact with and Incidental Ingestion of Soil

Chemical exposure due to direct contact with soil (i.e., the soil pathway) by future on-site residents was estimated using the same intake equations as for the construction worker. Dose input parameters used for future on-site residents (such as FI, AF, ABS, and CF) were the same as those selected for the construction worker, except as described below.

The 95 percent UCL of the mean, or the maximum detected value, whichever was lower, from the surface soil (the upper 6 or 12 inches, as necessary, based on sampling intervals) were used as the EPC for both the oral and dermal exposure routes. As shown on Table 6-4, the soil ingestion rates for the adult and child were 100 and 200 mg/day, respectively. The dermal doses for the adult and child were based on exposed skin-surface areas of 4,590 and 2,650 cm^2, respectively. The exposure dose parameters EF, ED, BW, and AT assumed for the soil pathway were the same as those selected for the air pathway with respect to current off-site residents (inhalation exposure).

On-Site Resident-Beef Consumption

Exposure associated with consumption of beef derived from cattle grazing at TEAD-S was estimated using the equation:

(Equation 6-16)

$$Intake\ (mg/kg\text{-}day) = \frac{CB \times FI \times IR \times EF \times ED \times CF}{BW \times AT}$$

where

CB	=	Concentration of chemical in beef (mg/kg)
FI	=	Fraction of homegrown beef ingestion (0.44; USEPA 1989d)
IR	=	Beef ingestion rate (88 g/day for child; 199 g/day for adult)
EF	=	Exposure frequency (350 days/year)
ED	=	Exposure duration (6 years for child; 30 years for adult)
CF	=	Conversion factor (10^{-3} kg/g)
BW	=	Body weight (15 kg for a child; 70 kg for an adult)
AT	=	Averaging time (25,550 days for carcinogens = 70-year lifetime x 365 days/year; 10,950 days for noncarcinogens = 30-year exposure duration x 365 days/year); and 2,190 days for 6-year child exposure

The approach to estimating the exposure point concentrations in beef, vegetables, and milk grown on-site is described Section 6.1.2.3.5.

On-Site Resident Consumption of Homegrown Produce

Exposure by the future on-site resident from consumption of produce grown on-site was estimated using the equation:

(Equation 6-17)

$$Intake\ (mg/kg\text{-}day) = \frac{\sum_{i=1}^{5} (CP_i \times IR_i) \times FI \times EF \times ED \times CF}{BW \times AT}$$

where

CP_i	=	Concentration of chemical in i^{th} vegetable (mg/kg)
IR_i	=	Ingestion rate of corresponding vegetable (see bottom of Table 6-4)
FI	=	Fraction of homegrown produce consumption (0.40 for vegetables, USEPA 1989b)
EF	=	Exposure frequency (350 days/year)
ED	=	Exposure duration (6 years for child; 30 years for adult)
CF	=	Conversion factor (10^{-3} kg/g)

BW	=	Body weight (15 kg for a child; 70 kg for an adult)
AT	=	Averaging time (25,550 days for carcinogens = 70-year lifetime x 365 days/year; 10,950 days for noncarcinogens = 30-year exposure duration x 365 days/year) and 2,190 days for 6-year child exposure

It should be noted that the exposure from homegrown vegetables is based on the total exposure resulting from consumption of the five vegetables discussed in Section 6.1.2.3.5.

On-Site Resident Consumption of Milk

Exposure by the future on-site resident from consumption of milk produced on-site was estimated using the equation:

(Equation 6-18)

$$Intake\ (mg/kg\text{-}day) = \frac{CM \times IR \times FI \times EF \times ED}{BW \times AT}$$

where

CM	=	Concentration of chemical in milk (mg/L)
IR	=	Milk ingestion rate (0.81 L/day for child; 0.66 L/day for adult)
FI	=	Fraction of home produced milk consumption (value of 1.0 used)
EF	=	Exposure frequency (350 days/year)
ED	=	Exposure duration (6 years for child; 30 years for adult)
BW	=	Body weight (15 kg for a child; 70 kg for an adult)
AT	=	Averaging time (25,550 days for carcinogens = 70-year lifetime x 365 days/year; 10,950 days for noncarcinogens = 30-year exposure duration x 365 days/year; and 2,190 days for 6-year child exposure)

On-Site Resident Groundwater Ingestion

Exposure to site-related chemicals can occur by ingestion of contaminated groundwater by potential future on-site residents. The level of potential chemical exposure is a function of the groundwater chemical concentration, the ingestion rate, and the frequency and duration of exposure. The intake of contaminants through this pathway is estimated by:

(Equation 6-19)

$$Intake\ (mg/kg\text{-}day) = \frac{CW \times IR \times EF \times ED}{BW \times AT}$$

where

CW	=	Concentration of chemical in groundwater (mg/L)
IR	=	Ingestion rate (1 L/day for child; 2 L/day for adult)

EF	=	Exposure Frequency (350 days/year)
ED	=	Exposure duration (6 years for child; 30 years for adult)
BW	=	Body weight (15 kg for child; 70 kg for adult)
AT	=	Averaging time (25,550 days for carcinogens = 70-year lifetime x 365 days/year; 10,950 days for noncarcinogens = 30-year exposure duration x 365 days/year; and 2,190 days for 6-year child exposure)

On-Site Resident Groundwater Dermal Contact

Chemical exposure can occur when contaminated groundwater contacts dermal surfaces during showering and bathing and organic compounds are absorbed through the skin. The level of potential chemical exposure by dermal contact is a function of the groundwater chemical concentration, the available surface skin area, the chemical dermal permeability constant, and the exposure time, frequency, and duration. The intake of chemicals through this pathway is estimated by:

(Equation 6-20)

$$Absorbed\ Dose\ (mg/kg\text{-}day) = \frac{CW \times SA \times PC \times ET \times EF \times ED \times CF \times AAF}{BW \times AT}$$

where

CW	=	Concentration of chemical in groundwater (mg/L)
SA	=	Skin surface area available for contact (6,640 cm^2 for child; 20,000 cm^2 for adult)
PC	=	Chemical dermal permeability constant (cm/hr)
ET	=	Exposure time (0.16 hrs/day)
EF	=	Exposure frequency (350 days/yr)
ED	=	Exposure duration (6 years for child; 30 years for adult)
CF	=	Volumetric conversion factor for water (1 liter/1000 cm^3)
AAF	=	Absorbed-to-administered-dose adjustment factor (unitless)
BW	=	Body weight (15 kg for child; 70 kg for an adult)
AT	=	Averaging time (25,550 days for carcinogens = 70-year lifetime x 365 days/year; 10,950 days for noncarcinogens = 30-year exposure duration x 365 days/year; and 2,190 days for 6-year child exposure)

The default permeability constant (PC) for inorganic chemicals was assumed to be 1.0E-03 (the PC for water; USEPA 1989d). For organic chemicals, the PC was calculated using the following formula (Brown and Rossi 1989):

(Equation 6-21)

$$PC = 0.1 \left[K_{ow}^{0.75}/120 + K_{ow}^{0.75})\right]$$

The K_{ow} for the chemicals for which dermal exposure to chemicals in water was evaluated can be ascertained from Table 6-1, which summarizes the physicochemical properties for the organic chemicals of potential concern. Because a K_{ow} is not published for IMPA, the maximum PC determined for the groundwater COPCs, 9.6E-02 cm/hr (for phenanthrene), was conservatively used as a surrogate PC value for IMPA.

Doses calculated using the dermal PC represent absorbed doses of the chemicals. These doses required adjustment to an administered dose since, for purposes of risk characterization, they were compared with oral toxicity values which are expressed as administered doses. This adjustment was made using an absorbed-to-administered-dose adjustment factor, which was calculated as follows:

(Equation 6-22)

$$\text{AAF} = \text{Absorbed-to-administered-dose Adjustment Factor (unitless)} = \frac{1.0}{\text{Oral Absorption Efficiency}}$$

When available, chemical-specific oral absorption efficiencies were used. In the absence of chemical-specific information, an oral absorption efficiency default value of 100 percent was assumed for VOCs, and a value of 50 percent was assumed for SVOCs and inorganic chemicals.

On-Site Resident - Inhalation of VOCs from Groundwater

Exposure associated with the inhalation of VOCs in groundwater by potential future on-site residents during showering or bathing was estimated using the equation:

(Equation 6-23)

$$Intake\ (mg/kg\text{-}day) = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT}$$

where

CA	=	Average concentration of chemical in air (mg/m^3)
IR	=	Inhalation rate (0.6 m^3/hour for child; 0.8 m^3/hour for adult)
ET	=	Exposure time (0.16 hours/day)
EF	=	Exposure frequency (350 days per year)
ED	=	Exposure duration (6 years for child; 30 years for adult)
BW	=	Body weight (15 kg for child; 70 kg for adult)
AT	=	Averaging time (25,550 days for carcinogens = 70-year lifetime x 365 days/year; 10,950 days for noncarcinogens = 30-year exposure duration x 365 days/year; and 2,190 days for 6-year child exposure)

The average air VOC concentration (CA) in a shower stall was estimated based on the rate of volatilization of VOCs from water droplets during a shower. VOCs in droplets were assumed to be released through a process of molecular diffusion in both the water and air phases, which were modeled using two-film gas-liquid mass transfer theory. A more detailed description of the model used is included in Appendix H of this report.

6.1.3 Toxicity Assessment

The purpose of the toxicity assessment is to weigh available evidence regarding the potential for COPCs to cause adverse effects in exposed individuals and to provide, where possible, an estimate of the relationship between the extent of exposure to a chemical and the increased likelihood and/or severity of adverse effects. An overview of the toxicity of the COPCs is given in this section. Toxicity information was obtained primarily from the IRIS (USEPA 1997) and the HEAST (USEPA 1995). The National Center for Environmental Assessment (NCEA) was contacted for provisional values if instructed to do so by HEAST.

6.1.3.1 *Carcinogens*

A slope factor (formerly called potency factor) and an accompanying weight-of-evidence determination are the toxicity data most commonly used to evaluate potential carcinogenic risks in an exposed population.

In determining the weight-of-evidence, available data are evaluated to determine the likelihood that the agent is a human carcinogen. The evidence is characterized separately for human studies and animal studies as either sufficient, limited, inadequate, no data, or evidence of no effect. The characterizations of these two types of data are combined and, based on the extent to which the agent has been shown to be a carcinogen in experimental animals, or humans, or both, the agent is given a provisional weight-of-evidence classification. The USEPA classification system, based on the strength of evidence that a chemical is a human carcinogen, places each chemical into one of the following classes: A—sufficient human evidence; B1—limited human evidence but sufficient animal evidence; B2—inadequate human evidence but sufficient evidence in animals (both B1 and B2 are considered probable carcinogens); C—no evidence in humans and limited evidence in animals; D—no adequate data (non-classifiable); and E—evidence of noncarcinogenicity for humans.

The USEPA's Carcinogen Assessment Group calculates slope-factor estimates of the excess cancer risk due to continuous exposure to a chemical throughout the course of a 70-year lifetime—for suspected carcinogens. Slope factors are usually the upper 95th percent confidence limit of the slope of the dose-response curve and are expressed in units of $(mg/kg\text{-}day)^{-1}$. The dose-response assessment generally entails an extrapolation from high doses administered to experimental animals to exposure levels expected from human contact with the site-related chemical in the environment. Inhalation and oral chronic slope factors

for the COPCs identified at the 11 sites are shown in Tables 6-5 and 6-6. Oral slope factors were used to evaluate dermal exposures if the type of cancer via ingestion exposure was not related to the gastrointestinal tract, including the liver.

6.1.3.2 *Noncarcinogens*

Reference doses (RfDs) or reference concentrations (RfCs) developed by the USEPA are estimates of the daily dose of a chemical to which humans, including sensitive subpopulations, can be exposed without an appreciable risk of deleterious effects during a lifetime. The RfD is generally expressed in units of milligrams per kilogram of bodyweight per day (mg/kg-day). The basis of an RfD is usually the highest level tested in animal experiments at which no adverse effects were demonstrated (i.e., NOAEL or No Observed Adverse Effect Level). The NOAEL is divided by uncertainty and modifying factors to obtain an RfD. Verified chronic inhalation and oral RfDs, which have been peer reviewed, are given in Tables 6-7 and 6-8. The RfC is generally expressed in units of milligrams per cubic meter of air (mg/m^3). The basis of an RfC is the same as an RfD, except that inhalation studies are the primary source of toxicological data.

The off-site child resident, the potential future on-site child resident, and the potential future construction worker were assumed to be subject to sub-chronic exposures (2 weeks to 7 years). Therefore, the noncarcinogenic hazards to these receptors were evaluated with the use of sub-chronic inhalation and oral RfDs. These values are listed in Tables 6-9 and 6-10.

Oral RfDs were used to evaluate dermal exposures if the critical effects via ingestion exposure were not related solely to the gastrointestinal tract, including the liver.

6.1.4 Risk Characterization

For each complete exposure pathway within each land use scenario identified for the nine sites within SWMU 13 and the two within SWMU 17, the CDI estimates described in Section 6.1.2.4 and the toxicity values described in Section 6.1.3 were used to quantify pathway risks for each COPC. Potential carcinogenic risk to each receptor population was determined by multiplying the lifetime averaged daily intake of a COPC (expressed in mg/kg-day) by the slope factor (expressed in $(mg/kg\text{-}day)^{-1}$). The slope factor converts estimated chronic daily intakes averaged over a lifetime of exposure to incremental risk of an individual developing cancer.

Potential noncarcinogenic chemical hazards were determined by calculating the ratio of a receptor's exposure level for a site chemical to its reference dose, which results in a hazard quotient. The sum of the hazard quotients indicates the overall hazard index associated with the exposure. The greater the index is above unity, the greater the level of concern.

Table 6-5. Chemicals of Potential Concern: Inhalation Toxicity Values for Potential Carcinogenic Effects

Contaminants	Inhalation Slope Factor $(mg/kg\text{-}day)^{-1}$	Weight of Evidence	Type of Cancer\ Target Organ\ Species	Source
Explosives				
1,3-Dinitrobenzene	NA	NA	NA	NA
2,4-Dinitrotoluene	NA	NA	NA	NA
2,6-Dinitrotoluene	NA	NA	NA	NA
Fluoroacetic Acid	NA	NA	NA	NA
HMX	NA	D	NA	IRIS
RDX	NA	C	NA	IRIS
Nitrobenzene	NA	B2	NA	HEAST
Tetryl	NA	NA	NA	NA
1,3,5-Trinitrobenzene	NA	NA	NA	NA
2,4,6-Trinitrotoluene	NA	C	NA	IRIS
Agent Breakdown Products				
Isopropyl Methyl Phosphonic Acid (IMPA)	NA	D	NA	IRIS
Semi-Volatile Organic Compounds				
Acenapthylene	NA	D	NA	IRIS
Acenaphthene	NA	NA	NA	NA
Anthracene	NA	D	NA	IRIS
Bromacil	NA	NA	NA	NA
Bis(2-ethylhexyl)phthalate	NA	B2	NA	IRIS
2-Chloronaphthalene	NA	NA	NA	NA
Dimethylnaphthalenes	NA	NA		NA
Fluorene	NA	D	NA	IRIS

Table 6-5. Chemicals of Potential Concern: Inhalation Toxicity Values for Potential Carcinogenic Effects (continued)

Contaminants	Inhalation Slope Factor $(mg/kg\text{-}day)^{-1}$	Weight of Evidence	Type of Cancer\ Target Organ\ Species	Source
Methylnaphthalenes	NA	NA	NA	NA
2-Methylnaphthalene	NA	NA	NA	NA
4-Methylphenol	NA	C	NA	IRIS
N-nitrosodiphenylamine	NA	B2	NA	IRIS
Naphthalene	NA	D	NA	IRIS
3-Nitrotoluene	NA	NA	NA	NA
Palmitic Acid	NA	NA	NA	NA
Phenanthrene	NA	D	NA	IRIS
TPH	NA	NA	NA	NA
Trimethylnaphthalenes	NA	NA	NA	NA
Volatile Organic Compounds				
Acetone	NA	NA	NA	NA
Benzene	2.9E-02	A	Respiratory/lungs/humans	IRIS
Bromodichloromethane	NA	B2	NA	IRIS
Chloroform	8.1E-02	B2	Carcinomas/liver/mouse	IRIS
Chloromethane	6.3E-03	C	Carcinomas/kidney/mouse	HEAST
Dibromochloromethane	NA	C	NA	IRIS
1,2-Dichlorobenzene	NA	D	NA	IRIS
1,4-Dichlorobenzene	NA	C	NA	IRIS
1,1-Dichloroethylene	1.8E-01	C	Adenocarcinomas/kidney/ mouse	IRIS
Ethylbenzene	NA	D	NA	IRIS

Table 6-5. Chemicals of Potential Concern: Inhalation Toxicity Values for Potential Carcinogenic Effects (continued)

Contaminants	Inhalation Slope Factor $(mg/kg\text{-}day)^{-1}$	Weight of Evidence	Type of Cancer\ Target Organ\ Species	Source
Methyl-n-butyl ketone/ 2-hexanone	NA	NA	NA	NA
Methylene chloride	1.60E-03	B2	Adenomas & carcinomas/ liver, lungs/mouse	IRIS
Methyl isobutyl ketone	NA	NA	NA	NA
Monochlorobenzene	NA	NA	NA	NA
1,1,2,2-Tetrachloroethane	2.0E-01[a]	C	Carcinomas/liver/mouse	IRIS
Toluene	NA	D	NA	IRIS
1,1,1-Trichloroethane	NA	D	NA	IRIS
1,1,2-Trichloroethane	5.7E-02	C	Carcinomas/liver/mouse	IRIS
Trichloroethylene	6.0E-03	B2	NA/liver/NA	NCEA[a]
Xylene (all isomers)	NA	D	NA	IRIS
Metals				
Arsenic	1.5E+01	A	NA	IRIS
Barium	NA	NA	NA	NA
Beryllium	8.4E+00	B2	Respiratory/lungs/rats	IRIS
Cadmium	6.3E+00	B1	Respiratory/lungs/humans	IRIS
Chromium[b]	4.1E+01	A	Cancer/lungs/humans	IRIS
Copper	NA	D	NA	IRIS
Lead	NA	B2	NA	IRIS
Mercury	NA	D	NA	IRIS
Nickel[c]	8.4E-01	A	Respiratory/lungs	IRIS
Selenium	NA	D	NA	IRIS

Table 6-5. Chemicals of Potential Concern: Inhalation Toxicity Values for Potential Carcinogenic Effects (continued)

Contaminants	Inhalation Slope Factor $(mg/kg\text{-}day)^{-1}$	Weight of Evidence	Type of Cancer\ Target Organ\ Species	Source
Silver	NA	D	NA	IRIS
Thallium	NA	NA	NA	NA
Uranium	NA	NA	NA	NA
Zinc	NA	D	NA	IRIS
Anions				
Bromide	NA	NA	NA	NA
Chloride	NA	NA	NA	NA
Fluoride	NA	NA	NA	NA
Nitrate	NA	NA	NA	NA
Sulfate	NA	NA	NA	NA

Note.—NA denotes not available.

[a]Values from NCEA (National Center for Environmental Assessment) are provisional.

[b]Values for chromium (VI) valence state.

[c]Value for nickel refinery dust.

Table 6-6. Chemicals of Potential Concern: Ingestion Toxicity Values for Potential Carcinogenic Effects

Contaminants	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$	Weight of Evidence	Type of Cancer\ Target Organ\ Species	Source
Explosives				
1,3-Dinitrobenzene	NA	NA	NA	NA
2,4-Dinitrotoluene	NA	NA	NA	NA
2,6-Dinitrotoluene[a]	6.8E-01	B2	Carcinomas/liver/rat	IRIS
Fluoroacetic Acid	NA	NA	NA	NA
HMX	NA	D	NA	IRIS
RDX	1.1E-01	C	Carcinomas/liver/mouse	IRIS
Nitrobenzene	NA	B2	NA	HEAST
Tetryl	NA	NA	NA	NA
1,3,5-Trinitrobenzene	NA	NA	NA	NA
2,4,6-Trinitrotoluene	3.0E-02	C	Transitional cell carcinomas/bladder/rat	IRIS
Agent Breakdown Products				
Isopropyl Methyl Phosphonic Acid (IMPA)	NA	D	NA	IRIS
Semi-Volatile Organic Compounds				
Acenaphthylene	NA	D	NA	IRIS
Acenaphthene	NA	NA	NA	NA
Anthracene	NA	D	NA	IRIS
Bromacil	NA	NA	NA	NA
Bis(2-ethylhexyl)phthalate	1.4E-02	B2	Carcinomas/liver/mouse	IRIS
2-Chloronaphthalene	NA	NA	NA	NA
Dimethylnaphthalenes	NA	NA	NA	NA
Fluorene	NA	D	NA	IRIS

Table 6-6. Chemicals of Potential Concern: Ingestion Toxicity Values for Potential Carcinogenic Effects (continued)

Contaminants	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$	Weight of Evidence	Type of Cancer\ Target Organ\ Species	Source
Methylnaphthalenes	NA	NA	NA	NA
2-Methylnaphthalenes	NA	NA	NA	NA
4-Methylphenol	NA	C	NA	IRIS
N-nitrosodiphenylamine	4.9E-03	B2	Transitional cell carcinomas/bladder/rat	IRIS
Naphthalene	NA	D	NA	IRIS
3-Nitrotoluene	NA	NA	NA	NA
Palmitic Acid	NA	NA	NA	NA
Phenanthrene	NA	D	NA	IRIS
TPHC	NA	NA	NA	NA
Trimethylnaphthalenes	NA	NA	NA	NA
Volatile Organic Compounds				
Acetone	NA	NA	NA	NA
Benzene	2.9E-02	A	Leukemia/NA/human	IRIS
Bromodichloromethane	6.2E-02	B2	Adenocarcinomas/kidney/mouse	IRIS
Chloroform	6.1E-03	B2	Carcinomas/kidney/rat	IRIS
Chloromethane	1.3E-02	C	Carcinomas/liver/mouse	HEAST
Dibromochloromethane	8.4E-02	C	Carcinomas/kidney/mouse	IRIS
1,2-Dichlorobenzene	NA	D	NA	IRIS
1,4-Dichlorobenzene	2.4E-02	C	Tumors/liver/mouse	HEAST
1,1-Dichloroethylene	6.0E-01	C	Pheochromocytomas/adrenals/rat	IRIS
1,2-Dimethylbenzene/O-Xylene	NA	NA	NA	NA
1,3-Dimethylbenzene/M-Xylene	NA	NA	NA	NA
Ethylbenzene	NA	D	NA	IRIS

Table 6-6. Chemicals of Potential Concern: Ingestion Toxicity Values for Potential Carcinogenic Effects (continued)

Contaminants	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$	Weight of Evidence	Type of Cancer\ Target Organ\ Species	Source
Methyl-n-butyl ketone/ 2-hexanone	NA	NA	NA	NA
Methylene chloride	7.5E-03	B2	Carcinomas/liver/mouse	IRIS
Methyl isobutyl ketone	NA	NA	NA	NA
Monochlorobenzene	NA	NA	NA	NA
1,1,2,2-Tetrachloroethane	2.0E-01	C	Carcinomas/liver/mouse	IRIS
Toluene	NA	D	NA	IRIS
1,1,1-Trichloroethane	NA	D	NA	IRIS
1,1,2-Trichloroethane	5.7E-02	C	Carcinomas/liver/mouse	IRIS
Trichloroethylene	1.1E-02	B2	NA/liver/NA	NCEA[b]
Xylene	NA	D	NA	IRIS/HEAST
Metals				
Arsenic	1.5E+00	A	NA/skin/human	IRIS
Barium	NA	NA	NA	IRIS/HEAST
Beryllium	4.3E+00	B2	Tumors/all/rat	IRIS
Chromium[c]	NA	A	NA	IRIS
Copper	NA	D	NA	IRIS
Lead	NA	B2	NA	IRIS
Mercury	NA	D	NA	IRIS
Nickel	NA	NA	NA	NA
Selenium	NA	D	NA	IRIS
Silver	NA	D	NA	IRIS
Thallium	NA	NA	NA	NA

Table 6-6. Chemicals of Potential Concern: Ingestion Toxicity Values for Potential Carcinogenic Effects (continued)

Contaminants	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$	Weight of Evidence	Type of Cancer\ Target Organ\ Species	Source
Uranium	NA	NA	NA	NA
Zinc	NA	D	NA	IRIS
Anions				
Bromide	NA	NA	NA	NA
Chloride	NA	NA	NA	NA
Fluoride	NA	NA	NA	NA
Nitrate	NA	NA	NA	NA
Sulfate	NA	NA	NA	NA

Note.—NA denotes not available.

[a]Value for 2,4/2,6-dinitrotoluene mixture.

[b]Values from NCEA (National Center for Environmental Assessment) are provisional.

[c]Chromium (VI) valence state.

Table 6-7. Chemicals of Potential Concern: Chronic Inhalation Toxicity Values for Potential Noncarcinogenic Effects

Contaminants	Chronic Inhalation RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Explosives					
1,3-Dinitrobenzene	NA	NA	NA	NA	NA
2,4-Dinitrotoluene	NA	NA	NA	NA	NA
2,6-Dinitrotoluene	NA	NA	NA	NA	NA
Fluoroacetic Acid	NA	NA	NA	NA	NA
HMX	NA	NA	NA	NA	NA
RDX	NA	NA	NA	NA	NA
Nitrobenzene	6.0E-04	NA	Hematological effects	10,000/1	HEAST, Table 2
Tetryl	NA	NA	NA	NA	IRIS/HEAST
1,3,5-Trinitrobenzene	NA	NA	NA	NA	NA
2,4,6-Trinitrotoluene	NA	NA	NA	NA	IRIS/HEAST
Agent Breakdown Products					
Isopropyl Methyl Phosphonic Acid (IMPA)	NA	NA	NA	NA	IRIS/HEAST
Semi-Volatile Organic Compounds					
Acenaphthylene	NA	NA	NA	NA	NA
Acenaphthene	NA	NA	NA	NA	NA
Anthracene	NA	NA	NA	NA	NA
Bromacil	NA	NA	NA	NA	NA
Bis(2-ethylhexyl)phthalate	2.9E-03[a]	NA	NA	NA	NCEA[b]
2-Chloronaphthalene	NA	NA	NA	NA	NA
Dimethylnaphthalenes	1.1E-04	NA	NA	NA	[c]

Table 6-7. Chemicals of Potential Concern: Chronic Inhalation Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Chronic Inhalation RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Fluorene	NA	NA	NA	NA	NA
Methylnaphthalenes	1.1E-04	NA	NA	NA	(c)
2-Methylnaphthalenes	NA	NA	NA	NA	(d)
4-Methylphenol	NA	NA	NA	NA	NA
N-nitrosodiphenylamine	NA	NA	NA	NA	NA
Naphthalene	1.1E-04[a]	NA	NA	NA	NCEA[b]
3-Nitrotoluene	NA	NA	NA	NA	NA
Palmitic Acid	NA	NA	NA	NA	NA
Phenanthrene	NA	NA	NA	NA	NA
TPH	NA	NA	NA	NA	NA
Trimethylnaphthalenes	1.1E-04	NA	NA	NA	(c)
Volatile Organic Compounds					
Acetone	NA	NA	NA	NA	NA
Benzene	1.73E-03	low	Hematological effects	1,000/1	NCEA[b]
Bromodichloromethane	NA	NA	NA	NA	NA
Chloroform	NA	NA	NA	NA	NA
Chloromethane	8.6E-02	medium	Neurological effects	30/10	NCEA[b]
Dibromochloromethane	NA	NA	NA	NA	NA
1,2-Dichlorobenzene	4.0E-02	NA	Decreased weight gain	1,000/1	HEAST, Table 2
1,4-Dichlorobenzene	2.3E-01	medium	Liver weight gain	100/1	IRIS
1,1-Dichloroethylene	NA	NA	NA	NA	NA
Ethylbenzene	2.9E-01	low	Developmental toxicity	300/1	IRIS

Table 6-7. Chemicals of Potential Concern: Chronic Inhalation Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Chronic Inhalation RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Methyl-n-butyl ketone/2-Hexanone	1.4E-03	low	Neurological effects	10,000/1	NCEA[b]
Methylene chloride	8.6E-01	NA	Liver toxicity	100/1	HEAST
Methyl isobutyl ketone	2.0E-02	NA	Liver and kidney effects	1,000/1	HEAST, Table 2
Monochlorobenzene	5.0E-03	NA	Liver and kidney effects	10,000/1	HEAST, Table 2
1,1,2,2-Tetrachloroethane	NA	NA	NA	NA	NA
Toluene	1.1E-01	medium	Neurologic effects	300/1	IRIS
1,1,1-Trichloroethane	2.9E-01	medium	Central nervous system effects	300/1	NCEA[b]
1,1,2-Trichloroethane	NA	NA	NA	NA	NA
Trichloroethylene	NA	NA	NA	NA	NA
Xylene	NA	NA	NA	NA	NA
Metals					
Arsenic	NA	NA	NA	NA	NA
Barium	1.0E-04	NA	Fetotoxicity	1,000/1	HEAST, Table 2
Beryllium	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA
Chromium[c]	1.1E-06[e]	NA	NA	NA	NA
Copper	NA	NA	NA	NA	NA
Lead	4.3E-04	NA	NA	NA	[f]
Mercury	8.6E-05	medium	Neurotoxicity	30/1	IRIS
Nickel	NA	NA	NA	NA	NA
Selenium	NA	NA	NA	NA	NA

Table 6-7. Chemicals of Potential Concern: Chronic Inhalation Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Chronic Inhalation RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Silver	NA	NA	NA	NA	NA
Thallium	NA	NA	NA	NA	NA
Uranium	NA	NA	NA	NA	NA
Zinc	NA	NA	NA	NA	NA
Anions					
Bromide	NA	NA	NA	NA	NA
Chloride	NA	NA	NA	NA	NA
Fluoride	NA	NA	NA	NA	NA
Nitrate	NA	NA	NA	NA	NA
Sulfate	NA	NA	NA	NA	NA

Note.—NA denotes not applicable or not available.

[a]No chronic inhalation RfD has been derived for this chemical. Sub-chronic value was used.

[b]Values from NCEA (National Center for Environmental Assessment) are provisional.

[c]Value for naphthalene.

[d]The USEPA specifically states not to use naphthalene values for 2-methylnaphthalene (ref. NCEA Issue Paper for 2-methylnaphthalene).

[e]Values for chromium (VI) valence state.

[f]Calculated from the federal air quality standard.

Table 6-8. Chemicals of Potential Concern: Ingestion Toxicity Values for Potential Noncarcinogenic Effects

Contaminants	Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Explosives					
1,3-Dinitrobenzene	1.0E-04	low	Increased splenic weight	3,000/1	IRIS
2,4-Dinitrotoluene	2.0E-03	high	Neurotoxicity	100/1	IRIS
2,6-Dinitrotoluene	1.0E-03	NA	Increased mortality	3,000/1	HEAST
Fluoroacetic Acid[a]	2.5E-05	low	Increased heart weight; altered spermatogenesis	3,000/1	IRIS
HMX	5.0E-02	low	Hepatic lesions	1,000/1	IRIS
RDX	3.0E-03	high	Inflammation of prostate	100/1	IRIS
Nitrobenzene	5.0E-04	low	Hematologic and hepatic lesions	10,000/1	IRIS
Tetryl	1.0E-02	NA	Liver, kidney, and spleen effects	10,000/1	HEAST
1,3,5-Trinitrobenzene	5.0E-05	low	Increase in splenic weight	10,000/1	IRIS
2,4,6-Trinitrotoluene	5.0E-04	medium	Liver effects	1,000/1	IRIS
Agent Breakdown Products					
Isopropyl Methtyl Phosphonic Acid (IMPA)	1.0E-01	low	No adverse effects observed	3,000/1	IRIS
Semi-Volatile Organic Compounds					
Acenaphthylene	3.0E-02	NA	NA	NA	[b]
Acenaphthene	6.0E-02	low	Hepatoxicity	3,000/1	IRIS
Anthracene	3.0E-01	low	No observed effects	3,000/1	IRIS
Bis(2-ethylhexyl)phthalate	2.0E-02	medium	Liver weight gain	1,000/1	IRIS
Bromacil	NA	NA	NA	NA	NA
2-Chloronaphthalene	8.0E-02	low	Dyspnea, abnormal appearance, liver enlargement	3,000/1	IRIS
Dimethylnaphthalenes	4.0E-02	NA	Not specified	NA	[c]

Table 6-8. Chemicals of Potential Concern: Ingestion Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Fluorene	4.0E-02	low	Decrease in RBC	3,000/1	IRIS
Methylnaphthalenes[d]	4.0E-02	NA	Not specified	NA	[c]
2-Methylnaphthalenes[d]	NA	NA	NA	NA	[d]
4-Methylphenol	5.0E-03	NA	Maternal death	1,000/1	HEAST
N-nitrosodiphenylamine	NA	NA	NA	NA	NA
Naphthalene	4.0E-02	NA	Not specified	1,000/1	NCEA[e]
3-Nitrotoluene	1.0E-02	NA	Spleen lesions	10,000/1	HEAST
Palmitic Acid	NA	NA	NA	NA	IRIS/HEAST
Phenanthrene	3.0E-02	NA	NA	NA	[b]
TPH	NA	NA	NA	NA	IRIS/HEAST
Trimethylnaphthalenes	4.0E-02	NA	Not specified	NA	[c]
Volatile Organic Compounds					
Acetone	1.0E-01	low	Increased liver and kidney weight; nephrotoxicity	1,000/1	IRIS
Benzene	NA	NA	NA	NA	NA
Bromodichloromethane	2.0E-02	medium	Renal cytomegaly	1,000/1	IRIS
Chloroform	1.0E-02	medium	Liver effects	1,000/1	IRIS
Chloromethane	NA	NA	NA	NA	NA
Dibromochloromethane	2.0E-02	medium	Hepatic lesions	1,000/1	IRIS
1,2-Dichlorobenzene	9.0E-02	low	No effects observed	1,000/1	IRIS
1,4-Dichlorobenzene	NA	NA	NA	NA	NA
1,1-Dichloroethylene	9.0E-03	medium	Hepatic lesions	1,000/1	IRIS
Ethylbenzene	1.0E-01	low	Liver and kidney toxicity	1,000/1	IRIS

Table 6-8. Chemicals of Potential Concern: Ingestion Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Methyl-n-butyl ketone/2-Hexanone	NA	NA	NA	NA	NA
Methylene chloride	6.0E-02	medium	Liver toxicity	100/1	IRIS
Methyl isobutyl ketone	8.0E-02	NA	Liver and kidney effects	3,000/1	HEAST
Monochlorobenzene	2.0E-02	medium	Histopathilogic changes in liver	1,000/1	IRIS
1,1,2,2-Tetrachloroethane	NA	NA	NA	NA	NA
Toluene	2.0E-01	medium	Changes in liver and kidney weight	1,000/1	IRIS
1,1,1-Trichloroethane	NA	NA	NA	NA	NA
1,1,2-Trichloroethane	4.0E-03	medium	Clinical serum chemistry	1,000/1	IRIS
Trichloroethylene	6.0E-03	low	Hepatotoxicity	3,000/1	NCEA[e]
Xylene	2.0E+00	medium	Hyperactivity, decrease in body weight, increase in mortality	100/1	IRIS
Metals					
Arsenic	3.0E-04	medium	Hyperpigmentation	3/1	IRIS
Barium	7.0E-02	medium	Increase in blood pressure	3/1	IRIS
Beryllium	5.0E-03	low	No adverse effects	100/1	IRIS
Cadmium[f]	5.0E-04	high	Significant proteinuria	10/1	IRIS
Chromium[g]	5.0E-03	low	No effects reported	500/1	IRIS
Copper	3.7E-02	NA	Gastrointestinal irritation	NA	HEAST
Lead	4.3E-04	NA	NA	NA	[h]
Mercury	3.0E-04	NA	Kidney effects	1,000/1	HEAST
Nickel	2.0E-02	medium	Decrease in body and organ weight	300/1	IRIS
Selenium	5.0E-03	high	Clinical selenosis	3/1	IRIS
Silver	5.0E-03	low	Argyria	3/1	IRIS

Table 6-8. Chemicals of Potential Concern: Ingestion Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Thallium[i]	8.0E-05	low	Increased level of SG02 and LDH	3000/1	IRIS
Uranium[j]	3.0E-03	medium	Decreased body weight; moderate nephrotoxicity	1,000/1	IRIS
Zinc	3.0E-01	medium	Decrease in erythrocyte	3/1	IRIS
Anions					
Bromide	NA	NA	NA	NA	NA
Chloride	NA	NA	NA	NA	NA
Fluoride[k]	1.2E-01	NA	Skeletal fluorosis	NA/NA	IRIS
Nitrate[l]	7.0E+00	high	Clinical signs of methemoglobinemia	1/1	IRIS
Sulfate	NA	NA	NA	NA	NA

Note.—NA denotes not applicable or not available.

[a]Value for sodium fluoroacetate; corrected for molecular weight differences.

[b]Value for pyrene (from IRIS).

[c]Value for naphthalene used.

[d]EPA specifically states that naphthalene value should not be used for d-methylnaphthalene.

[e]NCEA (National Center for Environmental Assessment) values are provisional.

[f]Value for cadmium intake through water.

[g]Values for chromium (VI).

[h]Derived from action level in drinking water.

[i]Value for the thallium oxide.

[j]Value for uranium salts.

[k]The chronic oral RfD for fluoride is designed to protect against dental fluorosis in children, the sensitive population for this effect. IRIS also provides a safe exposure level for adults to protect against a more severe endpoint, skeletal fluorosis.

[l]IRIS value, expressed as amount of nitrogen in nitrate molecule, was modified to derive value for nitrate.

Table 6-9. Chemicals of Potential Concern: Sub-chronic Inhalation Toxicity Values for Potential Noncarcinogenic Effects

Contaminants	Sub-Chronic Inhalation RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Explosives					
1,3-Dinitrobenzene	NA	NA	NA	NA	NA
2,4-Dinitrotoluene	NA	NA	NA	NA	NA
2,6-Dinitrotoluene	NA	NA	NA	NA	NA
Fluoroacetic Acid	NA	NA	NA	NA	NA
HMX	NA	NA	NA	NA	NA
RDX	NA	NA	NA	NA	NA
Nitrobenzene	6.0E-03	NA	Hematological effects	1,000/1	HEAST, Table 2
Tetryl	NA	NA	NA	NA	NA
1,3,5-Trinitrobenzene	NA	NA	NA	NA	NA
2,4,6-Trinitrotoluene	NA	NA	NA	NA	NA
Agent Breakdown Products					
Isopropyl Methyl Phosphonic Acid (IMPA)	NA	NA	NA	NA	NA
Semi-Volatile Organic Compounds					
Acenaphthylene	NA	NA	NA	NA	NA
Acenaphthene	NA	NA	NA	NA	NA
Anthracene	NA	NA	NA	NA	NA
Bromacil	NA	NA	NA	NA	NA
Bis(2-ethylhexyl)phthalate	2.9E-03	Medium to low	Pulmonary effects	1,000/1	NCEA[a]
2-Chloronaphthalene	NA	NA	NA	NA	NA
Dimethylnaphthalenes	1.1E-04	NA	NA	NA	[b]
Fluorene	NA	NA	NA	NA	NA
Methylnaphthalenes	1.1E-04	NA	NA	NA	[b]

Table 6-9. Chemicals of Potential Concern: Sub-chronic Inhalation Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Sub-Chronic Inhalation RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
2-Methylnaphthalenes	NA	NA	NA	NA	(c)
4-Methylphenol	NA	NA	NA	NA	NA
N-nitrosodiphenylamine	NA	NA	NA	NA	NA
Naphthalene	1.1E-04[a]	NA	Nasal effects	3,000/1	NCEA[e]
3-Nitrotoluene	NA	NA	NA	NA	NA
Palmitic Acid	NA	NA	NA	NA	NA
Phenanthrene	NA	NA	NA	NA	NA
TPH	NA	NA	NA	NA	IRIS/HEAST
Trimethylnaphthalenes	1.1E-04	NA	NA	NA	(c)
Volatile Organic Compounds					
Acetone	NA	NA	NA	NA	NA
Benzene	1.7E-02	low	Hematological effects	100/1	NCEA[a]
Bromodichloromethane	NA	NA	NA	NA	NA
Chloroform	NA	NA	NA	NA	NA
Chloromethane	8.6E-02	medium	Neurological effects	30/10	NCEA[a]
Dibromochloromethane	NA	NA	NA	NA	NA
1,2-Dichlorobenzene	4.0E-01	NA	Decreased weight gain	100/1	HEAST, Table 2
1,4-Dichlorobenzene	7.1E-01	NA	Increased liver weight	30/1	HEAST
1,1-Dichloroethylene	NA	NA	NA	NA	NA
Ethylbenzene	2.9E-01	low	Developmental toxicity	300/1	NCEA
Methyl-n-butyl ketone/2-Hexanone	1.4E-03[d]	NA	NA	NA	NCEA[a]
Methylene chloride	8.6E-01	NA	Liver toxicity	100/1	HEAST
Methyl isobutyl ketone	2.0E-01	NA	Liver and kidney effects	100/1	HEAST, Table 2

Table 6-9. Chemicals of Potential Concern: Sub-chronic Inhalation Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Sub-Chronic Inhalation RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Monochlorobenzene	5.0E-02	low	Liver and kidney effects	300/1	NCEA[a]
1,1,2,2-Tetrachloroethane	NA	NA	NA	NA	NA
Toluene	1.1E-01	medium	Neurological effects	300/1	NCEA[a]
1,1,1-Trichloroethane	2.9E-01[d]	NA	NA	NA	NCEA[a]
1,1,2-Trichloroethane	NA	NA	NA	NA	NA
Trichloroethylene	NA	NA	NA	NA	NA
Xylene	NA	NA	NA	NA	NA
Metals					
Arsenic	NA	NA	NA	NA	NA
Barium	1.0E-03	NA	Fetotoxicity	100/1	HEAST, Table 2
Beryllium	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA
Chromium[c]	1.1E-06[a]	NA	Diffuse nasal symptoms	NA	NA
Copper	NA	NA	NA	NA	NA
Lead	4.3E-04[d]	NA	NA	NA	[f]
Mercury	8.6E-05	NA	Neurotoxicity	30/1	HEAST
Nickel	NA	NA	NA	NA	NA
Selenium	NA	NA	NA	NA	NA
Silver	NA	NA	NA	NA	NA
Thallium	NA	NA	NA	NA	NA
Uranium	NA	NA	NA	NA	NA
Zinc	NA	NA	NA	NA	NA

Table 6-9. Chemicals of Potential Concern: Sub-chronic Inhalation Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Sub-Chronic Inhalation RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Anions					
Bromide	NA	NA	NA	NA	NA
Chloride	NA	NA	NA	NA	NA
Fluoride	NA	NA	NA	NA	NA
Nitrate	NA	NA	NA	NA	NA
Sulfate	NA	NA	NA	NA	IRIS/HEAST

Note.—NA denotes not applicable or available.
[a]Values from NCEA (National Center for Environmental Assessment) are provisional.
[b]Value for naphthalene.
[c]The EPA specifically states not to use naphthalene values for 2-methylnaphthalene.
[d]Chronic value used since a subchronic value has not been established.
[e]Value for chromium (VI) isomer.
[f]Calculated from federal air quality standard.

Table 6-10. Chemicals of Potential Concern: Sub-chronic Ingestion Toxicity Values for Potential Noncarcinogenic Effects

Contaminants	Sub-Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Explosives					
1,3-Dinitrobenzene	1.0E-03	NA	Spleen changes	100/1	HEAST
2,4-Dinitrotoluene	2.0E-03	NA	Neurotoxicity	100/1	HEAST
2,6-Dinitrotoluene	1.0E-02	NA	Increased mortality	300/1	HEAST
Fluoroacetic Acid[a]	2.5E-05[b]	NA	NA	NA	IRIS
HMX	5.0E-02[b]	NA	NA	NA	IRIS
RDX	3.0E-03	NA	Prostrate inflammation	100/1	HEAST
Nitrobenzene	5.0E-03	NA	Hematological effects	1,000/1	HEAST
Tetryl	1.0E-01	NA	Liver, kidney, and spleen effects	1,000/1	HEAST
1,3,5-Trinitrobenzene	5.0E-04	NA	Increased spleen weights	1,000/1	HEAST
2,4,6-Trinitrotoluene	5.0E-04	NA	Liver effects	1,000/1	HEAST
Agent Breakdown Products					
Isopropyl Methyl Phosphonic Acid (IMPA)	1.0E-01[b]	NA	None	3,000/1	IRIS
Semi-Volatile Organic Compounds					
Acenaphthylene	3.0E-01	NA	NA	NA	[c]
Acenaphthene	6.0E-01	NA	Hepatotoxicity	300/1	HEAST
Anthracene	3.0E+00	NA	None observed	300/1	HEAST
Bromacil	NA	NA	NA	NA	IRIS/HEAST
Bis(2-ethylhexyl)phthalate	2.0E-02	Medium	Reproductive toxicity	3,000/1	NCEA [f]
2-Chloronaphthalene	8.0E-02[b]	NA	NA	NA	IRIS
Dimethylnaphthalenes	4.0E-02[a]	NA	NA	NA	[d]
Fluorene	4.0E-01	NA	Decreased erythrocytes	300/1	HEAST

Table 6-10. Chemicals of Potential Concern: Sub-chronic Ingestion Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Sub-Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Methylnaphthalenes	4.0E-02	NA	NA	NA	[c]
2-Methylnaphthalenes	NA	NA	NA	NA	NA
4-Methylphenol	5.0E-03	NA	Maternal death	1,000/1	HEAST
N-nitrosodiphenylmine	NA	NA	NA	NA	IRIS/HEAST
Naphthalene	4.0E-02[b]	NA	NA	NA	NCEA[f]
3-Nitrotoluene	1.0E-01	NA	Spleen lesions	1,000/1	HEAST
Palmitic Acid	NA	NA	NA	NA	IRIS/HEAST
Phenanthrene	3.0E-01[a]	NA	NA	NA	[c]
TPH	NA	NA	NA	NA	NA
Trimethylnaphthalenes	4.0E-02[b]	NA	NA	NA	[d]
Volatile Organic Compounds					
Acetone	1.0E+00	NA	Hepatoxicity	300/1	HEAST
Benzene	NA	NA	NA	NA	NA
Bromodichloromethane	2.0E-02	NA	Kidney effects	1,000/1	HEAST
Chloroform	1.0E-02	NA	Liver lesions	1,000/1	HEAST
Chloromethane	NA	NA	NA	NA	NA
Dibromochloromethane	2.0E-01	NA	Liver lesions	100/1	HEAST
1,2-Dichlorobenzene	9.0E-02[b]	NA	NA	NA	IRIS
1,4-Dichlorobenzene	NA	NA	NA	NA	NA
1,1-Dichloroethylene	9.0E-03	NA	Liver lesions	1,000/1	HEAST
Ethylbenzene	1.0E-01	low	Liver and kidney lesions	1,000/1	NCEA[f]
Methyl-n-butyl ketone (2-Hexanone)	NA	NA	NA	NA	NA

Table 6-10. Chemicals of Potential Concern: Sub-chronic Ingestion Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Sub-Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Methylene chloride	6.0E-02	NA	Liver toxicity	100/1	HEAST
Methyl isobutyl ketone	8.0E-01	NA	Liver and kidney effects	300/1	HEAST
Monochlorobenzene	2.0E-02[b]	NA	NA	NA	IRIS
1,1,2,2-Tetrachloroethane	NA	NA	NA	NA	NA
Toluene	2.0E+00	NA	Altered kidney and liver weight	100/1	HEAST
1,1,1-Trichloroethane	NA	NA	NA	NA	NA
1,1,2-Trichloroethane	4.0E-02	NA	Clinical chemistry alterations	100/1	HEAST
Trichloroethylene	6.0E-03[b]	NA	NA	NA	NCEA[f]
Xylene	2.0E+00[b]	NA	NA	NA	IRIS
Metals					
Arsenic	3.0E-04	NA	Keratosis; hyperpigmentation	3/1	HEAST
Barium	7.0E-02	NA	Increased blood pressure	3/1	HEAST
Beryllium	5.0E-03	NA	None observed	100/1	HEAST
Cadmium	5.0E-04[b]	NA	NA	NA	IRIS
Chromium[g]	2.0E-02	NA	No effects observed	100/1	HEAST
Copper	3.7E-02	NA	Gastrointestinal irritation	NA	HEAST
Lead	4.3E-04[b]	NA	NA	NA	[h]
Mercury	3.0E-03	NA	Autoimmune effects	1,000/1	HEAST
Nickel	2.0E-02	NA	Decreased organ and body weight	300/1	HEAST
Selenium	5.0E-03	NA	Clinical selenosis	3/1	HEAST
Silver	5.0E-03	NA	Argyria	3/1	HEAST
Thallium[i]	8.0E-04	NA	Increased SGOT and LDH	300/1	HEAST
Uranium[j]	3.0E-03[b]	NA	NA	NA	IRIS

Table 6-10. Chemicals of Potential Concern: Sub-chronic Ingestion Toxicity Values for Potential Noncarcinogenic Effects (continued)

Contaminants	Sub-Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Uncertainty/ Modifying Factor	Source
Zinc	3.0E-01	NA	Decrease blood enzymes	3/1	HEAST
Anions					
Bromide	NA	NA	NA	NA	IRIS/HEAST
Chloride	NA	NA	NA	NA	IRIS/HEAST
Fluoride	6.0E-02[k]	NA	Objectional dental fluorosis	1/1	HEAST
Nitrate	7.0E+00[b]	NA	NA	NA	IRIS
Sulfate	NA	NA	NA	NA	IRIS/HEAST

Note.—NA denotes not applicable or available.
[a]Value for sodium fluoroacetate, adjusted to account for differences in molecular weight.
[b]Chronic RfD used since a subchronic RfD has not been established.
[c]Value for pyrene (from HEAST).
[d]Value for naphthalene (from NCEA).
[e]The EPA specifically states that naphthalene value should not be used for 2-methylnaphthalene (ref. NCEA Issue Paper).
[f]Values from NCEA (National Center for Environmental Assessment) are provisional.
[g]Value for chromium (VI) isomer.
[h]Calculated from action level in drinking water.
[i]Value for thallium sulfate.
[j]Value for uranium salts.

It should be noted that the total cancer risk and noncarcinogenic hazard to the current on-site worker at SWMU 13 and SWMU 17 is the summation of all the contributions evaluated at each site within each SWMU. For the off-site resident, the total carcinogenic risk and noncarcinogenic hazard from inhalation of volatiles and fugitive dust emissions are the summation of the contributions from all sites within both SWMUs 13 and 17. The risk calculations for each site are presented in Appendix H and are summarized in the tables and text of Sections 7.0 and 8.0 of this report.

6.1.5 Uncertainty Analysis

There are several categories of uncertainties associated with site risk assessments, including contaminant selection and concentration, exposure assessment, and sources of uncertainty inherent in the toxicity values used to characterize risk. The estimates of human-health risks developed for this risk assessment required a number of assumptions concerning exposure assessment. These are discussed below along with other uncertainties that could affect the numerical risk estimates.

6.1.5.1 *Chemicals of Potential Concern*

Organic COPCs consist of compounds that exceeded the certified reporting limit and were not found in corresponding field blanks, trip blanks, or laboratory QC samples. Tentatively identified compounds (TICs) that could not be identified by the laboratory are discussed in Section 7.0 on a SWMU-specific basis.

Metal COPCs were established by screening site data against background levels. Although background samples were taken from several soil types, the soil types were not delineated according to soil type or depth. Naturally occurring levels of metals and anions could exhibit significant variability according to soil type and depth, resulting in a potential for a higher or lower exposure estimate. Indeed, some of these naturally occurring levels of arsenic are driving the risk at many of the sites at SWMUs 13 and 17.

6.1.5.2 *Exposure Pathways*

Although this human health risk assessment did not assess every conceivable exposure scenario possible, all exposure pathways were evaluated according to risk contribution. Since total risk to human health is a sum of all complete pathways known to exist, the lack of quantification of less significant pathways may underestimate the risk. However, those pathways not quantified represent small sources of exposure and are not expected to influence risk management decisions.

A potential exposure pathway that was not quantitatively evaluated at this time is the ingestion of meat from wildlife grazing in contaminated areas of TEAD-S. The possibility

of this type of exposure is acknowledged, but the risk contribution was deemed to be negligible since the ingestion of contaminants via the beef pathway utilized 95th percentile ingestion rates of meat. This would compensate for other meat sources such as venison, if they are occurring. It should be noted that hunting is not allowed on the facility.

Exposure by on-site workers was evaluated based on the assumption that a single worker at each SWMU spends equal time at each site within that SWMU during the course of a work day. The worker may actually spend less time, if any, and thus the exposure time may be significantly lower than the values used in the human health risk assessment. Therefore, it is likely that the risk calculations represent an overestimate of the actual risk.

Plant and animal uptake factors for site-related chemicals were estimated using values reported in the literature. Cattle grazing patterns were estimated in terms of the amount of dry feed ingested from contaminated areas. These assumptions could result in either increased or decreased exposure estimates.

6.1.5.3 *Contaminant Fate and Transport*

The evaluation of human health risks assumed that environmental media concentrations determined from sampling will remain at the same levels over the assumed periods of exposure. This assumption is likely to result in an overestimation of risk, since concentrations, especially of organic contaminants, are expected to decline over the long-term as natural fate and transport processes degrade, dilute, or remove site contaminants. The rate of the degradation, removal, and/or dilution of chemicals in soil, groundwater, surface water, and sediment are not known; therefore, the magnitude of the overestimate is difficult to determine.

6.1.5.4 *Exposure Point Concentrations*

The exposure point concentrations used for assessing risks associated with the RME case were either the maximum detected value or the upper 95th UCL of the mean value (whichever was less). Nondetected values were treated as concentrations equal to one-half their detection limit. This procedure could underestimate or overestimate the risk depending on the actual concentrations (if present) of the chemical below the detection limits. This uncertainty is most likely when estimating concentrations for those chemicals detected infrequently.

6.1.5.5 *Exposure Levels*

The amount of exposure that an individual receives is highly dependent on the details related to their human-activity patterns. There is considerable variability regarding the values assumed in calculating human intake factors. For instance, estimates of soil ingestion rates

for all populations are subject to ongoing debate. This may again result in overestimating or underestimating the risk on an individual basis. Additionally, exposure levels estimated for this project did not take into account the fact that individuals such as on-site workers and construction workers would be required to wear personal protective equipment when working in contaminated areas. This results in an overestimate of risk for these potential receptors.

6.1.5.6 *Toxicity Values*

Quantification of risk from exposure to a chemical cannot be accomplished in the absence of reliable, appropriate toxicity values (reference doses, slope factors) for all routes and exposure periods. For the COPCs at SWMUs 13 and 17, toxicity values are not available for some chemicals by some exposure routes. In addition, oral absorption efficiencies were estimated to adjust oral toxicity values from an administered dose to an absorbed dose for the evaluation of dermal exposure to groundwater. This adds considerable uncertainty to the numerical risk values associated with the sites, but it is not possible to estimate the degree of this uncertainty.

6.1.5.7 *Cancer-Risk Estimates*

The predicted cancer risk due to chemical exposure is often based on cancer-dose response data in animals. There is a long-standing controversy in the scientific community as to the best way animal data should be extrapolated to humans. In general, the USEPA follows a conservative procedure in the derivation of slope factors, so cancer risk estimates based on these values could be considerably higher than the actual risk.

The cancer risks calculated for children are less certain than those calculated for adults. The method utilized in this risk assessment assumes cancer risks are simply proportional to total dose. Actual cancer risk to a child only exposed during childhood could be higher or lower depending on the detailed mechanism of carcinogenicity for each chemical.

6.1.5.8 *Multiple Chemical Exposure*

The risk assessment approach assumes that health risks from multiple chemicals are additive, ignoring both synergistic and antagonistic effects among chemicals. Because of the number of chemicals evaluated at these sites, it is difficult to determine if additivity is a major source of error.

6.1.5.9 *Summary of Uncertainties*

In summary, the estimation of exposure and risk are subject to a number of uncertainties that may lead to either an overestimate or underestimate of risk.

Assumptions made in this risk assessment that are likely to overestimate risk include:

- Exposure point concentrations used were the 95 percent UCL of the mean or the maximum detected value, whichever was lower.
- Environmental media concentrations are unchanged over time.
- All ingested soil comes from the contaminated source.
- Soil is ingested at the assumed rate for all populations.
- Human activity patterns and the resultant exposure patterns used to calculate a reasonable maximum exposure are assumed.
- Slope factors are equal to the 95 percent confidence limit of the best estimate of the slope of the dose-response curve.
- One-half the detection limit was used in calculating exposure point concentrations for samples where a specific chemical was not detected.
- Grazing cattle were assumed to have access to all sites at TEAD-S, thus leading to potentially overestimating the risk to future on-site residents.

Factors in this risk assessment that are likely to underestimate risk include:

- Not all exposure pathways for all chemicals were quantified.
- Toxicity values are not available for every chemical, for every exposure duration, or for all exposure routes.
- Risks from all tentatively identified compounds were not quantitatively evaluated.

Factors in which the direction of uncertainty cannot be determined or is unknown include:

- High detection limits exhibited for some analytes due to matrix interference effects.
- Analytical variations in chemical analyses.
- Lack of information on the interactions among the multiple chemicals contributing to cancer and noncancer risks.
- Assumption of potential future populations at the 11 release units within SWMUs 13 and 17.
- Method for quantifying less than lifetime exposures to carcinogens in childhood.

6.2 ECOLOGICAL RISK ASSESSMENT

Ecological assessments evaluate the likelihood that adverse ecological effects will occur as a result of exposure to stressors resulting from human activity. These stressors can be either chemical, physical, or biological in nature. Ecological effects can range from subtle alterations in physiological function in individuals in a specific plant or wildlife population, to the complete or partial loss of ecosystem function (Norton et al. 1992).

The scope of this ecological risk assessment is limited to the characterization of the TEAD-S environment and the evaluation of the potential risk to biota that may result from the release of hazardous substances at SWMUs 13 and 17. The scope of the ecological risk assessment does not include remedial-action impacts on the environment.

The objectives of this risk assessment are (1) to characterize environmental resources of each SWMU, (2) to identify potential impacts related to releases of contaminants at each SWMU, and (3) to determine if these contaminants have the potential to adversely affect the entire TEAD-S facility ecosystem. The first objective was met by reviewing existing data and literature. The second objective was met by evaluating exposure to toxicity criteria for each SWMU. The last objective was met by considering results of the SWMU analyses site-wide. No site-specific wildlife or vegetation samples were collected for analysis as part of this risk assessment.

6.2.1 Problem Formulation

This section serves to characterize the site and to focus efforts on areas and receptors most at risk. Information for the soil, vegetation, and wildlife at the TEAD-S facility was reviewed for development of the potential exposure pathways and selection of the ecological endpoints and key ecological receptors. A food web diagram (Figure 6-1) was developed with the information obtained in the following surveys.

6.2.1.1 *Site Characterization*

6.2.1.1.1 ***Soil Survey***. As described in Section 3.5.3, a general soils survey was conducted for TEAD-S. The purpose of this survey was to prepare a map of the soils of the facility area and to qualitatively describe the mapping units. The basis and primary source of information for the soil survey of TEAD-S was the SCS soil survey report for Tooele County (SCS 1992). Supplemental literature included Weston (1991) and Welsh and others (1987). These data sources were used to collect data and information obtained during the preparation of this report.

The SCS office in Salt Lake City, Utah, was contacted in order to coordinate the nomenclature used for soils in the TEAD-S area with the National Cooperative Soils Survey. Established or proposed soil series for this area were used. Soils mapping for this report entailed refining the soil delineations previously established by the SCS survey.

6.2.1.1.2 ***Vegetation Survey***. As described in Section 3.5.4, a general vegetation survey of the TEAD-S facility area was conducted, and a vegetation map was prepared. Information from the SCS soil-mapping unit, range site descriptions, aerial photography, selected literature, and field reconnaissance was combined to develop the range site descriptions and mapping unit delineations used in this report.

The plant-species list for the facility area was obtained by a reconnaissance survey in which plant species observed within the area were recorded. Plant species that could not be identified in the field were collected for later identification. Botanical nomenclature used follows Welsh (1987) and Weber (1987). Some plants lacking complete structures needed

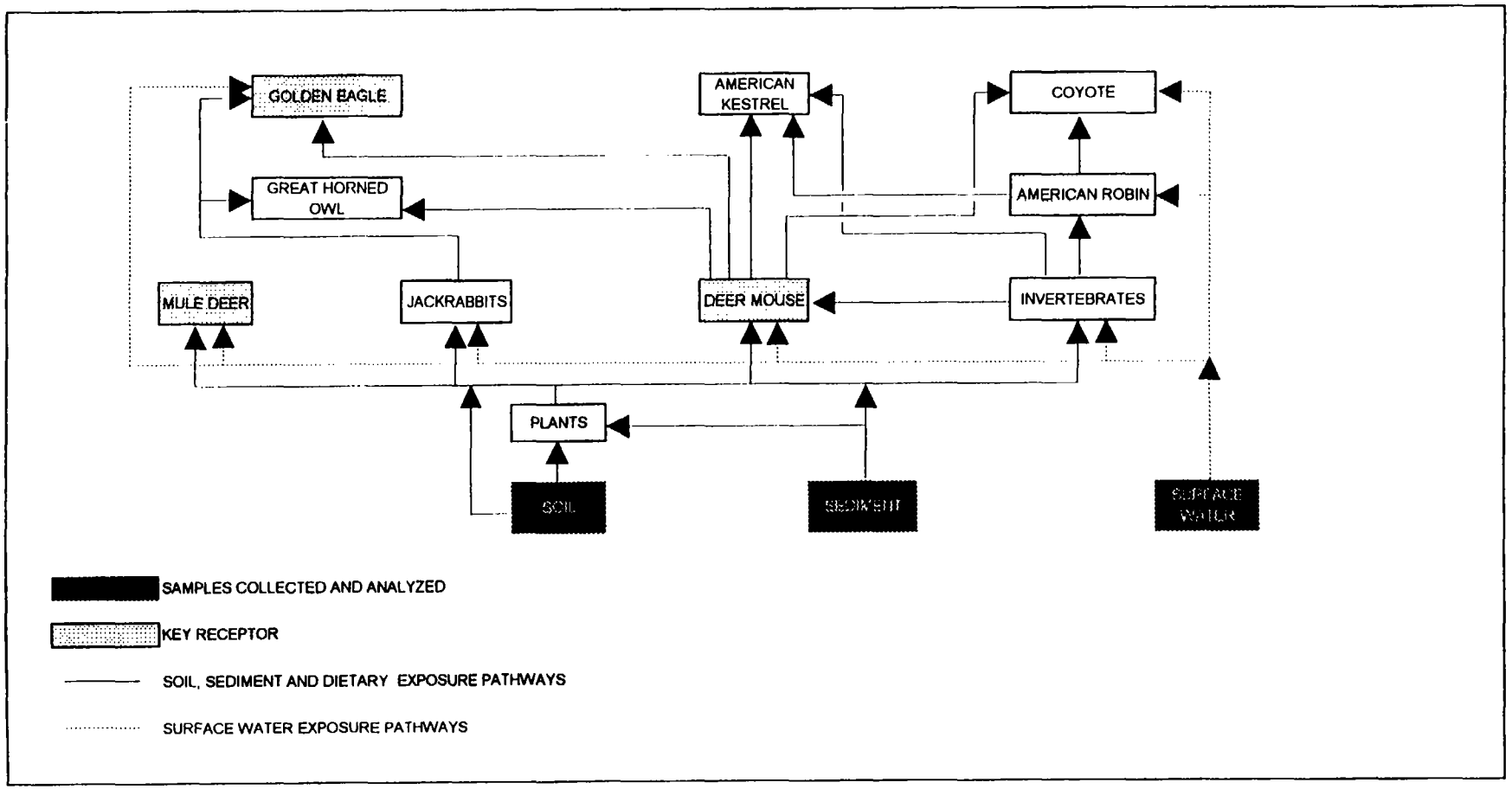


Figure 6-1. Food Web Diagram for TEAD-S

for field identification were collected and sent to Brigham Young University in Provo, Utah, and to Western State College in Gunnison, Colorado, for identification and verification.

6.2.1.1.3 *Wildlife Survey*. As described in Section 3.5.5, a general wildlife survey of the TEAD-S facility area was conducted. Table 3-8 also contains notations for actual observations by species identified on site during the field investigation. Wildlife species of concern were selected from lists prepared by the Army, by the State of Utah, and from known and potential endangered species, candidate species, migrating species, and state-sensitive species. Game species were also included. Information for this report was obtained from selected literature sources, field and road reconnaissance, and consultations with the U.S. Fish and Wildlife Service and Bureau of Land Management.

6.2.1.2 *Selection of Chemicals of Potential Concern*

One of the primary steps in the problem formulation is to identify the COPCs that are relevant to the individuals or ecosystems being investigated. This process takes into account not only the concentrations and toxicology of the COPCs present in various environmental media (i.e., soil, water, air), but also their mobility, persistence, bioaccumulation, and biomagnification potential.

A phased screening approach was used to identify the COPCs in the on-site media that had the potential of adversely affecting the selected ecological receptors. The first phase of the screening process involved examining detection limits, detection frequencies, and blank samples. The second phase included comparing concentrations to background samples. After this initial phased screening, COPCs that would be investigated for both the human health and the ecological risk assessments were selected. The COPCs selected for the human health risk assessment were derived from data for soil samples taken up to depths of 10 feet and from surface water samples. These were then used as the initial list for the ecological assessment. This list was further screened to only those found in the top 12 inches of soil plus surface water. Further screening was necessary through literature reviews and data searches to eliminate any of the selected COPCs that have no adverse effects or no documented effects on the biota. Some COPCs that were eliminated from the human health risk assessment because no effects data are available for human health, were included in the ecological assessment because effects data are available for biota. The final list of COPCs that were used for the ecological assessment is presented in Table 6-11. The behavior of the COPCs in soil, water, air, and biological systems can be found in Table 6-2.

6.2.1.3 *Selection of Key Ecological Receptors*

Ecological receptors are species representative to an ecosystem that either have been or have the potential to be exposed to COPCs. These key ecological receptors were selected by considering all potential exposure pathways and species (observed and potential) present at

Table 6-11. Ecological Chemicals of Potential Concern

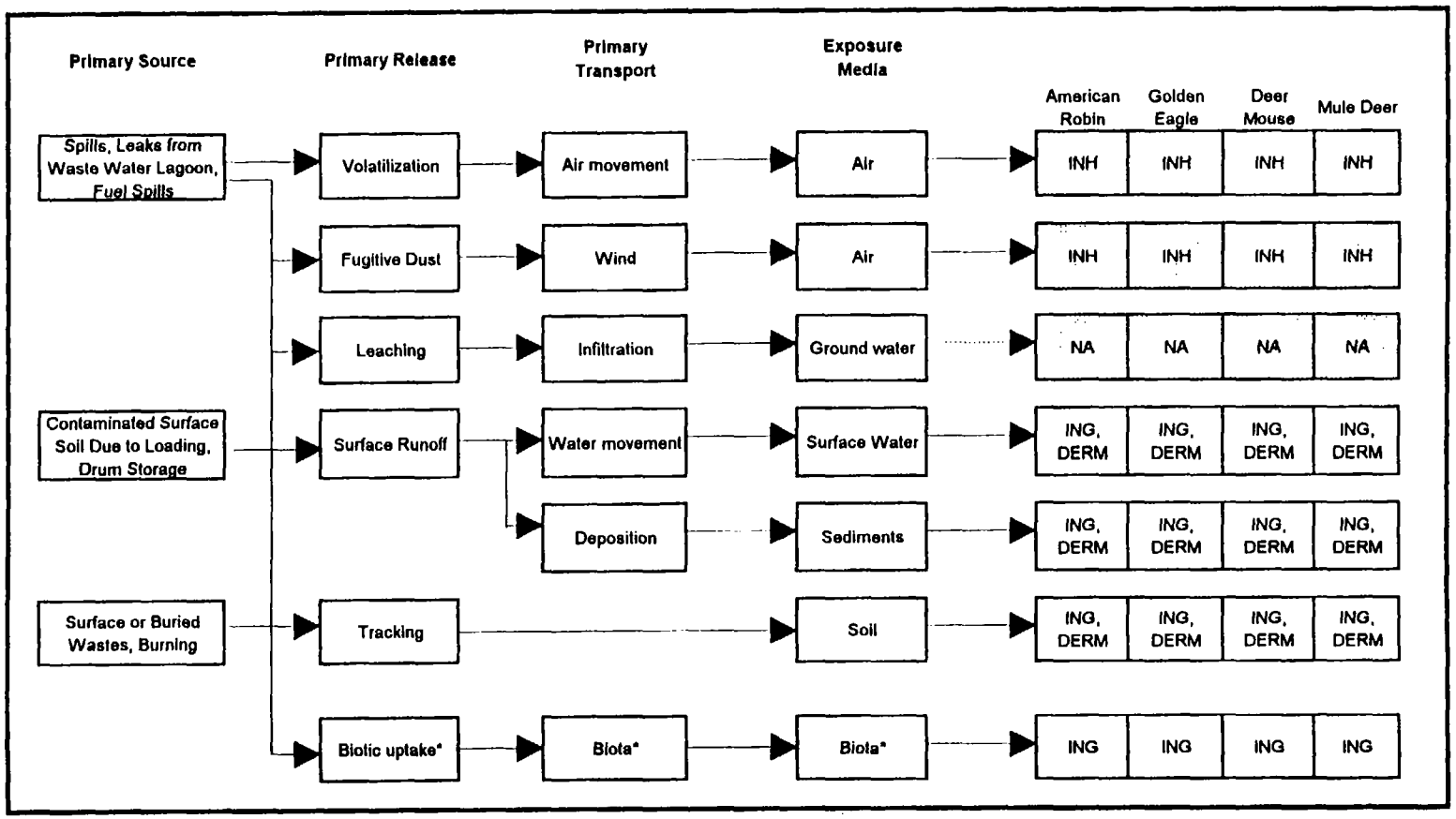
Chemical		
Metals	**SVOCs**	**Anions**
Arsenic	4-Methylphenol(p-cresol)	Chloride
Beryllium	Bis(2 ethylhexyl) phthalate	Fluoride
Chromium	Palmitic acid	Nitrate
Copper	**VOCs**	Nitrite
Lead	1,2-Dimethylbenzene (o-xylene)	Sulfate
Mercury	Acetone	**Explosives**
Nickel	Chloroform	2,6-Dinitrotoluene
Uranium	Chloromethane	HMX
Zinc	Methlylene chloride	**TPHC**
	Toluene	Total petroleum hydrocarbons (TPHC)

the SWMU being investigated. The conceptual site model (Figure 6-2) was used to depict potential routes of COPCs from the soil to the selected key ecological receptor. Ecological COPCs are listed in Table 6-11 above.

As part of the development of the food chains and food web, the following criteria were used in the selection of key ecological receptors:

- Species important to community structure or function.
- Species must be toxicologically sensitive to any or all of the COPCs.
- Species can or does occur at the site.
- Toxicological data for key species or related surrogates must be available in scientific literature.
- Threatened, endangered, and sensitive or candidate species hold a priority consideration for selection.

Wildlife species present at the TEAD-S facility used to select key receptor species are listed in Table 6-12.



▭	Insignificant pathway; not quantitatively evaluated
NA	Not Applicable
ING	Ingestion
INH	Inhalation
DERM	Dermal contact
.....	No exposure point
*	See Figure 6-12 (food web)

Figure 6-2. TEAD-S Conceptual Site Model

Table 6-12. Potential Ecological Receptor Species

Taxa	Species
Carnivores	Coyote
Small Herbivores	Northern pocket gopher
	Merriam's shrew
	Pinyon mouse
	Deer mouse
	Ord's kangaroo rat
Medium Herbivores	Black-tailed jackrabbit
	Nuttall's cottontail
Large Herbivores	**Mule deer**
	Pronghorn antelope
Passerine (Perching) Birds	**American robin**
	Western meadowlark
Raptors	Burrowing owl
	Great-horned owl
	American kestrel
	Bald eagle
	Golden eagle
	Red-tailed hawk
	Ferruginous hawk

Note.—Key species used in the ecological evaluation are highlighted.

6.2.1.4 *Selection of Ecological Endpoints*

Assessment endpoints are expressions of an environmental value deemed worthy of protection (e.g., threatened and endangered species, sensitive habitat, game animals) (Suter 1993). They represent the ultimate focus of the risk characterization.

In order to identify adverse impacts on the biota, the following factors were considered in the selection of assessment endpoints:

- The nature of actual and potential impacts were identified.
  - Whether community structure was affected through trophic structure alterations or other community level indicators of disturbance.
  - Whether the ecological processes such as primary production and nutrient cycling rates were altered.
  - Whether particular species were affected, in particular threatened or endangered ones.

- The potential intensity of impacts were evaluated as high, medium, or no effect.

- A degree of certainty was applied to differentiate between (1) circumstances where either data or references are sufficient for probability projections to be made and (2) situations where the stress-response relationships are poorly understood or of a highly infrequent occurrence.

- If warranted by the first three considerations, a probable time scale of recovery could be derived following cessation of the stressor.

Measurement endpoints are a means of relating COPCs to an assessment endpoint. These are quantifiable values that can be directly measured in the field or laboratory. Measurement endpoints provide a means to determine if the assessment endpoints have been affected and, if so, to what degree. The assessment and measurement endpoints used to evaluate the potential adverse effects from the selected COPCs are shown in Table 6-13.

6.2.2 Exposure Assessment

Site-related contamination may increase the potential that adverse effects may occur in targeted individuals of key receptor populations. To determine if this potential exists, the magnitude, frequency, duration, and paths of exposure to COPCs are evaluated by the following:

- Identifying potential sources
- Identifying potential release(s)

Table 6-13. Ecological Endpoints

Assessment Endpoints	Measurement Endpoints	Decision Parameter
Protection of mammals[a], avian species[b], and special status species[c], from adverse effects due to elevated concentrations of COPCs[d] in soils and surface water.	Concentration of COPCs in soil and surface water. Bioaccumulation model output for secondary and third-order consumers. Inhalation concentrations calculated from soil concentrations.	If hazard quotients calculated from COPC soil or water concentrations do not exceed 1.0 for ecological receptors, then no action, otherwise consider remedial alternatives.

[a]Mammals selected for assessment include deer mouse and mule deer.
[b]Avian species selected for assessment include the American robin.
[c]Special status species selected for assessment include the golden eagle.
[d]Chemicals of potential concern.

- Estimating COPC exposure point concentrations and estimated intakes
- Characterizing routes of exposure

The reasonable maximum exposure (RME) scenario was used for this assessment. Exposure point concentrations (EPCs) are represented by the UCL95 or maximum detected value, whichever was lower. Also, 95 percent values were utilized for receptor home range values and all receptor exposure parameters (e.g., ingestion rates and body weight).

The relative abundance of contaminants, the magnitude of the potential exposures, and the exposure pathways are summarized in each SWMU section. While discussed in a qualitative sense, potential key receptor activity patterns and species abundance at exposure points were not examined in detail due to the limited scope of this assessment.

6.2.2.1 *Identification of Potential Exposure Pathways*

The pathways for transport and distribution of contaminants to an individual are critical in assessing the potential impacts of a contaminant. A conceptual site model was developed for the exposure pathways for the TEAD facility (Figure 6-2). Exposure pathways are the mechanism by which a contaminant in an environmental medium (i.e., the source) contacts an ecological receptor. A complete exposure pathway includes:

- Contaminant source
- Release mechanism that allows contaminants to become mobile or accessible
- Transport mechanism that moves contaminants away from the release
- Ecological receptor
- Route of exposure (e.g., dermal or direct contact, inhalation, or ingestion)

The major exposure pathways at TEAD are direct contact with contaminated abiotic media (i.e., animals in contact with contaminated soil), ingestion of abiotic media (i.e., ingestion of soil or surface water by birds or animals), and ingestion of contaminated biological media (i.e., ingestion of plants or animals). Inhalation of organic vapors was analyzed for the deer mouse when appropriate. Burrowing animals would be expected to have the highest contact rate since volatile organics in burrow air may be higher than in ambient air.

6.2.2.2 *Exposure Point Concentrations*

Upper 95 percent confidence limits on the arithmetic mean (UCL95) values were used to represent the EPCs. Use of the UCL95 implies that the mean concentration will fall below this value 95 percent of the time. If a smaller data set was highly variable, the upper bound concentrations may exceed the maximum values. If this were the case or if only a single detect was reported, the maximum observed concentrations were used for the EPC estimate. It was assumed that the data were normally distributed. Soil (0 to 1 foot depth) and surface water were used as the basics of the EPCs.

6.2.2.3 *Media Ingestion Rates*

Daily media ingestion rates were obtained for the ecological receptors at TEAD-S. The ingestion rates are expressed in terms of kilograms media ingested per kilograms body weight per day (i.e., kg media/kg bw/day). Dietary ingestion is the kg diet/kg bw/day ingested by the animal. Soil ingestion rates are the product of the dietary ingestion rate and the fraction of soil in the diet, which is expressed as kg soil/kg bw/day:

(Equation 6-20)

Soil Ingestion Rate (kg soil/kg bw/day)	=	Dietary Ingestion Rate * Soil Fraction in Diet (kg diet/kg bw/day)

Water ingestion is the daily volume of water (L/kg bw/day) ingested and corrected for receptor body weight. Exposure parameters for bird and animal intakes are presented in Table 6-14, which includes references for the various data sources. The 95th percentile for each exposure parameter was used as the basis for the media ingestion rate.

6.2.2.4 *Home Range Data and Area Use Factors*

Home range is the area that an animal is expected to occupy for feeding, breeding, and any other aspects of life history. The migratory species have more than one area in which they live (e.g., one area for breeding in summer and then migrating to another location in the

Table 6-14. Exposure Parameters for the Terrestrial Ecological Receptors

Receptor		Body Weight (g)	Inhalation Rate (m3/day)	Surface Area (cm2)	Dietary Ingestion Rate (kg/kg bw/d)	Water Ingestion Rate (L/kg bw/d)	Home Range (ha)	Soil Ingestion (Percent of Diet)	Soil Ingestion Rate (kg/kg bw/d)
Deer Mouse	Minimum	14.0	0.023	86.0	0.070	0.056	0.0	2.0	0.001
	Maximum	31.5	0.025	91.0	0.450	0.340	0.9	2.4	0.011
	Mean	20.5	0.024	88.5	0.192	0.147	0.1	2.2	0.004
	SD	4.1	0.001	3.5	0.090	0.073	0.2	0.3	0.000
	95th percentile	29.0	0.025	93.4	0.372	0.251	0.4	2.4	0.009
Mule Deer	Minimum	45000.0	11.5	13016.5	0.025	0.019	40.0	2.0	0.0005
	Maximum	180000.0	34.8	32050.3	0.036	0.021	900.0	2.0	0.0007
	Mean	87175.0	23.1	22533.4	0.030	0.020	470.0	2.0	0.0006
	SD	62561.3	16.5	13459.0	0.008	0.002	608.1	0.0	0.0000
	95th percentile	163125.0	33.6	31098.6	0.035	0.021	857.0	2.0	0.0007
American Robin	Minimum	5.5	0.007	182.0	0.670	0.034	0.01	9.3	0.062
	Maximum	83.6	0.060	198.0	1.520	0.140	0.42	10.4	0.158
	Mean	54.0	0.03	190.0	0.963	0.083	0.11	9.9	0.095
	SD	28.4	0.04	11.3	0.299	0.053	0.10	0.8	0.002
	95th percentile	84.8	0.06	205.7	1.388	0.133	0.24	10.3	0.144
Golden Eagle	Minimum	3000.0	1.19	2530.0	0.065	0.009	1830.0	2.800	0.002
	Maximum	5172.0	1.43	2970.0	0.140	0.011	3494.0	2.800	0.004
	Mean	4196.4	1.31	2750.0	0.100	0.010	2401.3	2.800	0.003
	SD	903.7	0.17	311.1	0.025	0.001	946.6	NA	NA
	95th percentile	5147.1	1.42	3181.2	0.133	0.011	3332.6	2.800	0.004

Source:

Body Weight	*EPA, 1993; Rust E&I, 1996; Fitzgerald et al., 1994*
Dietary Ingestion Rate	*EPA, 1993*
Water Ingestion Rate	*EPA, 1993*
Home Range	*EPA, 1993; Fitzgerald et al., 1994*
Soil Ingestion Fraction	*Beyer et al., 1994*
Habitat -mammals	*Burt and Grossenheider, 1980*
Habitat - birds	*Udvardy, 1977*
Feeding Habits-mammals	*Burt and Grossenheider, 1980; Fitzgerald et al., 1994*
Feeding Habits-birds	*Udvardy, 1977*
Surface Area	*EPA, 1993*

fall). No additional adjustment was applied to reduce the exposure to reflect migration in order to conservatively reflect exposure by nonmigratory species.

The home range values for the ecological receptors at TEAD-S were obtained, in part, from the USEPA's *Wildlife Exposure Handbook* (USEPA 1993a). The home range was used to calculate an area use factor (AUF) by dividing the area of each SWMU by the home range for each receptor as shown in Table 6-15. When the SWMU is smaller than the home range, the AUF is less than 1. This reflects the fact that the animal feeds and moves over an area larger than the SWMU and, therefore, exposure at the SWMU is reduced. When the SWMU area exceeded the home range, a value of 1 was used in the intake equations (i.e., exposure does not increase above 100 percent).

6.2.2.5 *Exposure Intakes*

Exposure intakes were calculated for each media by multiplying the EPC by the media ingestion rate and by the AUF:

(Equation 6-21)

$$\text{Exposure Intake} = \text{Media Ingestion Rate} * \text{Media concentration} * \text{AUF}$$

6.2.2.5.1 ***Exposure Intakes - Soil Ingestion Pathway.*** Exposure intakes for the soil ingestion pathway were calculated from soil data for each COPC by site. Intakes were estimated by multiplying the soil concentration (mg/kg) (also referred to as the EPC) by the soil ingestion rate (kg soil/kg bw/day) and the AUF to obtain mg/kg bw/day as follows:

(Equation 6-22)

$$\text{Exposure Intake}_{\text{mg/kg bw/day}} = \text{Soil Ingestion Rate}_{\text{(kg soil/kg bw/day)}} * \text{Soil Concentration}_{\text{(mg/kg soil)}} * \text{AUF}$$

Soil ingestion rates were summarized in Table 6-14 for each receptor. The 95th percentile for ingestion rates was used to obtain exposure intakes.

6.2.2.5.2 ***Exposure Intakes - Surface Water Ingestion Pathway.*** Surface water intakes were calculated by multiplying the daily water ingestion rate (Table 6-14) by the maximum surface water concentration of each analyte as follows:

(Equation 6-23)

$$\text{Exposure Intake}_{\text{(mg/kg-bw/day)}} = \text{Water Ingestion Rate}_{\text{(L/kg-bw/day)}} * \text{Water Concentration}_{\text{(mg/L)}}$$

Table 6-15. Home Range and Area Use Factors

SITE	Size-Acres	American Robin		Golden Eagle		Deer Mouse		Mule Deer	
		hr[a]	auf[b]	hr	auf	hr	auf	hr	auf
Boiler Blowdown	1.3	0.60	1.00E+00	8235	1.62E-04	1.05	1.00E+00	400	6.30E-04
Drainage Ditch	1.5		1.00E+00		1.80E-04		1.00E+00		7.00E-04
Chemical Unload	0.2		3.73E-01		2.70E-05		2.12E-01		1.05E-04
Wastewater Lagoons 1-3	0.7		1.00E+00		8.40E-05		6.60E-01		3.27E-04
Wastewater Lagoon 4	0.7		1.00E+00		8.40E-05		6.60E-01		3.27E-04
Fuel Spill	0.1		2.07E-01		1.50E-05		1.18E-01		5.80E-05
Drum Storage	0.3		4.56E-01		3.30E-05		2.59E-01		1.28E-04

[a]Home range in acres; 95 percent percentile except for mule deer.

[b]Area use factor.

Because most of the species at TEAD-S are adapted to an arid climate, surface water ingestion exposure estimates are likely to be highly conservative. AUFs were not applied because it was assumed that numerous home ranges could overlap at an isolated water source and that the isolated water source could serve entire populations.

6.2.2.5.3 ***Exposure Intakes - Dietary Contact Pathway.*** Exposure intakes for contact with soil were calculated as follows:

(Equation 6-24)

$$\text{Intake}_{(mg/kg/day)} = \text{Soil Concentration}_{(mg/kg)} * \text{Surface Area}_{(cm^2/day)} * \text{Adherence Factor}_{(kg/cm^2)} * \text{Absorption Factor}_{(unitless)} * \text{AUF}_{(unitless)} \div \text{BW}_{(kg)}$$

A value of $0.2 kg/cm^2$ was used for the adherence factor (ADF) (USEPA 1992a). A value of 0.1 was used for absorption of organics and 0.01 for inorganics (California EPA 1994). These parameters are consistent with Region 9 guidance for human health risk assessment.

6.2.2.5.4 ***Exposure Intakes - Soil Pathway.*** Since no biota analyses are available for TEAD-S, a dietary intake was derived using mean bioaccumulation factors (BAFs) calculated from the TEAD SWERA data as summarized in Table 6-16.

Table 6-16. Mean Bioaccumulation Factors for Dietary Intake

Metal	Plants	Jackrabbit	Invertebrates
As	0.0432	0.0136	0.0545
Cu	0.195	0.2	0.412
Hg	0.2	0.234	0.324
Ni	0.073	0.017	0.045
Pb	0.0396	0.514	0.0148
Zn	0.328	0.358	0.882

The BAFs are the ratio of analyte concentration in biotic tissue to the analyte concentration in soil calculated as follows:

(Equation 6-25)

$$BAF = \frac{concentration\ in\ tissue}{concentration\ in\ soil}$$

BAFs are specific for each receptor (Table 6-15). The BAFs, where available, were multiplied by soil concentration to obtain a dietary concentration from which intake was calculated as follows:

(Equation 6-26)

$$\text{Intake}_{(mg/kg/day)} = \text{Dietary Concentration}_{(mg/kg)} * \text{Dietary Intake Rate}_{(kg/kg\ bw/day)} * AUF$$

where

Dietary Concentration = Soil Concentration x BAF_i

The golden eagle was assumed to ingest only (i.e., 100 percent) small mammals. The TEAD-N jackrabbit BAF data (Rust E&I 1996) were used to predict concentrations in small mammals at TEAD-S:

(Equation 6-27)

$$\text{Intake}_{\text{golden eagle}} = \text{Soil Concentration} \times BAF_{JR} \times \text{Dietary Intake Rate} * AUF$$

Deer mice ingest both invertebrates and vegetation. The dietary components for the deer mouse were assumed to consist of 70 percent plants and 30 percent terrestrial invertebrates as calculated using appropriate BAFs, likewise obtained from the TEAD SWERA.

(Equation 6-28)

$$\text{Intake}_{\text{deer mice}} = (\text{Soil Concentration} * BAF_p * 0.7 + \text{Soil Concentration} * BAF_{IN} * 0.3) * \text{Dietary Intake Rate} * AUF$$

The robin ingests both invertebrates and vegetation. Dietary components were assumed to be equally split between plants and invertebrates.

(Equation 6-29)

$$\text{Intake}_{\text{robin}} = \text{Soil Concentration} \times 0.5 \times \text{Dietary Intake Rate} * AUF * (BAF_p + BAF_{INV})$$

For the mule deer, intakes were estimated with plant BAF (Rust E&I 1996) data accounting for 100 percent of the diet.

(Equation 6-30)

$$\text{Intake}_{\text{mule deer}} = \text{Soil Concentration} \times BAF_p \times \text{Dietary Intake Rate} \times AUF$$

6.2.2.5.5 ***Exposure Intakes - Air Inhalation Pathway***. Intakes of contaminants due to inhalation of air were estimated by the following equation:

(Equation 6-31)

$$Air\ Intake = \frac{IR \times CA}{BW}$$

where

Air Intake	=	mg/kg bw/day
IR	=	inhalation rate (m^3/day)
CA	=	air concentration (mg/m^3)
BW	=	body weight (kg)

Calculation of air intake is highly uncertain, in part, because of the limited nature of the data available and, in part, because information regarding the respiratory physiology (i.e., airway size, breathing rate, clearance mechanisms, and alveoli branching pattern) was unavailable in the literature reviewed. The air intakes were calculated as a component of screening, and very conservative parameters were used to overcome the lack of analytical data, pharmacokinetic information, and species-specific physiology data. No adjustment was made for absorption (i.e., absorption was conservatively assumed to be 100 percent of the inhaled dose).

The air concentrations, CA, were calculated from soil concentrations as follows:

$$CA = \frac{H \times Soil\ Concentration \times \rho \times CF}{\Theta_w + KS \times \rho + H \times \Theta_v}$$

(Equation 6-32)

where

CA = mg/m^3
H = Henry's Law Constant (cm^3 - H_2O/cm^3 - air)
Soil Concentration = mg/kg
ρ = soil bulk density (gm - soil/cm^3 - soil)
Θ_w = volumetric content of pore water in soil (cm^3 - H_2O/cm^3 - soil)
Θ_v = volumetric content of vapor in soil (cm^3 - vapor/cm^3 - soil)
KS = sorption coefficient (g/g - soil/g/cm^3 H_2O) = koc * foc
koc = soil water partition coefficient
foc = fraction organic carbon (g/g - soil)
CF = 1E-09 conversion factor

Air intakes were calculated for burrowing mammals only (i.e., the deer mouse), as burrowing mammals are expected to have the highest contact rate since VOCs in burrow air are likely to be higher than in ambient air. The 95th percentile air inhalation rate for the deer mouse is 0.025 m^3/day (USEPA 1993).

6.2.3 Toxicity Analysis

A toxicity assessment (1) evaluates the nature and extent of the adverse effects from exposure of key receptor species to the COPCs and (2) assesses the likelihood that exposure to concentrations of COPCs at the selected SWMUs will cause adverse effects. These involve a review of toxicity data on the selected COPCs and potential adverse effects on the key receptor species.

Toxicity benchmark values (TBVs) (Table 6-17) were compiled from existing sources (*Final TEAD Site-Wide Ecological Risk Assessment* (SWERA) (Rust 1996)). Screening criteria, or TBVs, were selected from the literature values and represent concentrations that, if exceeded at a particular location, might indicate a potential risk to ecological receptors.

Table 6-18 summarizes the toxicological studies reviewed for birds and mammals for the oral ingestion pathway. Where data for a key receptor species were unavailable, data for laboratory animals were reported. Many values for birds and mammals were reported as dietary concentrations (i.e., mg/kg diet or ppm). Dietary concentrations were converted to intakes (mg/kg body weight/day) using dietary ingestion rates. The conversion factors are described in Table 6-18.

In the toxicity assessment, toxicological data pertinent to the evaluation of ecological risk were reviewed and summarized. The following data sources were evaluated for toxicity information:

- Toxline (an online database specializing in toxicological data)
- USEPA documents
- Other sources including IRIS, HEAST, HSDB, and ATSDR

The toxicity assessment focused on endpoints or health effects that were likely to adversely affect populations of ecological receptors at the site, as opposed to health effects such as cancer that occur on an individual basis. This is consistent with current ecological risk assessment guidance. Health effects that potentially impact populations include increased mortality, high rates of morbidity, and reproductive effects. For the purposes of the risk assessment, reproductive effects include developmental effects (i.e., fetotoxicity, embryotoxicity), as well as indices of reproductive success such as litter size.

Table 6-17. Final Toxicity Benchmark Values Used for TEAD-S

Analyte	FINAL TBVS[a] (mg/kg bw/d)[b]			
	AR[c]	GE[d]	DM[e]	MD[f]
Acetone	NA[g]	NA	10	6
Arsenic	2.8	1.4	0.25	0.15
Beryllium	3.88	1.94	4.72	2.83
Bis(2-ethylhexyl) phthalate	2.08	1.04	11.33	6.8
Chloromethane	NA	NA	6	3.6
Chloroform	NA	NA	0.12	0.07
Chromium (III)	0.26	0.13	1.33	1.33
Copper	11.06	5.53	NA	NA
Copper_noncarn	NA	NA	4.33	2.6
p-Cresol	NA	NA	20.00	12.00
HMX	NA	NA	2.00	1.20
Dichloromethane	NA	NA	1.67	1
Fluoride	NA	NA	1.07	0.64
Lead	2.9	1.81	NA	NA
Lead (acetate)	NA	NA	1.33	0.8
Mercury	0.5	0.25	0.26	0.16
Nickel	5.82	2.91	NA	NA
Nickel_noncarn	NA	NA	17.56	10.53
Nitrate	27.16	13.58	16.67	10
Toluene	NA	NA	2.12	1.27
Total petroleum hydrocarbons	25.2	12.6	100	100
Uranium	2.38	1.19	3.33	2
Xylenes	NA	NA	16.67	10
Zinc	5.4	2.7	18.89	11.33
Zinc	NA	NA		

[a]Toxicity benchmark values.
[b]Milligrams per kilogram body weight per day.
[c]American robin.
[d]Golden eagle.
[e]Deer mouse.
[f]Mule deer.
[g]Not applicable.

Table 6-18. Toxicity Data Used for TEAD-S

Analyte	TBV[c]-Low (mg/kg bw/d)[d]	TBV-High (mg/kg bw/d)	Species	Study Endpoint UF	Study Duration UF	Intertaxon/T&E[a] UF[b] AR[e]	GE[f]	DM[g]	MD[h]	NOAEL[i] (mg/kg bw/d)	LOAEL[j] (mg/kg bw/d)	Study Description	Reference	Comment
Acetone	58.0	5800.0	Rat	10	10	NA[k]	NA	3	5	NA	5800 0	Oral LD50	RTECS, 1997 (J.Tox. Env. Health. 15:609. 1985)	
Acetone	30.0	3000.0	Mouse	10	10	NA	NA	3	5	NA	3000.0	Oral LD50	RTECS, 1997 (Pharm. Chem J 14:162. 1980)	Use this as most conservative TBV.
Acetone	80 0	8000 0	Dog	10	10	NA	NA	5	5	NA	8000.0	Oral LDLo.	RTECS, 1997 (Arch. Exp. Pathol. Pharmakol .18:218. 1984)	
Acetone	53.4	5340.0	Rabbit	10	10	NA	NA	5	5	NA	5340.0	Oral LD50	RTECS, 1997 (Food Agr. Organ. UN. Rep. Ser. 48:A86 1970)	
Acetone	54600.0	273000 0	Rat	5	1	NA	NA	3	5	NA	273000.0	Oral TDLo. 13 week male Paternal effects (spermatogenesis).	RTECS, 1997 (NTIS PB91-185975)	
Acetone	109200.0	546000.0	Mouse	5	1	NA	NA	3	5	NA	546000.0	Oral TDLo. 13 week. Changes in liver weight, spleen weight.	RTECS, 1997 (Natl. Tox. Prog. Tech. Rep. Ser. NIH 91-3122)	
Arsenic	14.0	42.0	Mallard	1	1	5	10	NA	NA	14	42	NOAEL @ 100 ppm in diet for behavior (LOAEL was 300 ppm for behavior and growth). Converted with 0.14 kg diet/kg bw from Camardese et al , 1990.	Camardese et al., 1990; Whitworth et al., 1991	Only avian value.
Arsenic	0.8	22 5	Rat	1	5	NA	NA	3	5	3.8	22.5	NOAEL (LOAEL was 22.5 mg/kg bw/day for growth, liver lesions)	Schroeder et al., 1968	Clear endpoint relating to effects on assessment endpoints.
Arsenic	0.4	NA	Grazer	1	5	NA	NA	5	4	2	NA	Maximum tolerated in diet 50 ppm, dwb (convert with 0.04 kg diet/kg bw from Sax, 1984)	Bodek et al., 1988	
Beryllium	19.4	485.0	Poultry	5	5	5	10	NA	NA	NA	485	Caused ricketts in poultry, other livestock at 0.5% of diet. Use 0.097 kg/kg bw/day from Wiseman (1987) to convert.	Friberg et al., 1979	Only value.
Beryllium	0.1	NA	Rat	1	5	NA	NA	3	5	0.54	NA	NOAEL for weight loss	Opresko et al., 1993	
Beryllium	14.2	42 5	Rat	3	1	NA	NA	3	5	NA	42 5	Mild weight loss, 2 yr study. Estimate with 0.085 g/g bw/d (Groton et al 1991).	WHO, 1990a	Long term study. Clear endpoint.
Bis(2-ethylhexyl) phthalate	10.4	156.0	Chicken	3	5	5	10	NA	NA	NA	156	*Decreased egg production and body* weight, for a 4 week exposure. Higher concentrations caused cessation of laying Used 1.45 kg body weight (Wiseman, 1987) for hen weight; 226 mg/hen/day intake.	WHO, 1992a	*Clear endpoint;* starling effect beneficial so inappropriate as TBV.
Bis(2-ethylhexyl) phthalate	2.4	NA	Starling	1	1	4	10	NA	NA	2 4	NA	*Increased body weight; 30 day exposure.* Converted from 25 ppm w/0.097 g/g bw/day for chicken (Wiseman, 1987)	WHO, 1992a	

Table 6-18. Toxicity Data Used for TEAD-S (continued)

Analyte	TBV[c]-Low (mg/kg bw/d)[d]	TBV-High (mg/kg bw/d)	Species	Study Endpoint UF	Study Duration UF	Intertaxon/T&E[e] UF[b] AR[e]	GE[f]	DM[g]	MD[h]	NOAEL[i] (mg/kg bw/d)	LOAEL[j] (mg/kg bw/d)	Study Description	Reference	Comment
Bis(2-ethylhexyl) phthalate	34.0	510.0	Rat	3	5	NA	NA	3	5	NA	510	Decreased body weight; testicular atrophy; 6,000-12,000 ppm diet converted with 0 085 (Groton et al., 1991)	WHO, 1992a	Clear endpoint
Bis(2-ethylhexyl) phthalate	52.0	780.0	Mouse	3	5	NA	NA	3	5	NA	780	Decreased body weight; male reproductive effects; 3,000-6,000 ppm diet converted with 0.26 g/g bw/day (EPA, 1993)	WHO, 1992a	
Chloromethane	18.0	1800.0	Rat	10	10	NA	NA	3	5	NA	1800	Oral LD50.	RTECS, 1997 (Prehled. Prumyslove. Toxikol. Org. Latky. 1986:86. 1986)	Only value.
Chloroform	9.1	908.0	Rat	10	10	NA	NA	3	5	NA	908	Oral LD50. Affected behavior, blood and body weight.	RTECS, 1997 (J. Environ Sci. Health Part B. 17:205. 1982)	
Chloroform	0.4	36.0	Mouse	10	10	NA	NA	3	5	NA	36	Oral LD50.	RTECS, 1997 (Arch. Toxicol. Suppl 2:371. 1979)	Most conservative value.
Chloroform	10.0	1000.0	Dog	10	10	NA	NA	5	5	NA	1000	Oral LDLo.	RTECS, 1997 (Q. J Pharm. Pharmacol. 7:205:1934).	
Chloroform	5.0	500.0	Rabbit	10	10	NA	NA	5	5	NA	500	Oral LDLo.	RTECS, 1997 (Arch. Exp. Pathol. Pharmakol .97:86. 1923)	
Chloroform	18.0	1260.0	Rat	7	10	NA	NA	3	5	NA	1260	Oral TDLo at day 6-15 of pregnancy. Fetotoxicity, developmental abnormalities	RTECS, 1997 (Toxicol Appl. Pharmacol 29:348.1974).	
Chloroform	57.1	4000.0	Rat	7	10	NA	NA	3	5	NA	4000	Oral TDLo at day 6-15 of pregnancy. Fetotoxicity.	RTECS, 1997 (J. Environ. Sci. Health Part B 18:333. 1983).	
Chloroform	435.4	2177.0	Mouse	5	1	NA	NA	3	5	NA	2177	Oral TDLo for 4 week study. Effects on newborn growth statistics, biochemistry and metabolic.	RTECS, 1997 (Neurobehav. Toxicol. 1:199, 1979).	
Chloroform	423.0	2115.0	Mouse	5	1	NA	NA	3	5	NA	2115	Oral TDLo for 4 week study. Effects on newborn	RTECS, 1997 (Environ. Health Perspective 46:127 1982).	
Chloroform	3.7	260.0	Rabbit	7	10	NA	NA	5	5	NA	260	Oral TDLo when given day 6-18 of pregnancy Fetotoxic, developmental abnormalities of musculoskeletal system.	RTECS, 1997 (Toxicol. Appl. Pharmacol 29.348. 1974)	
Chloroform	302 4	7560 0	Rat	5	5	NA	NA	3	5	NA	7560	Oral TDLo in 21 day study Changes in liver and body weight.	RTECS, 1997 (Gig Sanit 48·10. 1983)	
Chloroform	0.1	5.0	Rat	5	10	NA	NA	3	5	NA	5	Oral TDLo in 10 day study. Changes in liver, biochemistry.	RTECS, 1997 (Toxicology 14:23 1979)	

Table 6-18. Toxicity Data Used for TEAD-S (continued)

Analyte	TBV[c]-Low (mg/kg bw/d)[d]	TBV-High (mg/kg bw/d)	Species	Study Endpoint UF	Study Duration UF	Intertaxon/T&E[a] UF[b] AR[e]	GE[f]	DM[g]	MD[h]	NOAEL[i] (mg/kg bw/d)	LOAEL[j] (mg/kg bw/d)	Study Description	Reference	Comment
Chloroform	35.0	1750.0	Mouse	5	10	NA	NA	3	5	NA	1750	Oral TDLo in 14 day study. Changes in liver weight, spleen weight, body weight	RTECS, 1997 (Environ. Health. Perspec 46:127 1982)	
Chromium (III)	0.0	NA	Black duck (adult)	3	5	5	10	NA	NA	0.63	NA	NOAEL (toxicity endpoint unknown) for adults (0.63 mg/kg bw/d converted with 0.063 kg/kg bw/d for adult from EPA, 1993b)	CEPA, 1994	
Chromium (III)	0.4	1.3	Black duck (juvenile)	3	1	5	10	NA	NA	NA	1.26	10 ppm in diet decreased growth, survival in juvenules (1.26 mg/kg bw/d converted with 0.126 kg/kg bw/d for 100 gm duckling estimated from allometric equations in EPA, 1993) DJR (g/g bw/d)=(0.495 Wt^(0.704))/BW	CEPA, 1994; EPA, 1993	
Chromium (III)	1.3	2.6	Tern	1	1	5	10	NA	NA	1.28	NA	NOEL for wild populations. Concentration in major prey items 7.6 ppm converted by author. No effect on reproduction or population success.	CEPA, 1994	Use this study as it relates directly to assessment endpoint; test species related to receptor species
Chromium (III)	0.1	1.0	Turkey	3	5	5	10	NA	NA	NA	0.97	10 ppm in diet converted with 0.097 kg diet/kg bw/d for chicken (Wiseman, 1987) decreased egg production.	CEPA, 1994	
Chromium (III)	3.2	NA	Chicken	3	1	5	10	NA	NA	9.7	NA	NOEL (toxicity endpoint unknown) for 32 days was a 100 ppm diet.	CEPA, 1994	
Chromium (III)	6.7	NA	Cat	3	1	NA	NA	5	5	20	NA	NEL (toxicity endpoint unknown) for 80 day exposure to 50-1000 mg/cat/d; convert with assumed body weight of 2.5 kg.	NAS, 1974	Use this study as it is long-term; dietary exposure.
Chromium (III)	0.1	NA	Rat	3	5	NA	NA	3	5	1.78	NA	NEL(toxicity endpoint unknown) for rats exposed to 5 mg/L in drinking water; convert with ingestion rate of 0.356 L/kg bw/d (Perry et al., 1989)	NAS, 1974	
Copper	5.8	NA	Mallard	1	5	5	10	NA	NA	29	NA	NOAEL for weight gain, mortality	Opresko et al., 1993	
Copper	55.3	72.7	Chicken	1	1	5	10	NA	NA	55.29	72.653	NOAEL of 570 ppm for 10 wks. for weight gain, mortality At 749 ppm, mortality was 15%, weight reduced 30% relative to controls. Convert with 0.097 g/g bw/d, Wiseman, 1987.	Mehring et al., 1960	Long-term study with clear endpoints.
Copper	1.0	NA	Sheep	1	1	NA	NA	5	4	1	NA	Maximum chronic intake tolerated for grazers is 25-300 ppm in diet, dwb. Daily intake calculated with 0.04 kg diet/kg bw for cow (Sax, 1984)	Doherty et al., 1969; Bodek et al., 1988; Friberg et al., 1979	
Copper	4.0	20.0	Sheep	5	1	NA	NA	5	4	NA	20	Hemolysis after 9 weeks exposure.	Friberg et al., 1979	

Table 6-18. Toxicity Data Used for TEAD-S (continued)

Analyte	TBV[c]-Low (mg/kg bw/d)[d]	TBV-High (mg/kg bw/d)	Species	Study Endpoint UF	Study Duration UF	Intertaxon/T&E[a] UF[b] AR[e]	GE[f]	DM[g]	MD[h]	NOAEL[i] (mg/kg bw/d)	LOAEL[j] (mg/kg bw/d)	Study Description	Reference	Comment
Copper_noncarn[k]	13.0	NA	Rat	1	1	NA	NA	3	5	13	NA	Rats gained more weight on 50 ppm diet; converted with 0.26 kg diet/kg bw/d for mouse (EPA, 1993b).	Friberg et al., 1979	Use for non-carnivores. Actually a beneficial effect so if HQs high, check literature.
Copper	8.5	17.0	Pig	1	1	NA	NA	5	4	8.5	17	Pigs gained more weight on 250 ppm (8.5 mg/kg bw/d). At 500 ppm diet (17 mg/kg bw/d), anemia occurred. Converted with 0.034 kg/kg bw/d (Wiseman, 1987)	Friberg et al., 1979	
Copper_carn[m]	7.9	13.0	Mink	1	1	NA	NA	5	5	7.865	13	25-50 ppm NEL or beneficial. 100 ppm in diet decrease weight gain, may increase kit mortality due to effect on lactation. Controls had 60.5 ppm in diet. Study 357 d duration. Convert w/0.13 g/g/d EPA, 1993.	Aulerich et al., 1982	Use for carnivores.
Copper	0.3	NA	Dog	1	1	NA	NA	5	5	0.32	NA	Nutritional requirement	NAS, 1974b	
p-Cresol	60.0	NA	Rat	1	1	NA	NA	3	5	60	NA	NOAEL, chronic, oral study demonstrating decreased fetal weight.	IRIS, 1996	Only value.
Cyclotetramethylene tetranitramine (Hmx)	6.0	150.0	Rat	5	5	NA	NA	3	5	NA	150	TDLO (liver, kidney damage, decreased weight gain), subchronic exposure	Everett et al, 1986	Only value.
Dichloromethane	14.3	*100.0*	*Mouse*	7	1	NA	NA	3	5	NA	100	Increase mortality, decrease body weight in 36 week oral study.	CEPA, 1993a	
Dichloromethane	5.0	125.0	Rat	1	1	NA	NA	3	5	5	125	NOEL, 2 yr. study, based on non-neoplastic liver changes and toxicity. Decrease body weight and water consumption in 125 mg/kg bw/d group. Administered in drinking water.	CEPA, 1993a	Chronic lifetime no effect level.
2-6-Dinitrotoluene	3.4	51.0	Rat	3	5	N/A	N/A	3	5	N/A	51	LOAEL (weight gain, reproductive effects), subc.	USEPA, 1992	
2-6-Dinitrotoluene	2.3	35.0	Mouse	3	5	N/A	N/A	3	5	N/A	35	LOAEL (weight gain, reproductive effects), subc.	USEPA, 1992	
2-6-Dinitrotoluene	1.3	20.0	Dog	3	5	N/A	N/A	5	5	N/A	20	LOAEL (weight gain, reproductive effects), subc.	USEPA, 1992	Most appropriate for chronic studies
Fluoride	3.2	3.2	Rat	1	1	NA	NA	3	5	NA	3.2	LOAEL for bone mineralization effects, chronic study with drinking water ingestion. Skeletal effects inconsistant in rats; other studies report NOEL at 5 weeks at 12.7 mg/kg bw/d, LOEL at 21 d of 4.7 mg/kg bw/d.	CEPA, 1993b	Clear endpoint. Lower dosages in other studies have beneficial or unclear effects.

Table 6-18. Toxicity Data Used for TEAD-S (continued)

Analyte	TBV[c]-Low (mg/kg bw/d)[d]	TBV-High (mg/kg bw/d)	Species	Study Endpoint UF	Study Duration UF	Intertaxon/T&E[e] UF[b] AR[e]	GE[f]	DM[g]	MD[h]	NOAEL[i] (mg/kg bw/d)	LOAEL[j] (mg/kg bw/d)	Study Description	Reference	Comment
Fluoride	0.8	NA	Mouse	1	1	NA	NA	3	5	0.8	NA	Stimulate bone formation 20% above control. At 363.2 mg/L (79.9 mg/kg bw/d) converted with 0.22 L/kg bw/d ingestion rate for deer mouse (EPA, 1993), decreased survival occurred.	CEPA, 1993b	
Fluoride	0.3	NA	Dog	1	1	NA	NA	5	5	0 32	NA	Unspecified changes in bone at histopathologiecal level.	CEPA, 1993b	
Lead	14.5	43.5	Kestrel	1	1	5	8	NA	NA	14.5	NA	NOAEL (for survival, growth) from diet of 50 ppm (25 mg/kg bw/d) converted with 0 29 kg diet/kg bw (kestrel)(EPA, 1993b). A NOAEL of 14.5 mg/kg for survival, histopathology and reproduction also reported	Franson et al., 1983; Pattee, 1984; Hoffman et al , 1985a,b	Study concerns assessment endpoint and has test species closely related to site receptor species.
Lead (acetate)	3.2	NA	Chicken	3	1	5	10	NA	NA	9.7	NA	Dietary level of 100 ppm lead acetate tolerated, 8 wk study, 0.097 g/g bw/d (Wiseman, 1987) used to convert.	Ammerman et al , 1973	
Lead	1.3	6.5	Mice and rats	5	1	NA	NA	3	5	NA	6.5	LOAEL of 25 mg/kg diet lead salts. Caused impaired reproduction. Converted with 0.26 kg diet/kg bw (EPA, 1993).	Venugopal and Luckey, 1978	
Lead (acetate)	4.0	170.0	Rat	3	1	NA	NA	3	5	11.985	170	Females on 2000 ppm had higher mortality than controls; males on 500 and 2000, but not 1000 ppm diet had higher mortality. Controls high mortality over 2 year study. Uses 141 ppm as NOAEL, 2000 as LOAEL. Use 0.085 g/g/d, Groton et al., 1991 to convert.	Azar et al., 1973	Study concerns assessment endpoint and has test species closely related to site receptor species. Long term study.
Lead (acetate)	79.0	NA	Dog	1	1	NA	NA	5	5	79	NA	NOAEL for appearance, behavior, weight gain, mortality, or neurology for dogs on 500 ppm diet for 2 yr. Convert with ingestion rate of 0.158 g/g bw/d for red fox (EPA, 1993).	Azar et al., 1973	
Lead	1 2	NA	Grazer	1	1	NA	NA	5	4	1.2	NA	Maximum tolerated in diet 30 ppm, dwb. Convert with 0.04 kg diet/kg bw (Sax, 1984)	Bodek et al., 1988	
Mercury	2 5	12 5	Chicken	1	1	5	10	NA	NA	2.5	12.5	NOAEL for growth, 12.5 the LOAEL (convert with 0.097 kg diet/kg bw/day (Wiseman, 1987)). 12.5 mg/kg bw/d affects quail reproduction. 1.1 NEL for starling.	Thaxton et al , 1975; Thaxton and Parkhurst, 1973; Nicholson and Osborn, 1984.	Only inorganic avian data
Mercury (organic)	0 1	0.3	Pheasant	3	1	5	10	NA	NA	NA	0.25	LOAEL for reproductive effects in a 350 d study with organic mercury. This was a NOAEL for mortality.	Spann et al , 1972	

Table 6-18. Toxicity Data Used for TEAD-S (continued)

Analyte	TBV[c]-Low (mg/kg bw/d)[d]	TBV-High (mg/kg bw/d)	Species	Study Endpoint UF	Study Duration UF	Intertaxon/T&E[a] UF[b] AR[e]	GE[f]	DM[g]	MD[h]	NOAEL[i] (mg/kg bw/d)	LOAEL[j] (mg/kg bw/d)	Study Description	Reference	Comment
Mercury (organic)	0.0	0.2	Mallard	1	1	5	10	NA	NA	0.0315	0.189	Study with 3 generations fed 0.5 and 3 ppm methyl mercury in diet. NEL for body weight, mortality was 0.5 ppm Convert with 0.063 kg/kg bw/d EPA, 1993b. LOAEL 3 ppm for hatchling survival decrease 10%.	Heinz, 1976	
Mercury (organic)	0.1	0.2	Black duck	3	1	5	10	NA	NA	NA	0.189	Diet of 3 ppm methyl mercury over 2 yr. period reduced reproductive success. Convert with 0.063 kg/kg bw/d EPA, 1993	Finley and Stendall, 1978	
Mercury (organic)	0.1	0.7	Red-tailed hawk	3	1	5	4	NA	NA	0.3861	0.7128	No mortality (NEL) during 12 week study at 3 9 ppm methyl mercury in diet. Mortality (LEL) at 7 2 ppm. Converted with 0.099 kg diet/kg bw/d from EPA, 1993b.	Fimreite and Karsted, 1971	
Mercury (organic)	0.6	2.2	Rat	1	1	NA	NA	3	5	0.56	2.2	NOAEL for 2 yr. study with organic mercury. 2.2 mg/kg bw/d the LOAEL for growth, mortality.	Fitzhugh et al , 1950	
Mercury	14.0	NA	Rat	1	1	NA	NA	3	5	14	NA	NOAEL for 2 yr. study with inorganic mercury for reproduction, development. No LOAEL	Fitzhugh et al., 1950	
Mercury	0.8	3 9	Mouse	5	1	NA	NA	3	5	NA	3.9	Increased morbidity. Converted from 15 ppm in diet with 0.26 kg diet/kg bw/d (EPA, 1993)	Mitsumori et al., 1981	Use this study as mice may be more sensitive than rats based on Fitzhugh et al., 1950 study.
Mercury	0.1	NA	Mink	3	5	NA	NA	5	5	0.75	NA	NOAEL (toxicity endpoints unknown)	Aulerich et al., 1974	
Nickel	29.1	87.3	Chicken	3	1	5	10	NA	NA	NA	87.3	900 LEL for growth inhibition (estimated from 900 ppm diet and 0.097 kg/kg bw/d from Wiseman, 1987). 1,000 ppm a NEL in other studies.	Venugopal and Luckey, 1978.	Only avian value.
Nickel_noncarn	52.7	158.0	Rat	3	1	NA	NA	3	5	NA	158	TDlo for multigeneration study for effects on embryo or fetus.	RTECs, 1996	Use this study for non-carnivores as rat related to two site receptors. Long term.
Nickel	4 8	NA	Rat	1	5	NA	NA	3	5	24.15	NA	NOAEL for reproduction	Opresko et al , 1993	
Nickel_carn	4.0	NA	Cat, dog	3	1	NA	NA	5	5	12	NA	NEL for 200 day study	Venugopal and Luckey, 1978	Use this study for carnivores. Long term.
Nitrate, nitrite-nonspecific	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Nitrate	135.8	407 4	Poultry	3	1	5	10	NA	NA	NA	407.4	4.2 g/kg diet decreased weight gain, caused methemoglobinaemia, clinical changes in 4 week study. Convert with 0.097 g/g bw/d ingestion for chicken (Wiseman, 1987).	Atef et al , 1991	Primary citation. Long study with diet vehicle.

Table 6-18. Toxicity Data Used for TEAD-S (continued)

Analyte	TBV[c]-Low (mg/kg bw/d)[d]	TBV-High (mg/kg bw/d)	Species	Intertaxon/T&E[a] UF[b] Study Endpoint UF	Study Duration UF	AR[e]	GE[f]	DM[g]	MD[h]	NOAEL[i] (mg/kg bw/d)	LOAEL[j] (mg/kg bw/d)	Study Description	Reference	Comment
Nitrate	7.9	277.2	Japanese quail	7	5	5	10	NA	NA	NA	277.2	Increased mortality when given 3960 ppm or higher in drinking water. Convert with 0 059 BW^0.67 (EPA, 1993) for a 0.5 kg chicken, or ingestion rate of 0.07 L/kg bw/d.	Bruning-Fann and Kaneene, 1993	
Nitrate	8.0	279.3	Turkey	7	5	5	10	NA	NA	NA	279.3	Increased mortality when given 3990 ppm or higher in drinking water. Convert with 0.059 BW^0.67 (EPA, 1993) for a 0.5 kg chicken, or ingestion rate of 0.07 L/kg bw/d. Slowed growth.	Bruning-Fann and Kaneene, 1993	
Nitrate	50.0	5000 0	Rat	10	10	NA	NA	3	5	NA	5000	LD50 was 5 g sodium nitrate per kg..	Bruning-Fann and Kaneene, 1993	Use this value for monogastrics.
Nitrate	44.0	660.0	Cow	3	5	NA	NA	5	4	NA	660	Increased weight of pituitary gland. Effect on function unknown.	Bruning-Fann and Kaneene, 1993	
Nitrate	100.0	NA	Ruminant	1	1	NA	NA	5	4	100	NA	Acute poisoning occurs when forage exceeds 0.5% nitrate. Convert with 0.02 g/g bw/d from DIR=0.577BW^0.727/BW, BW=180 kg (EPA, 1993). Other studies show tolerate 1.2% in forage.	Bruning-Fann and Kaneene, 1993	Much discrepancy in ruminant values Use this to be conservative, but track through risk characterization.
Nitrate	30 0	NA	Ruminant	1	1	NA	NA	5	4	30	NA	Acute poisoning occurs when drinking water exceeds 500 ppm nitrate WIR=0.099BW^0 9/BW (EPA, 1993), or 0.06 L/kg bw/d for a 180 kg ruminant.	Bruning-Fann and Kaneene, 1993	
Nitrite	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Toluene	6.4	636.0	Rat	10	10	NA	NA	3	5	NA	636	Oral LD50.	RTECS, 1997 (Neurotoxicology 2:567. 1981)	Use this as most conservative TBV.
Toluene	104.0	7280.0	Rat	7	10	NA	NA	3	5	NA	7280	Oral TDLo for effects on embryo or fetus (fetotoxicity) when given day 6-19 of pregnancy.	RTECS, 1997 (Pediatr. Res. 36:811. 1994)	
Toluene	128.6	9000.0	Mouse	7	10	NA	NA	3	5	NA	9000	Oral TDLo for effects on embryo or fetus (fetotoxicity) when given day 6-15 of pregnancy. At 30000 mg/kg, developmental abnormalities occurred.	RTECS, 1997 (Teratology 19:41A. 1979)	
Toluene	8476.0	42380 0	Rat	5	1	NA	NA	3	5	NA	42380	Oral TDLo for effects on sensory organs, weight loss in 49 day study	RTECS, 1997 (Neurotoxicol Teratol 10.525. 1989)	
Toluene	1105.8	27645.0	Rat	5	5	NA	NA	3	5	NA	27645	Oral TDLo for effects on urine chemistry (proteinuria) in 3 week study.	RTECS, 1997 (Toxicologist 5:62. 1985)	
Toluene	32400.0	162000.0	Rat	5	1	NA	NA	3	5	NA	162000	Oral TDLo for effects on brain weight, liver weight, changes in kidney weight in 13 week study.	RTECS, 1997 (Natl. Toxicol Program Tech. Rep Ser. NTP-TR-371, 1990)	

Table 6-18. Toxicity Data Used for TEAD-S (continued)

Analyte	TBV[c]-Low (mg/kg bw/d)[d]	TBV-High (mg/kg bw/d)	Species	Intertaxon/T&E[e] UF[b]: Study Endpoint UF	Study Duration UF	AR[e]	GE[f]	DM[g]	MD[h]	NOAEL[i] (mg/kg bw/d)	LOAEL[j] (mg/kg bw/d)	Study Description	Reference	Comment
Toluene	45400.0	227000.0	Mouse	5	1	NA	NA	3	5	NA	227000	Oral TDLo for effects on brain weight, liver weight, changes in kidney weight in 13 week study.	RTECS, 1997 (Natl Toxicol. Program Tech. Rep. Ser. NTP-TR-371, 1990)	
Toluene	588.0	2940.0	Mouse	5	1	NA	NA	3	5	NA	2940	Oral TDLo for effects on liver weight, thymus weight, decreased immune response in 4 week study.	RTECS, 1997 (Environ. Res. vol. 49:93.1989)	
Toluene	168.0	8400.0	Mouse	5	10	NA	NA	3	5	NA	8400	Oral TDLo for effects on leucocyte count in 14 d study.	RTECS, 1997 (Drug Chem. Toxicol. 17:317. 1994)	
Total petroleum hydrocarbons	126 0	1260.0	mallard	1	1	5	10	NA	NA	126	1260	NOAEL of 20,000 ppm diet, 22 week study for mortality, body weight, food consumption, reproduction & hatching success. This was a LOAEL for serum chemistry, eggshell thickness. Converted with 0 063 kg/kg bw/d (EPA, 1993). 2000 ppm a NOAEL, all effects.	Stubblefield et al , 1995a	Only avian value.
Total petroleum hydrocarbons	500.0	10000.0	ferret	1	10	NA	NA	5	5	5000	NA	5 day NOAEL for serum chemistry. Minor effects noted were increased serum albumin, decreased spleen weight in treated females.	Stubblefield et al., 1995b	Best mammalian study as it was multiple dose, not single dose.
Total petroleum hydrocarbons	320.0	16000.0	mouse	5	10	NA	NA	3	5	NA	16000	LD50 range for three crude oils exceeded highest test doses of >10 - 16 g/kg bw.	Smith et al., 1980	
Uranium	118 5	5925 0	Chicken	5	10	5	10	NA	NA	NA	5925	Chicks ingected with 250 mg/kg bw Uranyl nitrate (UN) had tubular nephrosis and hepatic necrosis after 48 hr. MW U=238.03 g/mol; UN=502.13 g/mol. U dose = 118.5 mg/kg bw.	Harvey et al., 1986a	No avian studies with oral dosing. Less than 1% of ingested U absorbed from GI tract (Robinson et al, 1984); average human GI absorption 1-2% (Wrenn et al, 1985).
Uranium	11 9	595 1	Japanese quail	5	10	5	10	NA	NA	NA	595 075	Quail injected with 50 umol U/kg bw (11 9 mg/kg bw) had mild to severe kidney lesions 18 hr after injection Use 2% absorption to get an estimated oral dose	Kupsh et al , 1991	((50 umol/10^6)*238 03 g/mol*1000 mg/g)/2% Quail may be more sensitive than chicken so use quail studies.

Table 6-18. Toxicity Data Used for TEAD-S (continued)

Analyte	TBV[c]-Low (mg/kg bw/d)[d]	TBV-High (mg/kg bw/d)	Species	Study Endpoint UF	Study Duration UF	Intertaxon/T&E[a] UF[b] AR[e]	GE[f]	DM[g]	MD[h]	NOAEL[i] (mg/kg bw/d)	LOAEL[j] (mg/kg bw/d)	Study Description	Reference	Comment
Uranium	37.9	3792.0	Chicken	10	10	5	10	NA	NA	NA	3792	LD50 was 235 UN mg/kg bw (111.4 mg/kg bw U), and TDlo for lethality was 160 mg/kg bw (75.84 mg/kg bw U), which is 100-200 times the 14-21 day LD50 for rats Chicks insensitive to U. Adjust with 2% absorption for oral exposure relative to injection.	Harvey et al., 1986 b	
Uranium	11.9	595.1	Japanese quail	5	10	5	10	NA	NA	NA	595.075	Quail injected IV with 15 umol U/100 g bw died; 5 umol U/100 g bw (11.9 mg/kg bw) survived but moribund by 18 hr. Adjust with 2% absorption for oral exposure relative to injection.	Robinson et al., 1984	5 umol/100 g bw= 5 E-5 mol/kg bw=5E-5*238.03 g/mol *1000 mg/g/2%
Uranium	10.0	25.0	Mouse	1	1	NA	NA	3	5	10	25	NOAEL for 60 day study was 10 mg/kg/d for fertility, gestation, survival. LOAEL of 25 mg/kg/d caused embryolethality.	Paternain et al , 1989	Use this as is longer study providing NOAEL and LOAEL.
Uranium	0.2	5.0	Mouse	3	10	NA	NA	3	5	NA	5	Teratogenic when given by gavage day 6-15 of pregnancy at 5 mg/kg bw/d	Domingo, 1994	
Uranium	5.0	50 0	Mouse	1	1	NA	NA	3	5	5	50	When dosed from day 13 of pregnancy to 21 d postbirth, the 50 mg/kg dose decreased litter size, wheras the 5 mg/kg bw dose had no effect	Domingo, 1994	
Xylenes	50.0	500.0	Rat	1	5	NA	NA	3	5	250	500	NOAEL, short term study (body weight, survival, hepatic). Converted with body weight of 0.16 kg, inhalation rate of 0.126 m3/d (EPA, 1993)	CEPA, 1993c	Toxicity endpoints relate to assessment endpoints.
Xylenes	66.7	NA	Mouse	3	5	NA	NA	3	5	1000	NA	NOAEL.	CEPA, 1993c	
Zinc	23.0	NA	Bird	1	1	5	10	NA	NA	23	NA	Assumed based on nutritional requirements of 2.4-8.0 mg/kg bw/day for quail, ducks, chicken.	Wiseman, 1987	
Zinc	27.0	189.0	Mallard	7	1	5	10	NA	NA	NA	189	Ducks fed 3000 ppm in diet had decreased gonad size, probably impairment of function. Overt toxicity after 20 days. Mortality high by 60 days. Convert with 0 063 g/g/d, EPA, 1993	Gasaway and Buss, 1972	Appropriate endpoint. Long study.
Zinc	97.0	145 5	Chicken	1	1	5	10	NA	NA	97	145.5	Tolerate 1000 ppm in feed, but 1500 and above decreased growth. Carbonate >sulfate>oxide in toxicity Convert w/ 0 097 g/g/d from Wiseman, 1987.	Roberson and Schaible, 1960	
Zinc	97 0	485.0	Chicken	3	1	5	10	NA	NA	291	485	Minimal mortality (15%) at 10 wks on diet with 5000 ppm. Mortality was 2 5% for 3000 ppm treatment; use this as NOAEL Convert with 0.097 g/g/d from Wiseman, 1987.	Johnson et al., 1962	

Table 6-18. Toxicity Data Used for TEAD-S (continued)

Analyte	TBV[c]-Low (mg/kg bw/d)[d]	TBV-High (mg/kg bw/d)	Species	Study Endpoint UF	Study Duration UF	Intertaxon/T&E[a] UF[b] AR[e]	GE[f]	DM[g]	MD[h]	NOAEL[i] (mg/kg bw/d)	LOAEL[j] (mg/kg bw/d)	Study Description	Reference	Comment
Zinc	20.0	100.0	Mouse	1	5	NA	NA	3	5	NA	100	500 mg/L in drinking water causes histopathological changes. Convert with 0 2 L/kg bw/day from EPA, 1993. Use low UF because no direct link with population effects.	Friberg et al., 1979	
Zinc	56.7	340.0	Rat	3	1	NA	NA	3	5	170	340	0.2% in diet NOAEL for effects on fetus. 0.4% in diet caused reproductive effects. Study ranged from 16 to 40 days. Convert with 0.085 kg/kg bw/d from Groton et al , 1991.	Schlicker and Cox, 1968	Use this. Consistent with other rat study as well.
Zinc	34.0	850.0	Rat	5	5	NA	NA	3	5	NA	850	1% in diet toxic to rats (850 mg/kg bw/d converted with 0.085 kg/kg bw/d from Groton et al., 1991).	Lewis et al , 1957	
Zinc	11.3	NA	Pig	3	1	NA	NA	5	4	34	NA	NOAEL based on 1000 ppm diet and ingestion rate of 0.034 kg/kg bw/d (Wiseman, 1987). Duration of study 14 - 17 wks.	Sutton and Nelson, 1937	Study does not provide a LOAEL so not as useful as rat study.
Zinc	40.0	NA	Grazer	1	1	NA	NA	5	4	40	NA	Maximum tolerated in diet 300-1000 ppm, dwb.	Bodek et al., 1988	
Zinc	5.0	NA	Sheep	1	1	NA	NA	5	4	5	NA	No adverse effect on development of fetus when given to ewes during gestation.	James et al., 1966	

[a]Intertaxon/Threatened and Endangered.
[b]Uncertainty factor.
[c]Toxicity benchmark values.
[d]Milligrams per kilogram per body weight per day.
[e]American robin.
[f]Golden eagle.
[g]Deer mouse.
[h]Mule deer.
[i]No observed adverse effects level.
[j]Lowest observed adverse effects level.
[k]Not applicable.
[l]Non-carnivores.
[m]Carnivores.

Carcinogenicity and mutagenicity were not used as endpoints for the ecological risk assessment as these are effects that alter an individual's chance of survival. If cancer rates were very high, the endpoint for the population would be survival.

The literature was reviewed for data regarding no observed adverse effects levels (NOAELs). Chronic studies, wherein ecological receptors are exposed for entire lifetimes, were considered preferable to studies of shorter duration. If NOAELs were unavailable, the lowest observed adverse effects level (LOAEL) or other toxicity values were used. Where data were available, toxicity values for wildlife species likely to be found at TEAD were used. Where possible, data from short-term studies (i.e., single dose or less than a week) and dose levels or dietary intakes that resulted in mortality were avoided.

The USEPA, the State of Utah, and the USAEC agreed upon an approach for the use of uncertainty factors (UFs) for the TEAD SWERA. The same approach was adopted for the TEAD-S assessment. Table 6-19 presents the uncertainty factors for intertaxon differences, sensitive species endpoints, ecotoxicological study duration, and study endpoints.

Intertaxon extrapolation uncertainty factors adjust for the taxonomic differences between the TEAD-S receptors and the species used in the toxicological tests cited in Table 6-17. The premise is that there is less uncertainty when the test species is more closely related to the TEAD-S receptor. The maximum uncertainty for this category was 5, which was applied if the test species was in the same class as the TEAD-S receptor, but in a different order. Data were not extrapolated between taxonomic class (i.e., data for mammals were not applied to birds). When the TEAD-S receptor represented a special status species, the intertaxon UF was multiplied by 2. The taxonomic classification is given in Table 6-20.

When the HQ exceeds a value of 1, some element of ecological risk is assumed. When the exposure intakes are lower than the acceptable or "safe" intake (TBV), ecological risk is considered minimal or nonexistent, and the resulting HQ is less than 1. HQs between 1 and 10 are likely to be within the bounds of uncertainty for the assessment. HQs in excess of 10 may present an actual risk and bear further consideration.

6.2.4.1 *Risk Estimation*

HQs and HIs calculated for the SWMU 13 and 17 sites are reported in Sections 7 and 8.

6.2.4.2 *Uncertainty Analysis*

Along with the uncertainties discussed for the human health risk assessment in Section 6.1.5, there are additional considerations for the ecological risk assessment. These include:

- Derivation of exposure point concentrations
- Selection of ecological receptors

Table 6-19. Uncertainty Factors (UFs) Used at TEAD-S

Uncertainty Category	Duration/Endpoint	Uncertainty Factor
Intertaxon Extrapolation	Same class, different order	5
	Same order, different family	4
	Same family, different genus	3
	Same genus, different species	2
	Same species	1
	Special Status Species (includes Federal Threatened and Endangered (T&E) and State of Utah Sensitive species)	2
Study Duration	Acute (≤ 14 days)	10
	subacute, subchronic (15-30 days)	5
	Duration > 30 days	1
Study Endpoint	LD50, LC50	10
	TD_{LO} for lethality	7
	TD_{LO} for nonlethal/sublethal effects	5
	NOAEL/NOEL lethal *or* LOAEL/LOEL for nonlethal	3
	NOAEL for nonlethal	1

Note.—Special Status Species UF used *in addition to* other Intertaxon Extrapolation UFs where applicable.

Table 6-20. Taxonomic Classification for Uncertainty Factor Application

Common Name	Class	Order	Family	Genus	Species	Key Receptor	Status
American kestrel	Aves	Falconiformes	Falconidae	Falco	sparverius	N[a]	
American robin	Aves	Passeriformes	Turdidae	Turdus	migratorius	Y[b]	
Bald eagle	Aves	Falconiformes	Accipitridae	Haliaeetus	leucocephalus	N	
Barn Owl	Aves	Strigiformes	Tytonidae	Tyto	alba	N	
Belted kingfisher	Aves	Coraciiformes	Alcedinidae	Megaceryle	alcyon	N	
Black duck	Aves	Anseriformes	Anatidae	Anas	rubripes	N	
Blue Grouse	Aves	Galliformes	Tetraonidae	Dendragapus	obscurus	N	
Chicken	Aves	Galliformes	Phasianidae	Gallus	domesticus	N	
Golden eagle	Aves	Falconiformes	Accipitridae	Aquila	chrysaetos	Y	Utah SS
Gray partridge	Aves	Galliformes	Phasianidae	Perdix	perdix	N	
Great horned owl	Aves	Strigiformes	Strigidae	Bubo	virginianus	N	
Mallard duck	Aves	Anseriformes	Anatidae	Anas	platyrhynchos	N	
Mountain bluebird	Aves	Passeriformes	Turdidae	Sialia	currucoides	N	
Mourning dove	Aves	Columbiformes	Columbidae	Zenaida	macroura	N	
Partridge sp.	Aves	Galliformes	Perdicidae	NA[c]	NA	N	
Passerine	Aves	Passeriformes	NA	NA	NA	N	
Pelican sp.	Aves	Pelecaniformes	Pelicanidae	NA	NA	N	
Peregrine falcon	Aves	Falconiformes	Falconidae	Falco	peregrinus	N	
Quail sp.	Aves	Galliformes	Phasianidae	NA	NA	N	
Red-tailed Hawk	Aves	Falconiformes	Accipitridae	Buteo	jamaicensis	N	
Red winged blackbird	Aves	Passeriformes	Icteridae	Agelaius	phoeniceus	N	
Ring-necked pheasant	Aves	Galliformes	Phasianidae	Phasianus	colchicus	N	
Ring dove	Aves	Columbiformes	Columbidae	Streptopelia	risoria	N	
Spotted sandpiper	Aves	Charadriiformes	Scolopacidae	Actitis	macularia	N	
Tern sp.	Aves	Charadriiformes	Laridae,Sterninae	NA	NA	N	
Turkey	Aves	Galliformes	Phasianidae	Meleagris	gallopavo	N	
Black-tailed jackrabbit	Mammalia	Lagomorpha	Leporidae	Lepus	californicus	N	
Cat	Mammalia	Carnivora	Felidae	Felis	domesticus	N	
Cow	Mammalia	Artiodactyla	Bovidae	Bos	taurus	N	
Deer Mouse	Mammalia	Rodentia	Muridae	Peromyscus	maniculata	Y	
Dog	Mammalia	Carnivora	Canidae	Canis	familiaris	N	
Ferret	Mammalia	Carnivora	Mustelidae	Mustela	sp.	N	
Goat	Mammalia	Artiodactyla	Bovidae	Capra	hircus	N	
Grazer	Mammalia	Artiodactyla	NA	NA	NA	N	
Guinea pig	Mammalia	Rodentia	Caviae	Cavia	porcellus	N	
Hamster	Mammalia	Rodentia	Muridae	Cricetus	cricetus	N	
Kit Fox	Mammalia	Carnivora	Canidae	Vulpes	macrotis	N	
Least chipmunk	Mammalia	Rodentia	Sciuridae	Eutamias	minimus	N	
Long-tailed vole	Mammalia	Rodentia	Muridae	Microtus	longicaudus	N	
Mink	Mammalia	Carnivora	Mustelidae	Mustela	vision	N	
Mouse (lab)	Mammalia	Rodentia	Muridae	Mus	musculus	N	
Mule Deer	Mammalia	Artiodactyla	Cervidae	Odocoileus	hemionus	Y	
Pig	Mammalia	Artiodactyla	Suidae	Sus	scrofa	N	
Pocket Gopher	Mammalia	Rodentia	Geomyidae	Thomomys	bottae	Y	
Rabbit	Mammalia	Lagomorpha	Leporidae	Lepus	cuniculus	N	
Raccoon	Mammalia	Carnivora	Procyonidae	Procyon	lotor	N	
Rat, lab	Mammalia	Rodentia	Muridae	Rattus	norvegicus	N	
Red fox	Mammalia	Carnivora	Canidae	Vulpes	fulva	N	
Sheep	Mammalia	Artiodactyla	Bovidae	Ovis	aries	N	
Short-tailed shrew	Mammalia	Insectivora	Soricidae	Blarina	brevicauda	N	
Western Harvest Mouse	Mammalia	Rodentia	Muridae	Reithrodontomys	megalotis	N	

[a]N is not key receptor.

[b]Y means key receptor.

[c]NA means not applicable.

Sources: Udvardy, 1977; Palmer and Fowler, 1975; Burt and Grossenheider, 1980; Peterson Field Guides:Mammals; Fieldbook of Natural History; Audubon Society: Field Guide to North American Birds, Western Region.

- Toxicity data
- Contaminant uptake and bioavailability
- Contaminant interactions
- Surface water risk analysis
- Inhalation risk analysis

6.2.4.2.1 ***Derivation of Exposure Point Concentrations.*** Exposure point concentrations were derived from either the upper 95th percent confidence limit on analyte arithmetic means or the detected value in the case of a single detect.

Uncertainty exists because of the small number of analyses available for some of the potential contaminants. All concentration values utilized to calculate intakes are represented as point estimates, even though the contamination may be subject to both spatial and temporal variability. As noted, in some cases, insufficient data points were available to perform statistical analyses of the information. In these cases, the maximum value detected, which may have been the sole value, was used for the media concentration value. As a result, the hazards calculated may or may not be conservative.

6.2.4.2.2 ***Selection of Ecological Receptors.*** Receptors were selected to be representative of all species at SWMUs 13 and 17. The receptors include the deer mouse, which would be in regular contact with soil contaminants while burrowing. The golden eagle was chosen as a special status species (protected under the Eagle Protection Act) and as a receptor by virtue of its position at the top of the food chain and thus being exposed to bioaccumulative chemicals. Uncertainty is introduced using key receptors because it is unknown whether or not the appropriately exposed taxa were selected, which can influence the risk hazard results in either direction.

6.2.4.2.3 ***Toxicity Data.*** There is uncertainty in the toxicity values used to represent TBVs. In general, the TBV was the lowest of the NOAELs obtained from ecotoxicology literature. Although this is an accepted conservative approach, actual adverse health effects may not occur at the chosen intake. There is considerable variation between analyte/receptor-specific protective levels in the literature, which may indicate that hazards calculated using the TBVs may or may not be conservative.

6.2.4.2.4 ***Contaminant Uptake and Bioavailability.*** Bioaccumulation factors were used in calculation of hazards attributable to certain metal contaminants in receptor diet. These BAFs represent ingested tissue concentration relative to soil concentration. The BAFs were calculated from TEAD-N soils and biota samples. The actual bioavailability of the contaminants in TEAD-S soils is unknown, and available values vary widely, which may influence calculations of hazard values in either direction.

6.2.4.2.5 ***Contaminant Interactions.*** The contaminant interactions are unknown but can be synergistic, antagonistic, additive, or nonexistent. HIs are derived by adding chemical-specific and pathway-specific HQs for each receptor. Therefore, the presence of multiple COPCs of varying chemical groups may produce underestimation or overestimation of risks.

6.2.4.2.6 ***Surface Water Risk Analysis.*** The calculation of risks due to surface water ingestion may be conservative. Utilization of the sources of water may be intermittent, so that assumptions of continuous usage may lead to an overestimation of risk. The wastewater lagoons are fenced, which may prevent usage by mule deer and other large animals. Further, animals habituated to the desert may not use any surface water, instead fulfilling their requirements from dew or plant moisture. AUFs were not applied to calculation of water intakes, assuming that multiple home ranges may overlap at a water source, which could cause an underestimation of risk. As a result, uncertainty exists in either direction.

6.2.4.2.7 ***Air Inhalation Risk Analysis.*** Air exposure concentrations were calculated using a very conservative method, which assumes that the deer mouse is exposed 100 percent of the time to the burrow air. It is assumed that the burrow atmosphere is in equilibrium with the calculated soil pore space. Further, the model does not calculate burrow volume nor assume velocity of air movement or transfer rates. All of these factors tend to result in a high calculated inhalation dose.