

## 7. BASELINE RISK ASSESSMENT METHODOLOGY

The baseline risk assessment developed for WAG 5 (Holdren et al. 1999) evaluated the risk potential associated with contaminated media at ARA and PBF. The evaluation simulated a no action alternative, meaning that mitigative measures to reduce risk were not considered. The methodologies implemented to evaluate the baseline human health and ecological risks are outlined below, followed by a summary of the results for WAG 5. Components of the risk assessment specific to the selected remedies, such as contaminants of concern, contaminant concentrations, and risk estimates, are presented in more detail in Sections 8, 9, and 10.

### 7.1 Human Health Risk Evaluation Summary

The human health risk assessment approach used in the WAG 5 baseline risk assessment (BRA) was based on the EPA *Risk Assessment Guidance for Superfund* (RAGS) (EPA 1989, 1992a), INEEL Track 2 guidance (DOE-ID 1994), and INEEL cumulative risk assessment guidance protocol (LMITCO 1995). The tasks associated with development of the WAG 5 human health risk assessment included the following:

- Data evaluation
- Exposure assessment
- Toxicity assessment
- Risk characterization
- Qualitative uncertainty analysis.

These tasks are described in the subsections below.

#### 7.1.1 Data Evaluation

Data evaluation tasks that were completed as part of the BRA included site and contaminant screening and development of data sets for use in the risk assessment.

The site screening consisted of a review of previous risk assessments conducted for WAG 5 sites identified in the FFA/CO. As a result of the site screening, 15 of the individual sites identified in the FFA/CO were retained for quantitative risk assessment in the comprehensive BRA. The remaining sites either exhibited no risk potential (e.g., the site had no source of contamination) or a risk potential sufficiently below threshold values to preclude a significant contribution to cumulative risk. Individual sites with risk estimates greater than  $1E-07$  or hazard indices greater than 0.1 were retained.

In addition, because past and present activities associated with ARA and PBF facilities and structures are proximal or “co-located” to WAG 5 CERCLA sites, an analysis was performed to assess their potential impacts to cumulative risk estimates and to ensure that all historical releases were identified and assessed. The analysis included a review of past and present operational activities at ARA and PBF, existing facilities and structures, and management control procedures for mitigating the effects of future environmental releases of contaminants. Buildings and structures with a history of releases not subject to current management controls and those that possess the potential to impact cumulative risk at WAG 5 sites normally would be retained for consideration in the BRA. However, no such facilities or structures were identified in the facilities assessment analysis for WAG 5.

Contaminant screening consisted of comparing detected concentrations to INEEL background concentrations (Rood, Harris, and White 1996) and EPA 1E-06 risk-based concentrations (EPA 1995) for the most sensitive exposure pathway. Those contaminants that exceeded the screening criteria were identified as contaminants of potential concern and retained for quantitative analysis in the BRA. Potential exposure routes also were identified in conjunction with the contaminant screening.

All sampling data collected at WAG 5 sites were evaluated to determine whether the data were appropriate and adequate for use in the BRA. This evaluation was conducted generally in accordance with EPA guidance (EPA 1992a). As part of this analysis, sampling data sets were assumed to have lognormal distributions in accordance with EPA guidance on calculating concentration terms (EPA 1992a). However, true statistical distributions for the data were not determined. To calculate upper confidence limits on the means (UCLs), zero concentrations were assumed for all sampling results below minimum detection limits. The recommended method by the EPA to calculate upper confidence limits is to assign a value of one-half the detection limit for a sample result below the detection limit. However, this methodology was not used in the BRA because detection limits were not available for all of the sampling analyses. Assigning a zero value to all concentrations below detection limits allowed the upper confidence limits to be calculated consistently for all of the sampling results.

### **7.1.2 Exposure Assessment**

The process of exposure assessment quantifies the receptor intake of contaminants of potential concern for those exposure pathways with a potential to cause adverse effects. The assessment consists of estimating the magnitude, frequency, duration, and exposure route of contaminants to receptors. The following exposure assessment characteristics were identified:

- Exposed populations
- Complete exposure pathways
- Contaminant concentrations at the points of exposure for the complete exposure pathways
- Intake rates
- Intake factors.

The land-use assumptions and projections discussed in Section 6 were used to identify exposure scenarios, pathways, and routes. The exposure scenarios and default soil depths evaluated in the WAG 5 BRA are given in Table 2. The associated populations and exposure pathways are listed below and illustrated in Figures 4 and 5.

- Exposure scenarios
  - Occupational
  - Residential intrusion
- Exposure pathways
  - Groundwater pathway
  - Air pathway
  - Soil pathway

**Table 2.** Exposure scenarios and soil depths used in the WAG 5 baseline risk assessment.

Potentially Exposed Receptor	Land Use Scenario	Evaluated Exposure Pathways and Soil Depths
Occupational worker	Current industrial	Inhalation of volatiles (0–15 cm [0–0.5 ft]) <sup>a</sup> Inhalation of fugitive dust (0–15 cm [0–0.5 ft]) <sup>a</sup> Ingestion of surface soil (0–15 cm [0–0.5 ft]) <sup>a</sup> External radiation (0–1.22 m [0–4 ft]) <sup>b</sup>
Residential	Future residential	Inhalation of volatiles (0–3.05 m [0–10 ft]) <sup>c</sup> Inhalation of fugitive dust (0–3.05 m [0–10 ft]) <sup>c</sup> Ingestion of surface soil (0–3.05 m [0–10 ft]) <sup>c</sup> Ingestion of homegrown produce (0–3.05 m [0–10 ft]) <sup>c</sup> Ingestion of groundwater External radiation (0–3.05 m [0–10 ft]) <sup>c</sup>
Occupational worker	Future industrial	Inhalation of volatiles (0–15 cm [0–0.5 ft]) <sup>a</sup> Inhalation of fugitive dust (0–15 cm [0–0.5 ft]) <sup>a</sup> Ingestion of surface soil (0–15 cm [0–0.5 ft]) <sup>a</sup> External radiation (0–1.22 m [0–4 ft]) <sup>b</sup>

a. Exposure assessment considered the surface soil, defined as the top 0 to 15 cm (0 to 0.5 ft).

b. Exposure assessment considered the 0 to 1.22-m (0 to 4-ft) interval for undisturbed soil. Contamination below that depth is shielded by the topsoil.

c. Exposure assessment considered contamination within the 0 to 3.05-m (0 to 10-ft) interval because of the excavation required for a hypothetical basement.

- Exposure routes
  - Soil ingestion
  - Inhalation of fugitive dust
  - Inhalation of volatiles
  - External radiation exposure
  - Dermal absorption from soil (arsenic only)
  - Groundwater ingestion (residential scenario only)
  - Ingestion of homegrown produce (residential scenario only)
  - Dermal absorption of contaminants in groundwater (residential scenario only)
  - Inhalation of volatiles from indoor use of groundwater (residential scenario only).

Contaminant concentrations at the points of exposure for complete exposure pathways were based on detected concentrations as described in Section 7.1.1. If sufficient data were not available for calculating upper confidence limit concentrations, the maximum detected concentration was used. For radioactive contaminants, radioactive decay was incorporated into the intake calculations. Otherwise, no degradation mechanisms for reducing the toxicity of contaminants were considered.

Groundwater fate and transport modeling was used to predict the maximum contaminant concentrations that could occur in the aquifer from leaching and transport of nonradionuclide and radionuclide contaminants from WAG 5. The GWSCREEN model was used to simulate the potential release of contaminants from the release sites and the transport of the contaminants through the vadose zone to the aquifer. The maximum 30-year average groundwater concentration for each contaminant of potential concern was estimated at 100 and 1,000 years in the future, and at the time of maximum contaminant concentration up to 10,000 years (Holdren et al. 1999).

To calculate intake rates, default intake factors from EPA guidance (EPA 1989, 1991, and 1992a) and Track 2 guidance for the INEEL (DOE-ID 1994) were used. In conjunction with conversion factors and site-specific contaminant concentrations, these values were used to calculate contaminant intakes used in the risk calculations. The specific exposure parameters used for each receptor and exposure pathway are given in the RI/FS (Holdren et al. 1999, Appendix B). Generally, occupational scenarios simulate worker exposures for 8 hours/day, 250 days/year for 25 years and residential scenarios simulate exposures for 24 hours/day, 350 days/year, for 30 years. Standard values were used to simulate the human body (e.g., mass, skin area, inhalation rates, and soil ingestion rates).

To satisfy the objective of the WAG 5 comprehensive risk assessment, risks produced through the air and groundwater exposure pathways were analyzed cumulatively. Cumulative risks were estimated by calculating one risk number for each contaminant of potential concern in each air and groundwater exposure route (e.g., inhalation of fugitive dust and ingestion of groundwater) for each collection of sites in close proximity to one another. Analyzing the risks for the air and groundwater pathways in a cumulative manner is necessary because contamination from all sites within an area can contribute to local air and groundwater contaminant concentrations. Conversely, individual sites within a WAG are typically isolated from one another relative to the soil pathway exposure routes (e.g., external exposure and ingestion of soil). As a result, site-specific soil pathway exposures were analyzed. Generally, however, the BRA is comprehensive because risks are evaluated from all known and potential sites within WAG 5, and it is cumulative because risks from multiple sites are evaluated in the air and groundwater exposure pathways.

### **7.1.3 Conduct Toxicity Assessment**

Toxicity assessment is the process of characterizing the relationship between the intake of a substance and the incidence of an adverse health effect in the exposed population. Toxicity assessments evaluate the results from studies with laboratory animals or from human epidemiological studies. These evaluations are used to extrapolate from high levels of exposure, for which adverse effects are known to occur, to low levels of environmental exposures, for which effects can be postulated. The results of these extrapolations are used to establish quantitative indicators of toxicity.

Health risks from all routes of exposure are characterized by combining the chemical intake information with numerical indicators of toxicity (i.e., slope factors for carcinogens and reference doses for noncarcinogens). The toxicity constants that were used in the WAG 5 BRA were obtained from several sources. The primary source of information is the EPA online Integrated Risk Information System (IRIS). The IRIS database contains only those toxicity constants that have been verified by EPA work groups. The IRIS database is updated monthly and supersedes all other sources of toxicity information. If the necessary data are not available in IRIS, EPA Health Effects Assessment Summary Tables (HEAST) (EPA 1994a) are used. The toxicity constant tables are published annually and updated approximately twice per year. The HEAST contain a comprehensive listing of provisional risk assessment information that has been reviewed and accepted by individual EPA program offices, but has not had enough review to be recognized as high-quality, EPA-wide information (EPA 1994a).

Summaries of the toxicity profiles for the contaminants addressed in the selected remedies to mitigate unacceptable human health risk are given below.

**7.1.3.1 Arsenic.** Arsenic is classified as a metal. Acute exposure to arsenic causes severe throat irritation, gastrointestinal disturbance, and muscle spasms, which may be followed by vertigo, delirium, and coma. Facial edema also may be evident. Sensory loss and hematopoietic symptoms associated with acute exposure are usually reversible. Malaise and fatigue mark chronic exposure, either by ingestion or inhalation. Changes in the skin include hyperkeratosis. Anemia and neuropathy, liver injury, and “blackfoot disease” also result from chronic exposure.

Arsenic is a known carcinogen in humans. Ingestion is associated with increased incidence of skin cancer. Lung cancer results from inhalation. Insufficient data exist to determine the carcinogenic effects in animals.

The EPA oral slope factor for arsenic is  $1.8E+00$  (m/kg-day)<sup>-1</sup>, and the inhalation unit risk is  $2.4E-03$  (g/m<sup>3</sup>)<sup>-1</sup>. The confidence in the inhalation unit risk is somewhat uncertain because of the confounding variables in epidemiological studies and only one exposure dose was used in the animal studies. Confidence in the oral slope factor is relatively high because several studies show significant increases in the carcinogenic response.

**7.1.3.2 Lead.** Lead is classified as a metal. No critical effects of lead have been reported; however, many organs and systems are adversely affected by lead exposures. The major target organs and systems are the central nervous system, the peripheral nerves, the kidneys, the gastrointestinal system, and the blood system (Sittig 1985). Anemia is one of the early manifestations of lead poisoning. Other early effects of lead poisoning can include decreased physical fitness, fatigue, sleep disturbance, headache, aching bones and muscles, digestive symptoms, abdominal pains, and decreased appetite. The major central nervous system effects can include dullness, irritability, headaches, muscular tremors, inability to coordinate voluntary muscles, and loss of memory. The most sensitive effect for adults in the general population may be hypertension (Amdur, Doull, and Klaassen 1991).

Ingestion and inhalation of lead have the same effects on the human body. Large amounts of lead can result in severe convulsions, coma, delirium, and possibly death. A high incidence of residual damage, similar to that following infections or traumatic damage or injury, is observed from sustained exposure to lead. Most of the body burden of lead is in the bone (ATSDR 1990a). Lead effects in the peripheral nervous system are primarily manifested by weakness of the exterior muscles and sensory disturbances. Lead also has been shown to adversely affect sperm and damage other parts of the male reproductive system (ATSDR 1990a). Dermal absorption of inorganic lead compounds is reported to be much less significant than absorption by inhalation or oral routes of exposure (ATSDR 1990a).

The behavioral effects of lead exposure are a major concern, particularly in children. Exposure to lead can cause damage to the central nervous system, mental retardation, and hearing impairment in children. Levels of exposure that may have little or no effect on adults can produce important biochemical alterations in growing children that may be expressed as altered neuropsychological behavior (Martin 1991).

Though an ability of lead to cause cancer in humans has not been shown, the EPA has classified lead as a probable human carcinogen through both the ingestion and inhalation routes of exposure. Lead classification is based on the available evidence of cancer from animal studies. Rats ingesting lead demonstrated statistically increased incidence of kidney tumors (ATSDR 1990a). According to some epidemiological studies, lead workers developed cancer, but the data are considered inadequate to

demonstrate or refute the potential carcinogenicity of lead in humans. The EPA has not established toxicity values for lead.

**7.1.3.3 Polychlorinated Biphenyls.** Polychlorinated biphenyls (PCBs) comprise a physicochemically and toxicologically diverse group of 209 compounds. Their widespread use has made them ubiquitous in the environment. Aroclor-1242, which is contained in the sludge in the ARA-02 seepage pit, is a PCB.

Polychlorinated biphenyls are classified as probable human carcinogens. Data on carcinogenicity in humans following exposures to PCBs are inadequate because of confounding exposures or lack of exposure quantification (EPA 1993). Exposure to commercial PCB mixtures caused hepatocellular cancer in rats and mice, while most genotoxic and mutagenic bioassays with PCBs have been negative. The oral slope factor listed by the EPA in the IRIS database for PCBs is  $4.0E-01$  (mg/kg/day)<sup>-1</sup>.

Toxicity data for assessing the noncarcinogenic effects of Aroclor-1242 have not been approved. However, data for Aroclor-1254 were used to assess the potential toxicological effects. The oral reference dose for Aroclor-1254 in the IRIS database is  $2.0E-05$  (mg/kg/day)<sup>-1</sup>. Estimates developed using this reference dose for Aroclor-1242 are classified as qualitative.

The routes of entry of PCBs into the body are inhalation of fumes or vapors and percutaneous absorption of liquid, ingestion, and eye and skin contact.

Prolonged skin contact may cause the formation of sebaceous cysts, and pustules known as chloracne. Irritation of the eyes, nose, and throat also may occur. Acute and chronic exposure can cause liver damage.

**7.1.3.4 Cesium-137.** The radioactive isotope Cs-137 is a fission product of nuclear reactors and nuclear weapons detonations. The EPA classifies all radioactive substances as probable carcinogens in the IRIS database.

Cesium-137 is rapidly absorbed into the bloodstream and is distributed throughout the active tissues of the body. Metabolically, Cs-137 behaves as an analog of potassium. Its distribution throughout the body and energetic beta and gamma radiation from its daughter, Ba-137m, results in whole-body irradiation (Amdur, Doull, and Klaassen 1991). The radioactive half-life of Cs-137 is 30 years. Its biological half-life in adults is 50 to 150 days, and in children is 44 days. Cesium-137 exists in secular equilibrium with Ba-137m, which is the major contributor to the dose received from a 0.662-MeV gamma ray. The critical organ for Cs-137 exposure is the whole body.

**7.1.3.5 Radium-226.** Radium is a naturally occurring silvery white radioactive metal that can exist in several isotopes, and is formed by the decay of uranium and thorium in the environment. Radium-226 is a gamma emitter and has a 1,600-year half-life. The EPA classifies all radioactive substances as probable carcinogens in the IRIS database.

Exposure to radium is constant because it is present at very low levels in the surrounding environment. Exposure to higher levels of radium can occur to those who live in an area in which it is released into the air from the burning of coal or other fuels. Exposure also results if drinking water is taken from a source that is high in natural radium, such as a deep well or from a source near a disposal site.

No clear evidence indicates that long-term exposure to radium at the levels that are normally present in the environment is likely to result in harmful health effects. Exposure to higher levels of

radium over a long period of time may result in harmful effects including anemia, cataracts, fractured teeth, cancer (especially bone cancer), and death. Some of these effects may take years to develop (ATSDR 1990b).

**7.1.3.6 Silver-108m.** The information identified in a literature search to support the development of a toxicity profile for Ag-108m was very sparse (Holdren et al. 1999). Though radioactive silver has been administered as a tracer, information about the radiotoxicity of Ag-108m was not found. Silver-108m is not naturally occurring, has a half-life of 130 years (which was recently modified to 418 years [Firestone and Shirley 1999]), and is primarily a gamma emitter. The EPA classifies all radioactive substances as probable carcinogens in the IRIS database.

**7.1.3.7 Uranium-235 and -238.** Natural uranium contains three isotopes: U-234, U-235, and U-238. The abundance of each isotope in natural uranium is, respectively, 0.006%, 0.72%, and 99.27% (ATSDR 1990c). Uranium can be found in the earth's crust at an average concentration of 2 ppm. The ambient air concentration of uranium in the United States ranges from 0.3 to 0.011 fCi/m<sup>3</sup> (1 fCi = 1E-03 pCi). The concentration in drinking water ranges from 0.07 to 653 pCi/L with a median value of 0.1 to 0.2 pCi/L.

In natural uranium, the radioactivity from U-238 accounts for about half the total radioactivity and the radiation from U-234 and U-235 accounts for the other half. Uranium emits primarily alpha radiation that cannot penetrate skin but can travel short distances in the body if the uranium is inhaled or ingested. Because natural uranium emits very small amounts of gamma radiation that can penetrate the skin, little danger, if any, exists from this type of radiation from uranium (ATSDR 1990c). Moreover, no human or animal studies have definitively linked inhalation or oral exposure to natural uranium to the development of cancer. However, the EPA classifies all radioactive substances as probable carcinogens in the IRIS database.

For the noncarcinogenic health risks associated with uranium, exposure to natural concentrations of uranium in food, water, air, and soil does not appear to have any toxic effects. Animals that have had oral, inhalation, or dermal exposure to large amounts of uranium have developed damage to the kidney tubules, but other systems were not affected. The only significant systemic health risk in humans from exposure to nonenriched uranium is potential damage to the kidneys. However, an increase in deaths from urogenital or renal diseases has not been noted in epidemiological studies and significant damage to human kidneys following exposure to uranium has not been identified in intravenous studies (ATSDR 1990c). Overall, studies in animals and humans indicate that exposure to uranium is unlikely to produce immunological or neurological effects.

#### **7.1.4 Risk Characterization**

The characterization of risk involves combining the results of the toxicity and exposure assessments to estimate health risks. These estimates are either a comparison of exposure levels with appropriate toxicity criteria or an estimate of the lifetime cancer risk associated with a particular intake. The nature and weight of evidence supporting the risk estimate, as well as the magnitude of uncertainty surrounding the estimate, also are considered in risk assessment.

To quantify human health risks, contaminant intakes are calculated for each contaminant by way of each applicable exposure route. As discussed above, these contaminant intakes are calculated values based on measured concentration estimates. To estimate human health risks, the contaminant-specific intakes are compared to the applicable chemical-specific toxicity data. The complete results of the BRA risk characterization process, including risk estimates for each retained site and groundwater and air pathway risks for each collection of sites, are presented in the RI/FS report (Holdren et al. 1999,

Appendix B). The generalized equations for calculating carcinogenic risk and noncarcinogenic hazard quotients are given below.

**7.1.4.1 Carcinogenic Health Effects.** The following calculations are used to obtain numerical estimates (i.e., unitless probability) of lifetime cancer risks. The risk probability is the product of the intake and the slope factor, as follows:

$$Risk = Intake \times SF \quad (1)$$

where

*Risk* = Potential lifetime cancer risk (unitless)

*Intake* = Chemical intake (mg/kg/day), or radionuclide intake (pCi)

*SF* = Slope factor, for chemicals (mg/kg/day)<sup>-1</sup>, or radionuclides (pCi)<sup>-1</sup>.

The linear low-dose equation shown above is valid at risk levels lower than 1E-02 (1 in 100). In accordance with EPA guidance (EPA 1989), risks that are greater than 1E-02 (1 in 100) are calculated using the following one-hit equation:

$$Risk = 1 - \exp(-Intake \times SF) \quad (2)$$

where

*Risk* = Potential lifetime cancer risk (unitless)

*Intake* = Chemical intake (mg/kg/day), or radionuclide intake (pCi)

*SF* = Slope factor for chemicals (mg/kg/day)<sup>-1</sup> or radionuclides (pCi)<sup>-1</sup>.

To develop a total risk estimate for a given site, cancer risks are summed separately across all potential carcinogens at the site, as shown in the following calculation:

$$Risk_T = \sum Risk_i \quad (3)$$

where

*Risk<sub>T</sub>* = Total cancer risk, expressed as a unitless probability

*Risk<sub>i</sub>* = Risk estimate for the i<sup>th</sup> contaminant.

Similarly, risk values for each exposure route are summed to obtain the total cancer risk for each potential carcinogen.

**7.1.4.2 Noncarcinogenic Effects.** Health risks associated with exposure to individual noncarcinogenic compounds are evaluated by calculating hazard quotients. The hazard quotient is the ratio of the intake rate to the reference dose, as follows:

$$HQ = Intake / RfD \quad (4)$$

where

$HQ$  = Noncarcinogenic hazard quotient (unitless)

$Intake$  = Chemical intake (mg/kg/day)

$RfD$  = Reference dose (mg/kg/day).

Hazard indices are calculated by summing hazard quotients for each chemical across all exposure routes. If the hazard index for any contaminant of potential concern exceeds unity, potential health effects may be a concern from exposure to the contaminant of potential concern. The hazard index is calculated using the following equation:

$$HI = \sum \frac{Intake_i}{RfD_i} \quad (5)$$

where

$HI$  = Hazard index (unitless)

$Intake_i$  = Exposure level (intake) for the  $i^{\text{th}}$  toxicant (mg/kg/day)

$RfD_i$  = Reference dose for the  $i^{\text{th}}$  toxicant (mg/kg/day).

In the foregoing equation, intake and reference dose are expressed in the same units and represent the same exposure time period.

### 7.1.5 Qualitative Uncertainty Analysis

The risk assessment results are very dependent on the methodologies applied to develop the risk estimates. These analysis methods were developed over a period of several years by INEEL risk management and risk assessment professionals to provide realistic, yet conservative estimates of human health risks at WAG 5. Nonetheless, if different risk assessment methods had been used, the BRA likely would have produced different risk assessment results. To ensure that the risk estimates are conservative (i.e., generate upper-bound risk estimates), health protective assumptions that tend to bound the plausible upper limits of human health risks were applied throughout the BRA. Therefore, risk estimates that may be calculated by other risk assessment methods are not likely to be significantly higher than the estimates developed for the WAG 5 Comprehensive RI/FS.

Uncertainty in the BRA is produced by uncertainty factors in all four stages of risk analysis (i.e., data collection and evaluation, exposure assessment, toxicity assessment, and risk characterization). The uncertainties associated with parameters used in the risk assessment are listed in Table 3. The conservative assumptions and uncertainties in the risk estimates for the five sites identified for remediation based on human health risk estimates in the WAG 5 Comprehensive RI/FS (Holdren et al. 1999) are summarized in Table 4. Qualitative consideration of the collective impact of all the assumptions indicates that the risks are more likely to be overestimated than underestimated.

**Table 3.** Human health baseline risk assessment uncertainty factors.

Uncertainty Factor	Effect of Uncertainty	Comment
Source term assumptions	May overestimate risk.	All contaminants are assumed to be completely available for transportation away from the source zone. In reality, some contaminants may be chemically or physically bound to the source zone and unavailable for transport.
Natural infiltration rate	May overestimate risk.	A conservative value of 10 cm/year was used for this parameter.
Moisture content	May overestimate or underestimate risk.	Soil moisture contents vary seasonally in the upper vadose zone and may be subject to measurement error.
Water table fluctuations	May slightly overestimate or underestimate risk.	The average value used is expected to be representative of the depth over the 30-year exposure period.
The mass of contaminants in soil was estimated by assuming a uniform contamination concentration in the source zone.	May overestimate or underestimate risk.	While not likely, most of the mass of a given contaminant at a given site may exist in a hotspot that was not detected by sampling. Such a condition could result in underestimating the mass of the contaminant used in the analysis. Assigning zero values to concentrations below detection limits also may cause mass to be underestimated. However, the 95% upper confidence limit on the mean (UCL) or the maximum detected contamination levels were used for all mass calculations. These concentrations are assumed to exist at every point in each waste site; therefore, the mass of contaminants used in the analysis is probably overestimated.
Plug flow assumption in groundwater transport	May overestimate or underestimate risk.	Plug flow models such as GWSCREEN (Rood 1994) are conservative relative to concentrations because dispersion is neglected and mass fluxes from the source to the aquifer differ only by the time delay in the unsaturated zone (the magnitude of the flux remains unchanged). For nonradiological contaminants, the plug flow assumption is conservative because dispersion is not allowed to dilute the contaminant groundwater concentrations. For radionuclides, the plug flow assumption may or may not be conservative. Based on actual travel time, the radionuclide groundwater concentrations could be overestimated or underestimated because a longer travel time allows for more decay. If the concentration decrease from the travel time delay is larger than the neglected dilution from dispersion, the model will not be conservative.
No migration of contaminants from the soil source before 1994 was modeled.	May overestimate or underestimate risk.	The effect of not modeling contaminant migration from the soil before 1994 is dependent on the contaminant half-life, radioactive ingrowth, and mobility characteristics.
Chemical form assumptions	May overestimate or underestimate risk.	In general, the methods and inputs used in contaminant migration calculations, including assumptions about chemical forms of contaminants, were chosen to err on the protective side. All contaminant concentration and mass are assumed available for transport. This assumption results in a probable overestimate of risk.

**Table 3. (continued).**

Uncertainty Factor	Effect of Uncertainty	Comment
Exposure scenario assumptions	May overestimate risk.	<p>The likelihood of future scenarios has been qualitatively evaluated as follows:</p> <p>Resident—improbable</p> <p>Industrial—credible.</p> <p>The likelihood of future residential development at the INEEL is small. If future residential use of this site does not occur, then the risk estimates calculated for future residents are likely to overestimate the risk associated with future use of this site.</p>
Exposure parameter assumptions	May overestimate risk.	Assumptions about media intake, population characteristics, and exposure patterns may not characterize actual exposures.
Receptor locations	May overestimate risk.	Groundwater ingestion risks are calculated for a point at the downgradient edge of an equivalent rectangular area. The groundwater risk at this point is assumed to be the risk from groundwater ingestion at every point within WAG 5 boundaries. Changing the receptor location will affect only the risks calculated for the groundwater pathway because all other risks are site specific or assumed constant at every point within the WAG 5 boundaries.
For the groundwater pathway analysis, homogeneous distribution in a large mass of soil was assumed for all contaminants.	May overestimate or underestimate risk.	Homogeneous distribution in the soil volume beneath WAG 5 is assumed for the total mass of each contaminant of potential concern. This assumption tends to maximize the estimated groundwater concentrations produced by the contaminant inventories because homogeneously distributed contaminants would not have to travel far to reach a groundwater well drilled anywhere within the WAG 5 boundary. However, groundwater concentrations may be underestimated for a large mass of contamination (located in a small area with a groundwater well drilled directly downgradient).
The entire inventory of each contaminant was assumed to be available for transport along each pathway.	May overestimate risk.	Only a portion of the inventory of each contaminant will be transported by each pathway.
Exposure duration	May overestimate risk.	The assumption that an individual will work or reside at a site for 25 or 30 years is conservative. Short-term exposures involve comparison to subchronic toxicity values, which are generally less restrictive than chronic values.
Conservative values were used to represent constants not dependent on contaminant properties.	May overestimate risk.	Conservative or upper-bound values were used for all parameters incorporated into intake calculations.
Some hypothetical pathways were excluded from the exposure scenarios.	May underestimate risk.	Exposure pathways are considered for each scenario and eliminated only if the pathway is either incomplete or negligible compared to other evaluated pathways.
Biotic decay was not considered.	May overestimate risk.	Biotic decay would tend to reduce contamination over time.

**Table 3.** (continued).

Uncertainty Factor	Effect of Uncertainty	Comment
Occupational intake value for inhalation is conservative.	May slightly overestimate risk.	Standard exposure factors for inhalation have the same value for occupational as for residential scenarios though occupational workers would not be onsite all day.
Use of cancer slope factors	May overestimate risk.	Slope factors are associated with 95% UCLs. They are considered unlikely to underestimate risk.
Toxicity values were derived primarily from animal studies for nonradioactive contaminants.	May overestimate or underestimate risk.	Extrapolation from animal to humans may induce error from differences in absorption, pharmacokinetics, target organs, enzymes, and population variability.
Toxicity values were derived primarily from high doses; however, most exposures are at low doses.	May overestimate or underestimate risk.	Linearity was assumed at low doses. The effect tends toward conservative exposure assumptions.
Toxicity values and classification of carcinogens	May overestimate or underestimate risk.	Not all values represent the same degree of certainty. All are subject to change as new evidence becomes available.
Lack of slope factors	May underestimate risk.	Contaminants of potential concern without slope factors may or may not be carcinogenic through the oral pathway.

**Table 4.** Summary of site-specific uncertainties and conservative assumptions for the human health baseline risk assessment.

Site	Uncertainties and Conservative Assumptions
ARA-12: ARA-III Radioactive Waste Leach Pond	<p>The 95% upper confidence limit (UCL) or maximum contaminant concentrations are assumed to exist over the entire site. This conservative assumption would probably lead to an overestimation of risk.</p> <p>Sampling was performed to the soil/basalt interface at depths of up to 7 ft. The residential scenario risks were calculated assuming that all 7 ft of soil would be excavated. This assumption may result in an overestimate of risk.</p> <p>In the absence of historical disposal data, the contaminant masses associated with the site were estimated based on source term volume and detected concentrations. This approach may result in an underestimate of risk.</p> <p>The hotspot detected during the global positioning radiometric scanner (GPRS) survey was not sampled. Therefore, risk was underestimated for ARA-12.</p> <p>Analytical results to confirm the GPRS data were not available for the baseline risk assessment. For the baseline risk assessment, the GPRS data were converted using the assumption that the elevated gamma radiation was caused by Cs-137. However, analytical results received after the publication of the Proposed Plan (DOE-ID 1999b) indicate that Ag-108m is the contaminant responsible for the elevated gamma reading. Therefore, Cs-137 risks for this site are overestimated. The risks from Ag-108m are not underestimated, however, because the maximum concentration detected in 1993 and used in the baseline risk assessment is higher than the recently detected maximum concentration.</p>
ARA-23: Radiologically Contaminated Soils and Subsurface Structures in and Around ARA-I and ARA-II	<p>Three aspects of the ARA-23 radionuclide source term calculations impact the results of the site risk assessment. First, the 95% UCL or maximum contaminant concentrations were assumed to exist over the nearly 170,000-m<sup>2</sup> site area. The true contaminant soil concentrations may be less than the 95% UCL or maximum detected concentrations over much of the site. Therefore, this assumption may result in an overestimate of the risk for the site.</p> <p>Second, the GPRS survey (see Section 4.2.1.6) indicated that an area of Cs-137 contamination was not considered during the calculation of the average Cs-137 concentration for the site. The GPRS survey was used to identify the 10-pCi/g Cs-137 isopleth for the site, and the Cs-137 samples evaluated in the baseline risk assessment were collected at this isopleth. The survey indicated high levels of contamination within this isopleth, but soil samples were not collected to verify this indication. Omission of the contamination within the isopleth probably produced an underestimation of the site average Cs-137 concentration and a corresponding underestimation of the site risk.</p> <p>Sampling at the site was performed down to a depth of only 2 ft. Contamination was detected at this depth; therefore, the conservative assumption that contamination extended all the way to a depth of 2 ft below ground surface was incorporated into the risk assessment. Because the transport mechanism operative at this site is windblown deposition, the contamination is probably concentrated in the top few inches of surface soil. Therefore, this assumption may result in an overestimate of risk for the site.</p>

**Table 4. (continued).**

Site	Uncertainties and Conservative Assumptions
ARA-25: Soils Beneath the ARA-626 Hot Cells	<p>The 95% UCL or maximum contaminant concentrations were assumed to exist over the entire site. This conservative assumption would probably lead to an overestimation of risk.</p> <p>Sampling was performed to a depth of 0.5 ft. For the risk assessment, homogeneous contaminant concentrations were assumed for the entire soil interval to the soil/basalt interface at an estimated depth of 5 ft. This assumption may overestimate the risk.</p> <p>In the absence of quantified release data, the contaminant masses associated with the site were estimated based on estimated source term volume and detected concentrations. This approach may underestimate the risk.</p>
ARA-02: ARA-I Sanitary Waste Leach Field and Seepage Pit	<p>The source terms specific to the two sources associated with this site (i.e., the seepage pit and the septic tank soil) were assumed to exist over the entire surface of the two separate areas of the site. This conservative assumption probably causes an overestimation of the calculated risks at the site. However, the pipeline between the seepage pit and the septic tanks is assumed intact, which may underestimate the source term and the resultant risks.</p> <p>No attempt was made to estimate the amount of contamination that may have been released to the subsurface over the operational lifetime of the seepage pit. Only the current concentrations in the existing sludge were evaluated. Therefore, risks associated with past releases from the seepage pit are underestimated.</p> <p>For the seepage pit evaluation, data from the seepage pit sludge were combined with the soil samples outside the pit for risk assessment purposes. The combining of the data overestimates the risk for the types of contaminants found outside the tank when only the soil sample data are used.</p>
ARA-16: ARA-I Radionuclide Tank	<p>The 95% UCL or maximum contaminant concentrations were assumed to exist over the entire surface of the site. The site area (660 ft<sup>2</sup>) was conservatively assumed to equal the area of the grid for the 1997 sampling. This assumption may overestimate the risks for the site.</p> <p>The contents of the tank were not considered in the risk assessment because no evidence of release from the tank was observed. This approach may underestimate the risk.</p>

## 7.2 Ecological Risk Evaluation Summary

The WAG 5 ecological risk assessment (ERA) is a component of the phased approach developed for ERA at the INEEL. The results of the WAG 5 ERA will be integrated into an INEEL-wide evaluation of potential risks to ecological receptors as a component of the WAG 10 ERA. The ERA was conducted as outlined in the guidance for the INEEL (VanHorn, Hampton, and Morris 1995).

An ecological site and contaminant screening was conducted to determine which sites and contaminants would be subjected to further analysis in the WAG 5 Comprehensive RI/FS. The screening was completed and documented as part of the WAG 5 Work Plan (DOE-ID 1997a). A site-by-site evaluation of the risks to ecological resources as a result of exposure to contaminants at WAG 5 was developed in the RI/FS. The evaluation included a review of the screening completed in the Work Plan to ensure that sites or contaminants were not inappropriately omitted from further evaluation. Complete details of the WAG 5 ERA are presented in Sections 7 and 8 of the WAG 5 Comprehensive RI/FS report (Holdren et al. 1999). The primary components of the WAG 5 ERA, discussed below, include problem formulation, analysis, risk characterization, and transition to the INEEL-wide ERA.

**7.2.1.1 Problem Formulation.** The goal of the problem formulation step is to investigate the interactions between the stressor characteristics (i.e., contaminant characteristics), the ecosystem potentially at risk, and the potential ecological effects (EPA 1992b). Site screening was conducted to identify the sites that could pose unacceptable risk. Of the 55 sites in WAG 5, 16 were retained for quantitative evaluation in the ERA.

Contaminant screening and data evaluation were conducted to identify contaminants of potential concern and define exposure point concentrations. For the most part, the results of the data evaluation conducted for the human health BRA (see Section 7.1) were applied to the ERA. For those contaminants that were not retained for evaluation in the human health risk assessment, additional data evaluation to support the completion of the ERA was performed. Contaminant concentrations were compared to background concentrations and ecologically based screening levels. All radioactive contaminants were eliminated on the basis of this comparison.

Site-specific data characterizing contaminant concentration in biota for the INEEL ERAs are sparse. Consequently, the definition of assessment and measurement endpoints (i.e., ecological receptors) is based primarily on pathway and exposure analyses. Pathway and exposure models for contaminated surface and subsurface media (Figures 7 and 8) were combined with a food web analysis to characterize the potential risks illustrated in the ERA conceptual site model (see Figure 6).

**7.2.1.2 Analysis.** In the analysis component of the ERA, the likelihood and significance of an adverse reaction from exposure to stressors were evaluated. The exposure assessment involves relating contaminant migration to exposure pathways for ecological receptors. The behavior and fate of contaminants of potential concern in the terrestrial environment were presented in a general manner because formal fate and transport modeling was not conducted for the WAG ERA (Holdren et al. 1999). The ecological effects assessment consisted of a hazard evaluation and a dose-response assessment. The hazard evaluation involved a comprehensive review of toxicity data for contaminants to identify the nature and severity of toxic properties. The dose from multiple media (surface and subsurface soil) identified at WAG 5 was developed and used to assess the potential risk to receptors. Because dose-based toxicological criteria exist for few ecological receptors, development of appropriate toxicity reference values (TRVs) was necessary for the contaminants and functional groups at the INEEL. A semi-quantitative analysis was used, augmented by qualitative information and professional judgment as necessary.

Exposures for each functional group, threatened or endangered species, and sensitive species were estimated based on site-specific life history and, when possible, feeding habits. Quantification of group and individual exposures incorporated species-specific numerical exposure factors including body weight, ingestion rate, and the fraction of diet composed of vegetation or prey and soil consumed from the affected area. Parameters used to model contaminant intakes by the functional groups were derived from a combination of parameters that produced the most conservative overall exposure for the group. Parameter values and associated information sources are discussed in further detail in the WAG 5 RI/FS (Holdren et al. 1999, Appendix I). The development of the TRVs for those contaminants targeted for remediation based on unacceptable ecological risks is described below.

**7.2.1.2.1 Copper**—Copper is one of the least mobile of the trace elements and tends to be uniformly distributed in the soil horizon. Copper is one of seven essential plant micronutrients. Extensively complexed by humic materials, copper is readily available to plants when the soil pH is below 6, especially in soil with low organic matter and humic material content. Plants uptake is limited in soil with large amounts of organic matter. The recommended screening benchmark concentration for phytotoxicity in soil for copper of 100 mg/kg was used as the TRV for terrestrial plants (Will and Suter 1995).

Copper is an essential element for the normal function of several critical enzymes and the use of iron in animals. Copper deficiency is, therefore, usually a greater health concern than copper excess, though severe poisoning can result from the salt form. Depressed food intake, body-weight gain, egg number and weight, and organ weights are associated with copper excess in poultry (Stevenson and Jackson 1981). A no observed adverse effect level of 24 mg/kg/day was identified and used to develop TRVs for avian functional groups.

High doses of copper have caused liver, kidney, and stomach damage and anemia in a number of mammalian species. A quantified critical exposure of 66 mg/kg/day (a no observed adverse effect level) identified from a study of effects on rats and mice was used to develop mammalian TRVs. A mammalian TRV also was derived from a chronic feeding study in mink to determine growth and survival effects (Aulerich et al. 1982).

**7.2.1.2.2 Lead**—Lead is a ubiquitous trace constituent in rocks, soil, plants, water, and air. Lead is neither essential nor beneficial to living organisms. For plants, the recommended screening benchmark concentration for phytotoxicity in soil for lead of 50 mg/kg was used as the TRV for terrestrial plants (Suter, Will, and Evans 1993).

In birds and animals, lead affects the kidneys, blood, bone, and the central nervous system. Ingestion of lead shot is a significant cause of mortality among waterfowl that are partially or completely protected by law. Lead toxicity varies widely with the form and dose of administered lead. Generally, organic compounds are more toxic than inorganic compounds. For avian herbivores, a TRV was estimated using a study of mallards (Dieter and Finley 1978). The results of studies of avian insectivores (Eisler 1988), European starlings (Osborn, Eney, and Bull 1983), and American kestrels (*Falco sparverius*) (Colle et al. 1980) were used to develop TRVs for avian functional groups. Studies of rats administered lead in drinking water (Kimmel et al. 1980), lead toxicity of calves (Zmudzki et al. 1983), and lead toxicity of dogs (DeMayo et al. 1982) were used to develop TRVs for mammalian receptors.

**7.2.1.2.3 Mercury**—Mercury exists in the environment in three oxidation states. Because speciation is a major determinant of the fate, bioavailability, absorption, and toxicological characteristics of mercury compounds, lack of knowledge of the state of the mercury in INEEL soil is a large source of uncertainty in both exposure assessment and TRV development. The organic forms of mercury are generally more toxic but are unlikely to persist in the environment. However, toxic organic mercury may

form in biotic tissues and are known to biomagnify through ecosystems (Wren 1986; Scheuhammer 1987). Therefore, TRVs were developed from studies of the toxic effects of organic mercury to ensure that the TRVs are protective. This measure is highly conservative and tends to result in an overestimate of risks for receptors lower in the food web because the majority of mercury in soil and plants (i.e., the majority of exposures to plants and soil-dwelling and herbivorous animals) is inorganic. A TRV of 0.3 mg/kg was assigned for mercury for terrestrial plants based on the toxicological benchmark (Suter, Will, and Evans 1993).

Mercury exposure affects the central nervous system in both mammals and birds. Reproductive effects from lower doses have been observed. For herbivores, the effects of organic mercury compounds on galliformes (e.g., domestic chickens, quail, and pheasants) have been investigated by several groups. However, no study was reviewed that identified a no observed adverse effect level. The lowest observed adverse effect level for relevant endpoints (i.e., reproductive success) of several similar studies was found in a study of the effects of mercury on birds (Fimreite 1979). An avian TRV was derived from this study. Two studies examined the effects of mercury exposure on the reproductive competence of male and female rats (Khera and Tabacova 1973; Khera 1973). The no observed adverse effect level identified for both sexes was 0.25 mg/kg/day.

Much less information is available about mercury toxicity to herbivores. In a study of acute toxicity in mule deer (*Odocoileus hemionus*), 17.88 mg/kg was said to be the lethal dose of 50% of the exposed organisms (Eisler 1987). A number of studies have examined the effects of chronic ingestion on carnivorous mammals, particularly cats (e.g., Albanus et al. 1972; Charbonneau et al. 1976; Eaton, Secord, and Hewitt 1980) and mink (e.g., Aulerich, Ringer, and Iwamoto 1974; Wobeser, Neilson, and Schiefer 1976; Wren et al. 1987; Charbonneau et al. 1976). Results from these studies were used to develop mammalian TRVs.

**7.2.1.2.4 Selenium**—Selenium is a critical nutrient and component of several enzymes. Often selenium is found in high concentrations in areas in which soil has been derived from Cretaceous rocks (Eisler 1985). The recommended screening benchmark concentration for phytotoxicity in soil for selenium of 1 mg/kg was used as the TRV for terrestrial plants (Will and Suter 1995).

In animals, selenium deficiency is generally a greater threat to health than selenium poisoning (Eisler 1985). Selenium deficiency has been documented in a variety of species including fish, quail, ducks, poultry, rats, dogs, domestic grazing animals, antelope, monkeys, and humans (Eisler 1985). Selenium also can reduce the toxicity of other heavy metals such as thallium, arsenic, and copper (Wilber 1980).

Selenium has been reported to cause reproductive impacts such as growth retardation and decreased fertility, especially in birds (TOXNET 1994). Malformations in chickens and waterfowl have been widely reported (EPA 1993). The effects of sodium selenite in chickens, mallards, and black-crowned night herons were evaluated in studies by Ort and Latshaw (1978), Heinz et al. (1987), and Smith et al. (1988) to derive TRVs for avian receptors.

Selenium accumulates to high concentrations in certain species of plants (Eisler 1985). Livestock species ingesting these plants have been reported to exhibit toxic symptoms. Prolonged exposure to more moderate levels of selenium also results in deleterious effects (TOXNET 1994). In a study of the effects of selenium on rats (Rosenfeld and Beath 1954), selenium did not affect reproduction. Because no effect on growth in rats has been reported (Halverson, Palmer, and Guss 1966), a reproductive endpoint was selected to develop a TRV. Selenium doses as low as 3.2 mg/kg body weight have resulted in death in sheep (Eisler 1985). A TRV was developed for mammalian herbivores using these data.

**7.2.1.2.5 Thallium**—Thallium is a nonvolatile heavy metal element that is not used extensively by industry and is mainly introduced into the environment as a waste product of other metals. Thallium may be bioconcentrated by living organisms (Callahan et al. 1979). Thallium adversely affects protein synthesis and inhibits a number of enzymes. Toxic to plants, thallium inhibits chlorophyll formation and seed germination. The recommended toxicological benchmark of 1 mg/kg for thallium was used as the TRV for terrestrial plants (Will and Suter 1995).

A study of the acute toxicity of thallium in game birds including quail (Shaw 1933) formed the basis for the TRV for avian functional groups. In a study of three immature golden eagles (*Aquila chrysaetos*), the acute oral lethal dose of 50% of the exposed organisms was estimated to be between 60 and 120 mg/kg (Bean and Hudson 1976). Using the lower end of this range as the quantified critical exposure, a TRV for raptorial birds at the INEEL was derived.

Thallium is slightly more toxic to mammals than mercury. Rats exposed to thallium have shown effects on various neurological (Manzo et al. 1992) and reproductive (Formigli et al. 1986) endpoints. Because of the clear ecological relevance of reproductive impairment, a quantified critical exposure was selected from the study of thallium-induced testicular toxicity (Formigli et al. 1986).

**7.2.1.3 Risk Characterization.** Risk characterization is the final step of the WAG 5 ERA process. The risk evaluation determines whether risk is indicated from the contaminant concentrations and the calculated dose for the INEEL functional groups, threatened or endangered species, and species of concern and considers the uncertainty inherent in the assessment. For a WAG ERA, the risk characterization step has two components: a description of the estimation of risk and a summary of the results.

Risk is estimated by comparing the calculated dose to the TRV. If the dose from the contaminant does not exceed its TRV (i.e., if the hazard quotient [HQ] is less than 1.0 for nonradiological contaminants), adverse effects to ecological receptors from exposure to that contaminant are not expected and no further evaluation of that contaminant is required. Hence, the HQ is an indicator of potential risk. Hazard quotients are calculated using the following equation:

$$HQ = \frac{Dose}{TRV} \quad (6)$$

where

$HQ$  = Hazard quotient (unitless)

$Dose$  = Dose from all media (mg/kg/day or pCi/g/day)

$TRV$  = Toxicity reference value (mg/kg/day or pCi/g/day).

Hazard quotients for WAG 5 were derived for all contaminants, functional groups, threatened or endangered species, and species of concern identified in WAG 5 for each site of concern. The largest observed HQ across all functional groups within WAG 5 varies by at least three orders of magnitude. When information is not available to derive a TRV, then an HQ cannot be developed for that particular contaminant and functional group or species combination.

An HQ greater than the threshold value of 1 indicates that exposure to a given contaminant, at the concentrations and for the duration and frequencies of exposure estimated in the exposure assessment, may cause adverse health effects in exposed populations. However, the level of concern associated with

exposure may not increase linearly as the HQ values exceed the threshold value. Therefore, the HQs cannot be used to represent a probability or a percentage because an HQ of 10 does not necessarily indicate that adverse effects are 10 times more likely to occur than an HQ of 1. It is only possible to infer that the greater the HQ, the greater the concern about potential adverse effects to ecological receptors.

In general, the significance of an HQ exceeding 1 depends on the perceived “value” (i.e., ecological, social, or political) of the receptor (or species represented by that receptor), the nature of the endpoint measured, and the degree of uncertainty associated with the process as a whole. Therefore, the decision to take no further action, order corrective action, or perform additional assessment must be determined on a site-, chemical-, and species-specific basis. With the exception of threatened or endangered species (EPA 1992b), the unit of concern in ERA is usually the population as opposed to the individual. Therefore, exceeding conservative screening criteria does not necessarily mean that significant adverse effects to populations of receptors are likely.

Eight sites with HQs in excess of 1 were identified in the WAG 5 ERA. As shown in Table 5, an additional screening was performed in which contaminants were eliminated from further evaluation for either of two reasons: (1) the exposure point concentration did not exceed 10 times the background concentration, or (2) the HQ was less than 10. The INEEL-wide ecological risk assessment to be conducted in the WAG 10 comprehensive investigation will consider those WAG 5 sites eliminated in the additional screening: PBF-10, PBF-21, PBF-22, and PBF-26. Information from the INEEL-wide evaluation will be considered in the 5-year reviews for WAG 5. If indicated, additional remediation to protect ecological receptors from contamination at these sites will be considered.

Four sites, ARA-01, ARA-12, ARA-25, and PBF-16, were retained for evaluation of remedial alternatives in the Comprehensive Feasibility Study (Holdren et al. 1999) to address ecological HQs in excess of 10. Because these sites are small, it is less expensive to remediate than it is to characterize further. Three of these sites, ARA-01, ARA-12, and ARA-25, also exceed human health risk thresholds.

Principal sources of uncertainty apply to the use of data not specifically collected for ERA and in the development of the exposure assessment. Uncertainties inherent in the exposure assessment are associated with estimation of receptor ingestion rates, selection of acceptable HQs, estimation of site usage, and estimation of risk assessment parameters (e.g., plant uptake factors and bioaccumulation factors). Additional uncertainties are associated with the depiction of site characteristics, the determination of the nature and extent of contamination, and the derivation of TRVs. A large area of uncertainty is the inability to evaluate risk to many receptors because of the lack of appropriate toxicity data for many chemicals. This is especially a problem for certain receptors such as reptiles. In addition, because of the conservative nature of assumptions made to compensate for the lack of site-specific uptake and bioaccumulation factors, ecologically based screening levels for some chemicals are lower than their sample quantitation and detection limits. In the WAG 5 analysis, this occurs for metals, PCBs, and some other organics. All of these uncertainties likely influence risk estimates. The major sources and effects of uncertainties in the ERA are reviewed in Table 6.

**7.2.1.4 Transition to the INEEL-wide Ecological Risk Assessment.** The third phase of the ERA process is the WAG 10 (OU 10—04) ERA, which will integrate WAG ERAs to evaluate risk to INEEL-wide ecological resources. This assessment will evaluate effects resulting from past contamination and their potential for adversely impacting INEEL-wide ecological resources including residual impacts from completed interim or remedial actions.

**Table 5.** Results of WAG 5 ecological contaminant screening against 10 times background concentrations and concentrations equivalent to a hazard quotient of 10.

Site	Contaminant	Maximum Concentration (mg/kg)	95% UCL (mg/kg)	10x INEEL Background (mg/kg)	Maximum Hazard Quotient	Comment	WAG 5 Remediation?
ARA-01	Antimony	1.68E+01	—	4.80E+01	1.00E+01	Below 10 × background	no
	Arsenic	2.58E+01	—	5.80E+01	2.00E+01	Below 10 × background	no
	Cadmium	3.80E+00	—	2.20E+01	1.00E+03	Below 10 × background	no
	Copper	2.55E+01	—	2.20E+02	1.00E+01	Below 10 × background	no
	Lead	4.39E+01	—	1.70E+02	6.00E+01	Below 10 × background	no
	Selenium	<b>2.77E+01</b>	—	<b>2.20E+00</b>	<b>3.00E+02</b>	—	<b>YES</b>
	Thallium	<b>5.92E+01</b>	<b>3.70E+01</b>	<b>4.30E+00</b>	<b>3.00E+02</b>	—	<b>YES</b>
	Vanadium	6.80E+01	—	4.50E+02	2.00E+02	Below 10 × background	no
	Zinc	2.33E+02	—	1.50E+03	2.00E+01	Below 10 × background	no
ARA-12	Cadmium	6.52E+00	—	2.20E+01	2.48E+03	Below 10 × background	no
	Chromium(III)	4.69E+02	—	3.30E+02	9.31E+00	HQ < 10	no
	Copper	<b>6.23E+02</b>	—	<b>2.20E+02</b>	<b>3.00E+02</b>	—	<b>YES</b>
	Lead	1.58E+02	—	1.70E+02	3.38E+02	Below 10 × background	no
	Manganese	5.70E+02	—	4.90E+03	3.90E+01	Below 10 × background	no
	Mercury	<b>1.40E+00</b>	—	<b>5.00E-01</b>	<b>9.00E+01</b>	—	<b>YES</b>
	Selenium	<b>2.70E+00</b>	—	<b>2.20E+00</b>	<b>3.00E+01</b>	—	<b>YES</b>
	Zinc	3.76E+02	—	1.50E+03	5.29E+01	Below 10 × background	no
ARA-25	Arsenic	2.58E+01	—	5.8E+01	2.00E+01	Below 10 × background	no
	Cobalt	1.04E+02	—	1.10E+02	9.00E+01	Below 10 × background	no
	Copper	<b>2.27E+02</b>	—	<b>2.20E+02</b>	<b>4.00E+01</b>	—	<b>YES</b>
	Lead	<b>1.43E+03</b>	—	<b>1.70E+02</b>	<b>9.00E+02</b>	—	<b>YES</b>
	Manganese	1.40E+03	—	4.90E+03	6.00E+00	Below 10 × background	no
	Mercury	9.70E-02	—	5.00E-01	3.00E+00	Below 10 × background	no
	Nickel	3.88E+01	—	3.50E+02	6.00E+00	HQ < 10	no
	Selenium	6.59E-01	—	2.20E+00	3.00E+00	HQ < 10	no
	Silver	7.24E+00	—	NA	2.00E+00	HQ < 10	no
	Vanadium	1.04E+02	—	4.50E+02	1.00E+02	Below 10 × background	no
	Zinc	8.55E+02	—	1.50E+03	3.00E+01	Below 10 × background	no
	PBF-10 <sup>a</sup>	Chromium(III)	3.09E+02	—	3.30E+02	1.00E+01	Below 10 × background
PBF-16	Lead	3.21E+01	—	1.70E+02	6.00E+01	Below 10 × background	no
	Mercury	<b>7.10E-01</b>	—	<b>5.00E-01</b>	<b>5.00E+01</b>	—	<b>YES</b>
PBF-21 <sup>a</sup>	Cobalt	1.26E+01	—	1.10E+02	6.00E+00	Below 10 × background	no
	Copper	2.33E+01	—	2.20E+02	2.00E+00	Below 10 × background	no
PBF-22 <sup>a</sup>	Arsenic	1.22E+01	—	5.80E+01	8.33E+00	Below 10 × background	no
	Copper	4.84E+01	—	2.20E+02	2.06E+01	Below 10 × background	no
	Lead	6.84E+01	—	1.70E+02	4.40E+01	Below 10 × background	no
	Mercury	2.70E-01	—	5.00E-01	1.82E+01	Below 10 × background	no
	Nickel	4.10E+01	—	3.50E+02	1.37E+01	Below 10 × background	no
	Selenium	1.70E+00	—	2.20E+00	1.88E+01	Below 10 × background	no
PBF-26 <sup>a</sup>	Arsenic	7.90E+00	—	5.80E+01	7.90E+00	Below 10 × background	no
	Chromium(III)	6.40E+01	—	3.30E+02	1.95E+00	Below 10 × background	no

**Table 5.** (continued).

Site	Contaminant	Maximum Concentration (mg/kg)	95% UCL (mg/kg)	10× INEEL Background (mg/kg)	Maximum Hazard Quotient	Comment	WAG 5 Remediation?
	Copper	2.34E+02	1.10E+02	2.20E+02	9.98E+01	95% UCL below 10 × background	no
	Lead	4.30E+01	—	1.70E+02	9.79E+01	Below 10 × background	no
	Mercury	3.40E-01	—	5.00E-01	2.30E+01	Below 10 × background	no
	Nickel	4.50E+01	—	3.50E+02	1.50E+01	Below 10 × background	no
	Zinc	2.59E+02	—	1.50E+03	3.65E+01	Below 10 × background	no

a. Sites PBF-10, PBF-21, PBF-22, and PBF-26 will be evaluated in the INEEL-wide ecological risk assessment.

**Table 6.** Source and effects of uncertainties in the ecological risk assessment.

Uncertainty Factor	Effect of Uncertainty (level of magnitude)	Comments
Ingestion rates (soil, water, and food)	May overestimate or underestimate risk (moderate).	Ingestion estimates used for terrestrial receptors are based on data in the scientific literature. Food ingestion rates are calculated by using allometric equations available in the literature (Nagy 1987). Soil ingestion values are generally taken from Beyer, Connor, and Gerould (1994).
Acceptable hazard quotients	May overestimate or underestimate risk (high).	The magnitude of the hazard quotient indicates the level of concern for a functional group or species based on perceived importance.
Concentration factors and plant uptake factors	May overestimate or underestimate risk, and the magnitude of error cannot be quantified (high).	Few bioaccumulation factors or plant uptake factors are available in the literature because they must be both contaminant- and receptor-specific. In the absence of more specific information, values for these parameters are obtained from Baes et al. (1984) for metals and elements, and from Travis and Arms (1988) for organics.
Toxicity reference values (TRVs)	May overestimate (high) or underestimate (moderate) risk.	To compensate for potential uncertainties in the exposure assessment, various adjustment factors are incorporated to extrapolate toxicity from the test organism to other species.
Conservative TRVs may be below background concentrations	May overestimate (high) risk.	Because of compensation for potential uncertainties, the calculation of TRVs (see above comment) may result in risk being shown at INEEL background concentrations and give an erroneous indication of risk to certain receptors.
Lack of appropriate toxicity data to derive TRVs	Results in the inability to evaluate risk for many receptors and chemicals.	Those receptor groups and chemicals that could not be evaluated are data gaps in the assessment.
Use of functional grouping	May overestimate (moderate) risk.	Functional groups were designed as an assessment tool to ensure that the ERA address all species potentially present at a facility. A hypothetical species is developed using input values that represent the greatest exposure of the combined functional group members.
Site use factor	May overestimate (high) or underestimate (low) risk.	The site use factor is a percentage of the site of concern area compared to the home range of the receptor species. When the home range is not known for a species, a default value of 1.0 is used. This can result in an overestimate of the risk at small sites.

The INEEL-wide ecological risk assessment to be conducted in the WAG 10 comprehensive investigation will consider those WAG 5 sites eliminated in the additional screening: PBF-10, PBF-21, PBF-22, and PBF-26 (see Table 5). Further evaluation of these sites was deferred to WAG 10 for either of two reasons: (1) the exposure point concentrations do not exceed 10 times the background concentrations, or (2) the HQs are less than 10. The INEEL-wide ERA will be conducted as a component of the comprehensive RI/FS for OU 10-04. The WAG 10 comprehensive investigation will be referenced during the 5-year review process for WAG 5 to determine whether the decisions implemented by WAG 5 are still protective of the environment. Future remediation may be necessary if the WAG 10 INEEL-wide assessment indicates that a cumulative ecological risk is exceeded for a population of receptors or if land use changes.

### 7.3 Risk Assessment Summary

Unexpectedly high risks were estimated in the WAG 5 baseline risk assessment for Ra-226 at several sites. Further investigation revealed that reported Ra-226 concentrations were artificially high. In most cases, gamma-ray spectroscopy was the analytical method used to quantify Ra-226 concentrations. However, this method does not provide sufficient resolution to discriminate Ra-226 from U-235, a naturally occurring radioisotope. Therefore, a correction factor was developed (Giles 1998b). For those sites at which the corrected Ra-226 concentrations were at or below background values, Ra-226 was eliminated as a contaminant of potential concern in soil after the baseline risks were estimated (Holdren et al. 1999). The sites that were affected by the correction factor were ARA-01, ARA-02 (in soil around the seepage tanks, but not in the seepage pit sludge), ARA-16, and ARA-23. The appropriate background values for Ra-226 are 1.2 pCi/g for analytical methods that avoid U-235 interference and 2.1 pCi/g for results that include interference from U-235 (Giles 1998a).

Risk estimates for the future residential scenario and ecological risks were used to identify sites for remediation. After the modifications to the baseline risk assessment for Ra-226, seven sites were identified for evaluation of remedial alternatives in the feasibility study: ARA-02 Sanitary Waste System (the seepage pit sludge is the primary remediation target), ARA-16, and ARA-23 for human health risks; PBF-16 for ecological risks; and ARA-01, ARA-12, and ARA-25 for both human health and ecological risks. At five of these sites, ARA-01, ARA-12, ARA-23, ARA-25, and PBF-16, soil is the only medium that will be remediated. At ARA-02 and ARA-16, residual waste and subsurface structures also will be removed. Table 7 summarizes the risk assessment for these seven sites, which are described below:

**Table 7.** Individual sites and contaminants of concern addressed by the selected remedy for WAG 5.

Site	Contaminant of Concern	Exposure Pathway	Risk	Hazard Quotient
<b>Future Residential Exposure Scenario</b>				
ARA-01 (soil)	Arsenic	Dermal absorption from soil	2E-04 (2 in 10,000)	1
ARA-02 (seepage pit sludge)	Lead	Ingestion of soil	NA <sup>a</sup>	NA <sup>a</sup>
	Aroclor-1242	Dermal absorption from soil and ingestion of soil	1E-05 <sup>b</sup>	NA <sup>c</sup>
	Ra-226	External radiation exposure	2E-03 (2 in 1,000)	NA
	Cs-137	External radiation exposure	7E-05 <sup>b</sup> (7 in 100,000)	NA
	U-235	External radiation exposure	9E-05 <sup>b</sup> (9 in 100,000)	NA
	U-238	External radiation exposure	3E-05 <sup>b</sup> (3 in 100,000)	NA
ARA-12 (soil)	Ag-108m	External radiation exposure	2E-03 (2 in 1,000)	NA
	Cs-137	External radiation exposure	2E-04 (2 in 10,000)	NA

**Table 7. (continued).**

Site	Contaminant of Concern	Exposure Pathway	Risk	Hazard Quotient
ARA-16 (soil)	Cs-137	External radiation exposure	1E-04 (1 in 10,000)	NA
ARA-23 (soil)	Cs-137	External radiation exposure	5E-04 (5 in 10,000)	NA
ARA-25 (soil)	Arsenic	Dermal absorption from soil	3E-04 (3 in 10,000)	2
	Arsenic	Ingestion of soil	9E-05 <sup>d</sup> (9 in 100,000)	1
	Lead	Ingestion of soil	NA <sup>a</sup>	NA <sup>a</sup>
	Cs-137	External radiation exposure	2E-03 (2 in 1,000)	NA
	Ra-226	External radiation exposure	5E-03 (5 in 1,000)	NA
	Ra-226	Ingestion of soil	1E-05 <sup>d</sup> (1 in 100,000)	NA
<b>Current Occupational Exposure Scenario</b>				
ARA-12 (soil)	Ag-108m	External radiation exposure	1E-03 (1 in 1,000)	NA
ARA-16 (soil)	Co-60	External radiation exposure	2E-04 (2 in 10,000)	NA
	Cs-137	External radiation exposure	3E-04 (3 in 10,000)	NA
ARA-25 (soil)	Arsenic	Dermal absorption from soil	1E-04 (1 in 10,000)	1
	Cs-137	External radiation exposure	4E-03 (4 in 1,000)	NA
	Ra-226	External radiation exposure	1E-03 (1 in 1,000)	NA
<b>Future Occupational Exposure Scenario</b>				
ARA-12 (soil)	Ag-108m	External radiation exposure	6E-04 (6 in 10,000)	NA
ARA-25 (soil)	Arsenic	Dermal absorption from soil	1E-04 (1 in 10,000)	1
	Cs-137	External radiation exposure	4E-04 (4 in 10,000)	NA
	Ra-226	External radiation exposure	1E-03 (1 in 1,000)	NA
<b>Ecological Exposure Scenario</b>				
ARA-01 (soil)	Selenium	Ecological exposure	NA	≤ 1 to ≤ 300
	Thallium	Ecological exposure	NA	≤ 1 to ≤ 300
ARA-12 (soil)	Copper	Ecological exposure	NA	≤ 1 to ≤ 300
	Mercury	Ecological exposure	NA	≤ 1 to ≤ 90
	Selenium	Ecological exposure	NA	≤ 1 to ≤ 30
ARA-25 (soil)	Copper	Ecological exposure	NA	≤ 1 to ≤ 40
	Lead	Ecological exposure	NA	≤ 1 to ≤ 900
PBF-16 (soil)	Mercury	Ecological exposure	NA	≤ 1 to ≤ 50

a. Risks and hazard quotients could not be estimated for lead because human health toxicity data are not available. However, concentrations in excess of the EPA screening level of 400 mg/kg (EPA 1994b) will be remediated.

b. The cumulative risk for Aroclor-1242, Cs-137, U-235, and U-238 in the seepage pit is greater than 1E-04. Therefore, these constituents were identified as contaminants of concern.

c. Remedial decisions cannot be based on a hazard quotient for Aroclor-1242, a polychlorinated biphenyl, because EPA-approved reference doses are not available. However, Aroclor-1242 will be remediated in conjunction with the cleanup of the seepage pit sludge.

d. The cumulative risk for arsenic and Ra-226 in the ingestion of soil pathway for ARA-25 equals 1E-04. Therefore, arsenic was identified as a contaminant of concern for the soil ingestion pathway. Radium-226 is a contaminant of concern for both the external exposure and soil ingestion pathways.

- Contaminated soil sites: ARA-01, ARA-12, ARA-23, ARA-25, and PBF-16
  - Site ARA-01 (ARA-I Chemical Evaporation Pond) will be remediated to address human health risk from arsenic and potential risks to ecological receptors from exposure to selenium and thallium in soil.
  - Site ARA-12 (ARA-III Leach Pond) will be remediated to address human health risks from Ag-108m and Cs-137 and ecological risks from copper, mercury, and selenium in surface and subsurface soil. The area of elevated gamma activity to the southwest of the site also will be remediated.
  - Site ARA-23 (ARA-I and ARA-II Radiologically Contaminated Soil) will be remediated to address the human health risks from Cs-137. The site includes the radiologically contaminated soil around ARA-I and ARA-II and the remaining reactor foundation and the remaining underground utilities within the facility fences.
  - Site ARA-25 (ARA-I Contaminated Soil Beneath the ARA-626 Hot Cells) will be remediated to address human health risks from Ra-226, Cs-137, and arsenic and to address ecological risks from copper and lead.
  - Site PBF-16 (SPERT-II Leach Pond) will be remediated to address the ecological risks from mercury in surface soil.
- Site ARA-02 (ARA-I Sanitary Waste System) will be remediated to address the human health risks from the contaminants of concern (COCs) Aroclor-1242, Ra-226, Cs-137, U-235, and U-238 in the seepage pit sludge. The analytical results for the soil around the system (i.e., outside of the seepage pit and around the septic tanks) indicate that soil concentrations are below risk-based concentrations.
- Site ARA-16 (ARA-I Radionuclide Tank) will be remediated to address the human health risks from Cs-137 in the soil surrounding the tank. Because the ARA-16 tank is still in place and contains principal threat waste that could pose a risk should a release to the environment occur, the waste in the tank, the tank itself, and the associated piping also will be removed.

In addition to the contaminants with quantified risks in Table 7, PCBs and lead in the ARA-02 seepage pit sludge and lead in the ARA-25 soil also will be remediated. Human health risks associated with these contaminants were not quantifiable because approved reference doses are not available. Calculations using preliminary reference doses for PCBs at ARA-02 indicate that the noncarcinogenic hazard indices for dermal absorption and soil ingestion exceed 1. Though toxicity data are not available for lead, the concentrations detected at ARA-02 and ARA-25 exceed the EPA 400 mg/kg screening level (EPA 1994b).

The GWSCREEN results indicated that WAG 5 does not contain sources of contamination that have the potential to produce risk greater than 1E-04 or an HQ greater than 1 for groundwater exposure pathways (e.g., groundwater ingestion) (Holdren et al. 1999, Section 4.3). In addition, residential scenario cumulative risk estimates and hazard indices were less than 1E-04 and 1, respectively, for the combined sources within WAG 5 for the air and groundwater exposure pathways.