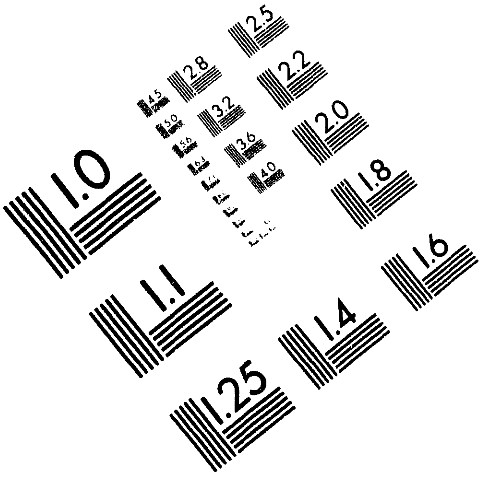
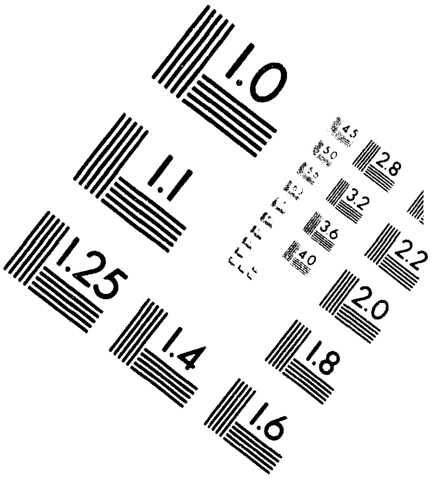

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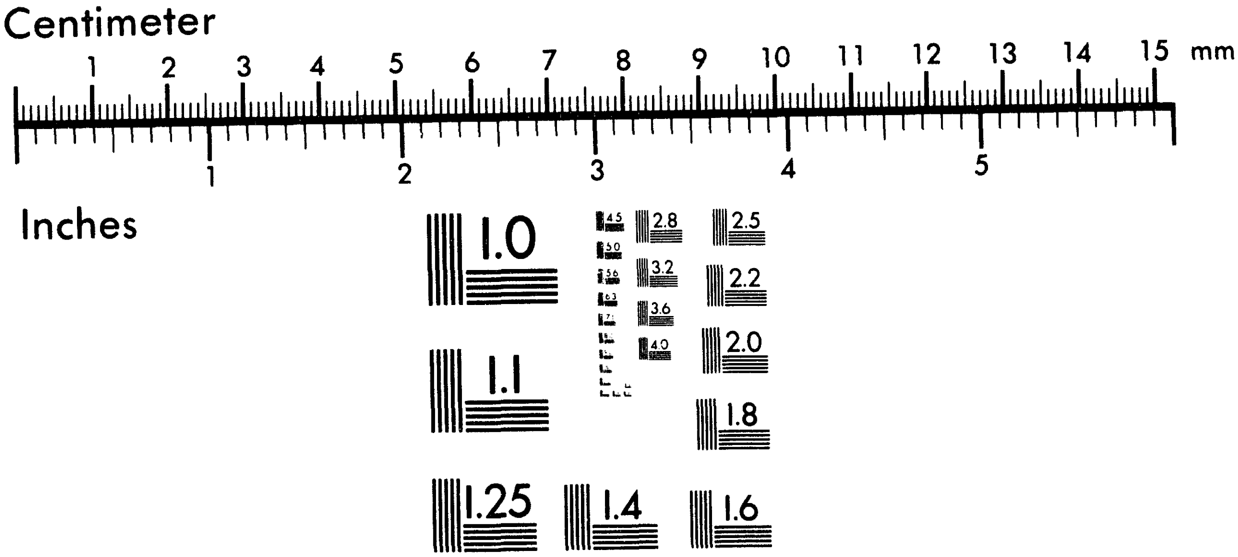

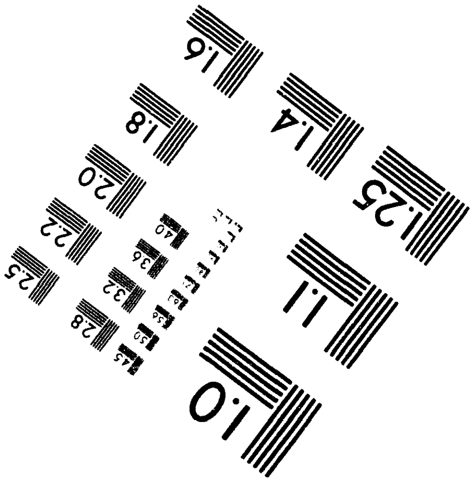
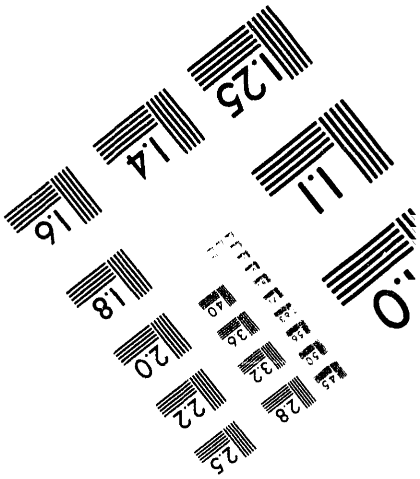

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BASELINE RISK ASSESSMENT OF GROUND WATER CONTAMINATION AT THE URANIUM MILL TAILINGS SITE NEAR GRAND JUNCTION, COLORADO

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June 1994

Prepared for
U.S. Department of Energy
UMTRA Project Office
Albuquerque, New Mexico

Prepared by
Jacobs Engineering Group Inc.
Albuquerque, New Mexico

CITIZENS' SUMMARY

This *Baseline Risk Assessment of Ground Water Contamination at the Uranium Mill Tailings Site Near Grand Junction, Colorado* evaluates potential impacts to public health or the environment resulting from ground water contamination at the former uranium mill processing site. The tailings and other contaminated material at this site were placed in an off-site disposal cell by the U.S. Department of Energy's (DOE) Uranium Mill Tailings Remedial Action (UMTRA) Project. The remedial activities at the site were conducted from 1989 to 1993. Currently, the UMTRA Project is evaluating ground water contamination. This risk assessment is the first document specific to this site for the Ground Water Project.

This risk assessment evaluates the most contaminated ground water that flows beneath the processing site toward the Colorado River. The monitor wells that have consistently shown the highest concentrations of most contaminants are used to assess risk. This risk assessment will be used in conjunction with additional activities and documents to determine what remedial action may be needed for contaminated ground water at the site.

There is no current use of ground water in the site vicinity. It is unlikely that ground water will be used in the future for household purposes because of the existing public water supply system. However, consistent with U.S. Environmental Protection Agency (EPA) guidance, this risk assessment will assume hypothetical future use of ground water resources, including the use of ground water from the alluvium for drinking, cooking, and bathing. The potential also exists for plants irrigated with contaminated ground water to accumulate the contaminants and for people to then be exposed by eating garden produce.

This risk assessment follows an approach outlined by the EPA. The first step is to evaluate ground water data collected from monitor wells at the site. Evaluation of these data showed that the contaminants of potential concern in the ground water are arsenic, cadmium, cobalt, fluoride, iron, manganese, molybdenum, nickel, sulfate, uranium, vanadium, zinc, and radium-226.

The next step in the risk assessment is to estimate how much of these contaminants people would be exposed to if they drank from a well installed in the contaminated ground water at the former processing site. Contaminant concentrations vary within the plume and vary each time a well is sampled. In addition, people vary in how much they weigh and drink. Therefore, this risk assessment uses probability distributions to determine how much of each contaminant would likely be ingested if someone installed a well at the site. The probability distributions describe how likely it is that a particular event will happen. For example, based on population survey data, probability distributions can describe what percentage of people drink a half-gallon of water each day and what percentage drink only one cup of water each day. Ingestion of contaminants could also occur from eating garden produce watered with contaminated ground water and eating fish from the Colorado River in the site vicinity.

The estimated amounts of contaminants that people might take in from these routes are then compared to the toxic effects these contaminant levels might cause. Based on this analysis, no adverse health effects would be expected following ingestion of garden produce watered with contaminated ground water. However, long-term irrigation with ground water would be toxic to the plants themselves due to accumulation of the contaminants in soil. Ingestion of fish would not be expected to represent a hazard to humans. However, direct consumption of the ground water from the former processing site could represent a concern.

If ground water at the Grand Junction site were used as drinking water, the most significant health hazards would be posed by high concentrations of sulfate, manganese, and fluoride. Sulfate intake from drinking ground water at the site would cause severe diarrhea in infants that could lead to dehydration. The manganese concentrations at the former mill site are at levels that have been reported in the literature to cause central nervous system problems when ingested over a long period of time. The fluoride concentrations in ground water at the site would result in tooth mottling in children when ingested over a long period of time. This contaminated ground water at the site is not currently being used for domestic purposes.

Other contaminants that occur in the ground water at levels associated with adverse noncancer health effects when ingested over a long period of time are vanadium, cadmium, iron, and arsenic. The vanadium concentrations in ground water could result in cramps and changes in blood chemistry. The concentrations of cadmium, iron, and arsenic detected in ground water at the site would be expected to result in adverse health effects only at the upper range of predicted intake levels. The adverse effect associated with cadmium would be the presence of protein in the urine. For iron, the health effects reported in the literature are liver damage, pigmentation of the skin, and, at very high intakes, diabetes. Cardiovascular problems and skin lesions have been observed elsewhere from exposures to arsenic at these levels.

The levels of arsenic in ground water at the site would also result in an intake level from drinking water above the EPA's maximum acceptable level for additional lifetime cancer risks of 1 in 10,000. The additional cancer risk estimated from ingesting contaminated ground water for a lifetime would be 3 in 10,000 for ground water containing the median arsenic concentration measured in monitor wells at the former mill site. The additional cancer risks estimated from ingestion of contaminated ground water containing the maximum concentrations of uranium or radium-226 would be below the EPA's maximum acceptable level (8 in 100,000 for uranium; 7 in 100,000 for radium-226).

This risk assessment also evaluated potential effects on livestock if the ground water were used to water them. Based on available information, sulfate and total dissolved solids (TDS) are the primary contaminants that could represent a hazard to livestock. The sulfate and TDS concentrations at the former mill site are high enough to cause adverse effects and probably death in cattle if the ground water were used as the sole source of long-term drinking water. Several contaminants could not be evaluated because information on drinking water concentrations that would be protective of livestock is not available.

Based on data and criteria available for a number of the contaminants of potential concern, no ecological threat exists to plants at harvestable maturity that may have roots in contact with soil saturated with the most contaminated ground water in the alluvial aquifer.

Potential use of this ground water would not be suitable for continuous irrigation of crops because fluoride, manganese, molybdenum, and vanadium could build up to toxic levels in the soil following long-term use. The ground water would also not be suitable as a body of water for fish to live in if a pond were created. However, the available surface water and sediment data from the Colorado River in the site vicinity suggest that contaminated ground water from the site has not adversely affected the surface water and sediment quality.

A long-term ground water monitoring plan has been proposed and is being implemented for the former mill site and vicinity. As additional water quality data are collected and interpreted, the monitoring plan will be updated annually to provide ongoing protection for public health and the environment.

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LIST OF ACRONYMS AND ABBREVIATIONS

Acronym	Definition
ac	acre
ACL	alternate concentration limit
$BaSO_4$	barite
BCF	bioconcentration factor
$CaCO_3$	calcite
CaF_2	fluorite
$CaSO_4$	gypsum
$CdCO_3$	octavite
CDH	Colorado Department of Health
cm	centimeter
DHHS	Department of Health and Human Services
DOE	U.S. Department of Energy
EPA	U.S. Environmental Protection Agency
F^-	fluoride
Fe^{2+}	ferrous iron
Fe^{3+}	ferric iron
ft	foot
FWQC	Federal Water Quality Criterion
ha	hectare
HDL	high-density lipoprotein
HEAST	Health Effects Assessment Summary Tables
IRIS	Integrated Risk Information System
Kd	distribution coefficient
km	kilometer
LOAEL	lowest-observed-adverse-effect level
m	meter
m^3	cubic meter
MCL	maximum concentration limit
μg	microgram
μg/gram	micrograms per gram
mg	milligram
mg/kg	milligrams per kilogram
mg/kg-day	milligrams per kilogram (of body weight) per day
mg/kg DW	milligrams per kilograms dry weight
mg/L	milligrams per liter
$MgCO_3$	magnesite
mi	mile
mV	millivolt
NAS	National Academy of Sciences
NCP	National Contingency Plan
ng	nanogram
NOAA	National Oceanic and Atmospheric Administration
NOAEL	no-observed-adverse-effect level
PEIS	programmatic environmental impact statement

LIST OF ACRONYMS AND ABBREVIATIONS (Continued)

Acronym	Definition
PET	potential evapotranspiration
PNL	Pacific Northwest Laboratory
ppm	parts per million
RDA	recommended daily allowance
RfD	reference dose
RRM	residual radioactive materials
SF	slope factor
SQC	sediment quality criteria
TDS	total dissolved solids
TOC	total organic carbon
UMTRA	Uranium Mill Tailings Remedial Action
UMTRCA	Uranium Mill Tailings Radiation Control Act
VP	vicinity property
yd^3	cubic yard

1.0 INTRODUCTION

The purpose of this baseline risk assessment is to determine whether ground water contamination at the former Climax uranium mill site in Grand Junction, Colorado, has the potential to adversely affect public health or the environment. The Grand Junction site is one of 24 abandoned uranium processing sites undergoing remediation in accordance with the requirements of the Uranium Mill Tailings Radiation Control Act (UMTRCA) of 1978 (42 USC §7901 *et seq.*) under the oversight of the U.S. Department of Energy (DOE).

Under the Uranium Mill Tailings Remedial Action (UMTRA) Surface Project, the DOE is stabilizing tailings in disposal cells that minimize radon and other radiation emissions and further contamination of ground water. At the Grand Junction site, the tailings and other contaminated materials have been removed and relocated to the Cheney disposal site, 18 miles (mi) (29 kilometers [km]) southeast of the processing site. Movement of the tailings was completed in November 1993.

Under the UMTRA Ground Water Project, ground water contamination at the 24 sites will be evaluated to determine whether any remedial action is necessary and to implement such action. The DOE was authorized to perform ground water restoration in 1988 amendments to the UMTRCA.

The evaluation strategy and remediation methodology for contaminated ground water at UMTRA Project sites are described in a programmatic environmental impact statement (PEIS) for ground water contamination (DOE, 1994). This baseline risk assessment is one of the site-specific documents prepared to evaluate potential health and environmental risks and provide information to assist in determining what remedial action is necessary. Based on the PEIS, additional ground water characterization, and this risk assessment, a site-specific environmental assessment will be prepared to evaluate and select a remedial action for the Grand Junction site.

This risk assessment follows the basic approach prescribed by the U.S. Environmental Protection Agency (EPA) for evaluating hazardous waste sites to assess potential health and environmental impacts resulting from ground water contamination at the Grand Junction site. The baseline risk assessment includes the following steps:

- Data evaluation.
    - Combining existing data from various site investigations and related reports.
    - Comparing sample results with background and tailings source data.
    - Selecting chemical data for use in the risk assessment.
- Exposure assessment.
    - Characterizing exposure settings.
    - Identifying exposure pathways.
    - Quantifying exposure.

- Toxicity assessment.
    - Identifying toxicity values.
    - Evaluating noncarcinogenic effects.
    - Evaluating carcinogenic effects from radionuclides and chemical carcinogens.

- Public health risk characterization.
    - Comparing toxicity ranges to predicted exposure ranges.
    - Combining risks across exposure pathways and multiple contaminants.
    - Characterizing uncertainties.

- Environmental risk.
    - Characterizing potential biota exposure pathways.
    - Identifying potential ecological receptors.
    - Evaluating environmental risk qualitatively.

These steps are used to estimate risks from potential current and future use of ground water and surface water.

2.0 SITE DESCRIPTION

2.1 SITE BACKGROUND

The Grand Junction site covers 114 acres (ac) (46 hectares [ha]). It is adjacent to the south side of the city of Grand Junction, Colorado, and to the north side of the Colorado River (Figure 2.1). The site consists of the tailings pile area, mill site, and effluent ponds from the former Climax uranium mill site, which was operated by the Climax Uranium Company between 1951 and 1970 (Figure 2.2).

Prior to removal of contaminated materials, which occurred between 1989 and 1993, the Grand Junction processing site contained an estimated 3.1 million cubic yards (yd^3) (2.4 million cubic meters [m^3]) of contaminated materials in the form of finely ground sand and slimes and contaminated soils. The tailings were covered with approximately 6 inches (15 centimeters [cm]) of soil, and sparse vegetation occurred on the site.

Remedial action at the Grand Junction site consisted of the removal and transport of contaminated materials from the processing site and vicinity properties (VP) to the Cheney disposal site.

Many VPs also exist in the Grand Junction area. VPs are homes, businesses, public buildings, and vacant lots that may have been contaminated during construction by the use of tailings as a building material or as fill material before the hazards associated with this material were known. The use of the tailings for these purposes is no longer allowed.

Generally, the amounts of residual radioactive materials (RRM) at the VPs are small, and the potential impact to human health and the environment appears minimal. However, the potential for ground water contamination beneath the VPs and the associated cost risk for remediation have not been quantified.

2.2 CLIMATE

The climate in the vicinity of Grand Junction is arid; annual precipitation averages 8.9 inches (22.6 cm). During the summer, thunderstorms are common.

A potential evapotranspiration (PET) value for Grand Junction of 71 inches (180 cm) per year has been reported. Thus, the ratio of PET to precipitation is approximately 8.

2.3 LAND USE

The former Climax mill site is located in a primarily urbanized area, with residential, commercial, and industrial development nearby (Figure 2.3). The

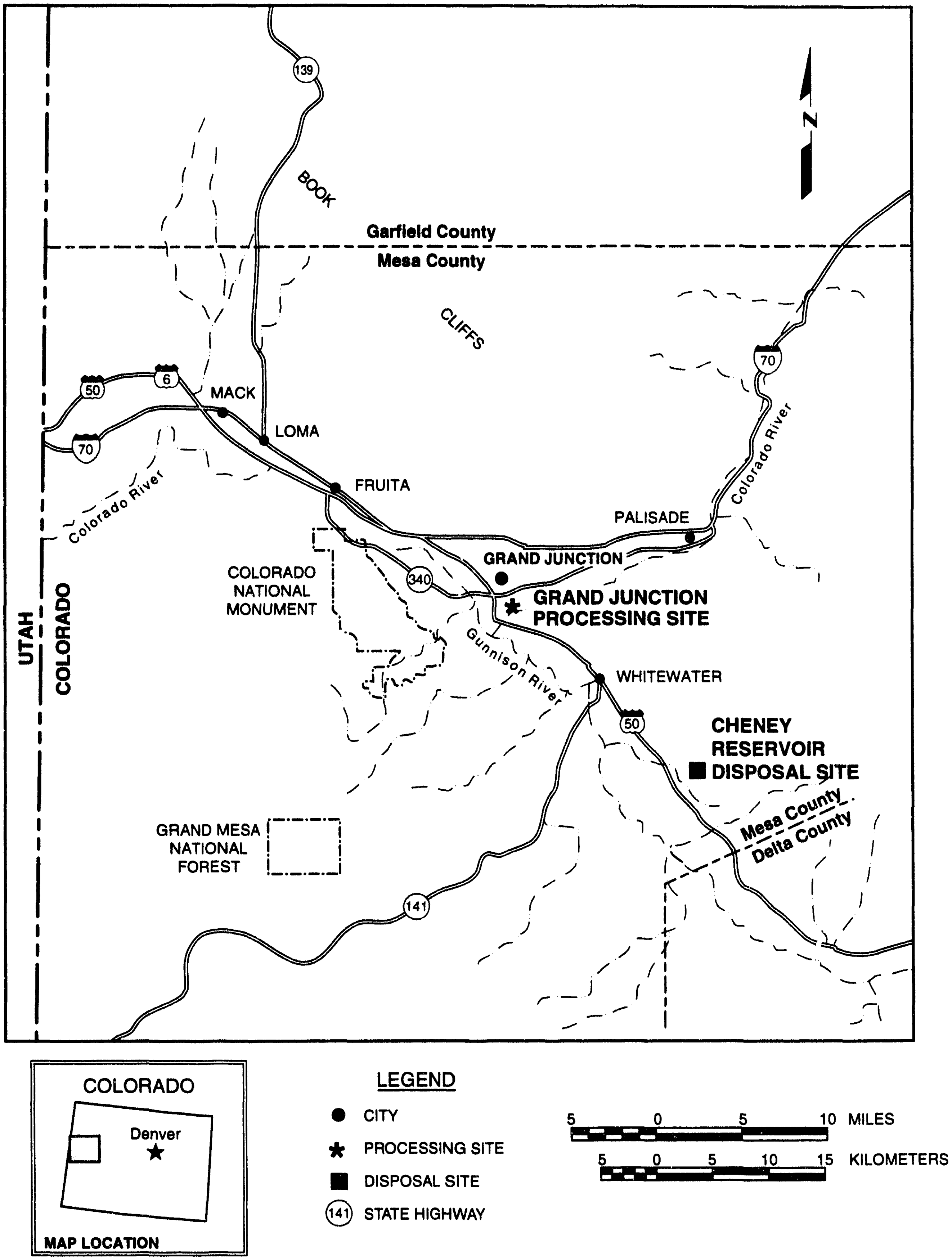


FIGURE 2.1
LOCATION OF GRAND JUNCTION SITE AND CHENEY RESERVOIR DISPOSAL SITE
GRAND JUNCTION, COLORADO

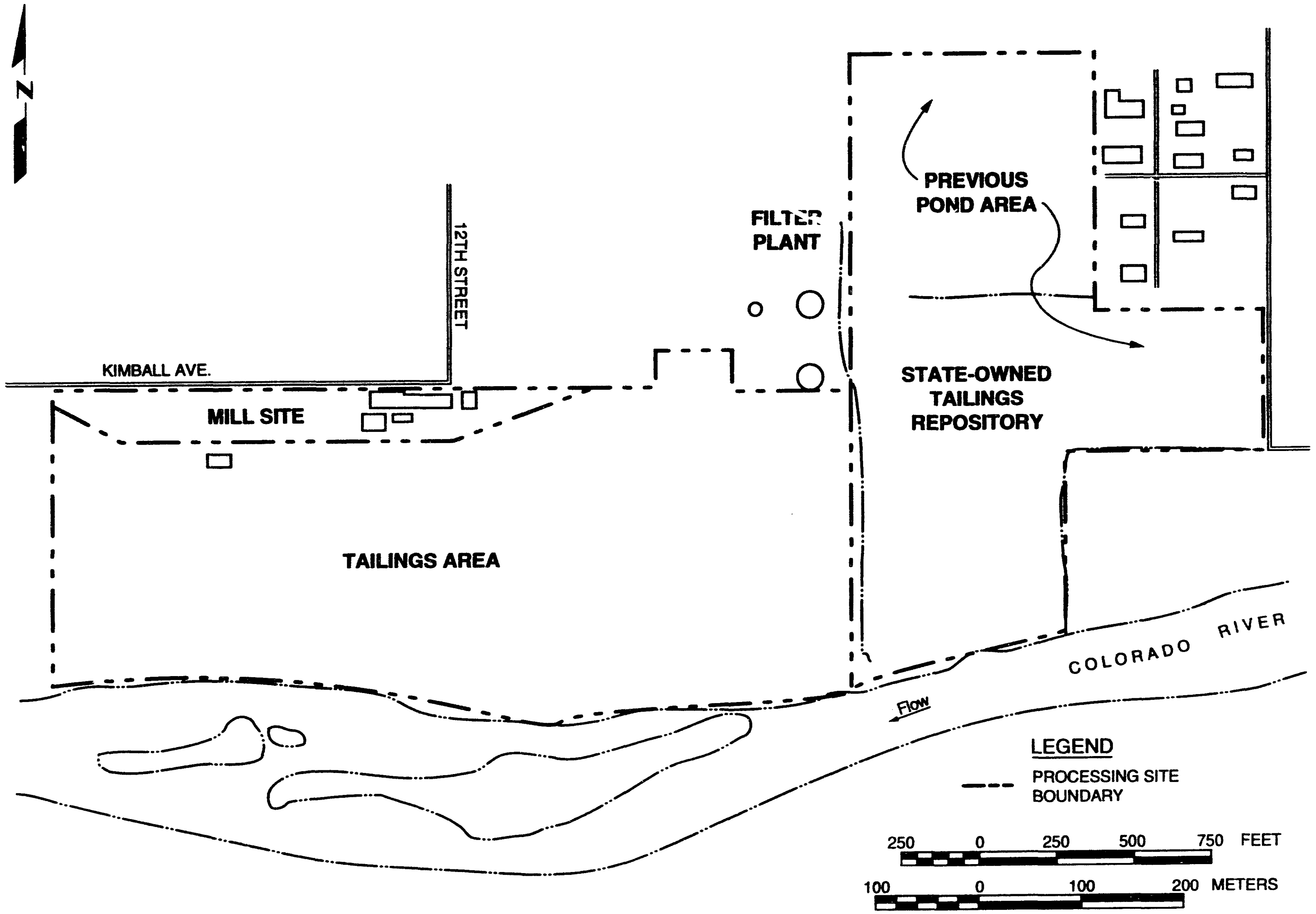


FIGURE 2.2
GRAND JUNCTION SITE MAP

MAC: SITE/GRJ/BRA/SITEMAP

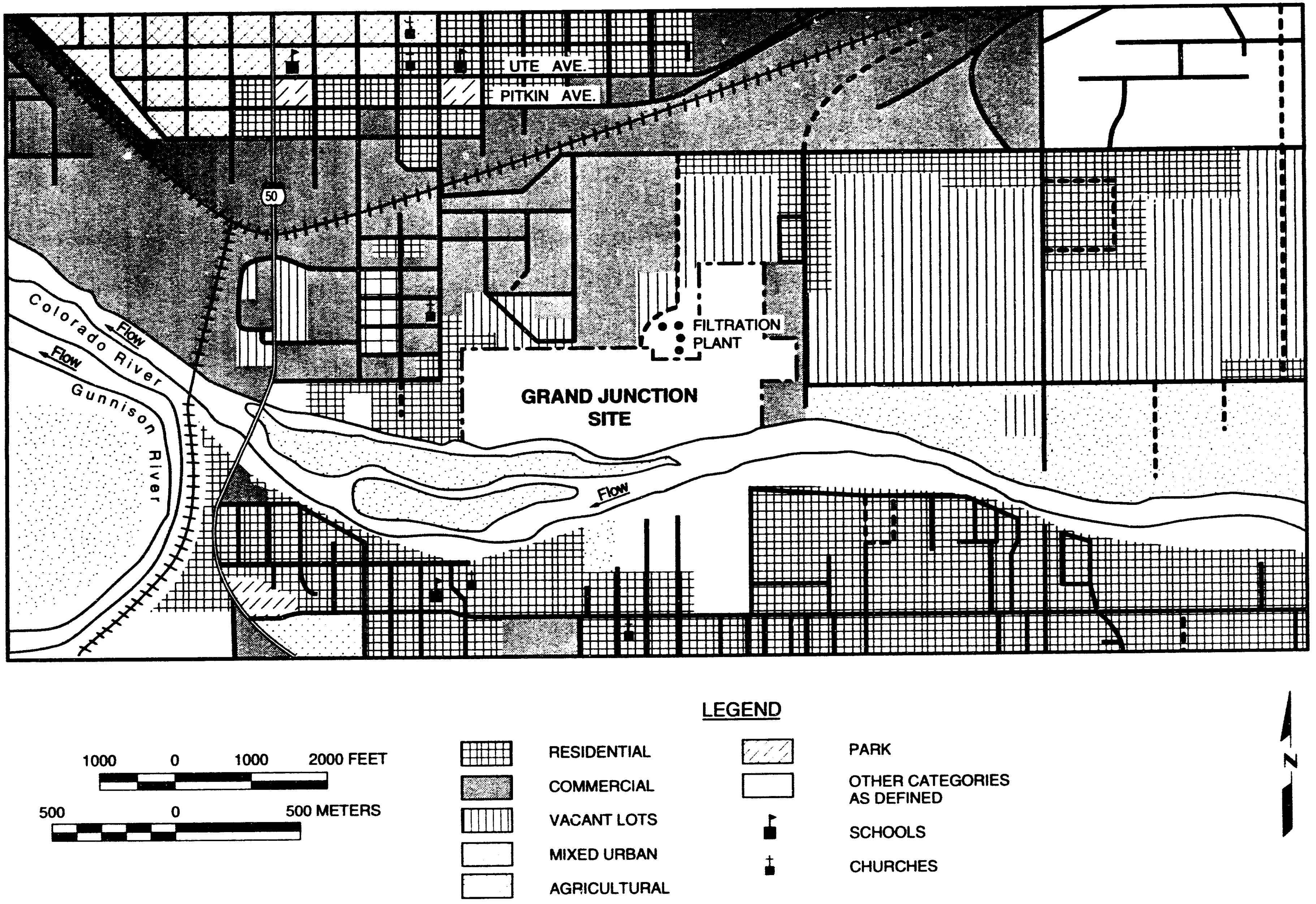


FIGURE 2.3
LAND USE AT THE GRAND JUNCTION SITE

MAC: SITE/GRJ/BRA/LANDUSE4.12

land within 0.5 mi (0.8 km) of the site north of the river has undergone a transition from residential to commercial and industrial uses. Residences have been replaced with commercial establishments to the northeast and west of the site. The area's proximity to the Denver and Rio Grande Western Railroad makes it desirable for industrial development. Commercial and industrial land use occurs immediately north and northeast of the tailings site. The railroad, situated approximately 0.5 mi (0.8 km) north of the processing site, is roughly the northern boundary of the area of industrial land use.

There is some residential development south of the site, across the Colorado River. A residential area west of the site is an older low income area, with many old houses interspersed with some commercial establishments.

The land east of the site is used for industrial purposes or is vacant. The vacant lands mark the transition between the urban area of the city and the rural residential and agricultural areas east of the city. The Grand Valley By-Products Company is located near the southeastern boundary of the mill site. The Grand Valley By-Products Company is a rendering plant that has been processing animal and animal by-products into hog and dairy feed for nearly 100 years.

Land and water use control

The former Climax mill site is outside the incorporated area of the city of Grand Junction and is therefore not subject to the zoning restrictions of the city. Land use outside the Grand Junction city limits is under the authority of the County Planning Commission. The commission provides recommendations to the Board of County Commissioners on land use activities requiring hearings such as rezoning, plan development, subdivisions, and conditional use permits. Land use activities governed by existing guidelines such as zoning regulations do not require hearings as long as code specifications are met.

County land use plans recommend the use of planned unit developments to ensure that all new construction conforms with the character of the area. The county encourages developers to draw up their own plan and site planning criteria, which are then subject to gross density limits, performance standards, and a requirement for buffer zones between substantially different types of development. The processing site and the surrounding area are currently zoned industrial.

The city of Grand Junction is planning on annexing the processing site area, at which time land use will be under the jurisdiction of the city. Development within the city limits of Grand Junction entailing a conditional use permit, rezoning, or a subdivision is subject to review and approval by the Grand Junction Planning Commission and/or the Grand Junction City Council. All other development is reviewed and approved by the City Community Development Department. With the support of other city agencies, the City Community Development Department reviews development plans for compliance with the city of Grand Junction Zoning and Development Code (Grand Junction City Council, 1989).

The site is located in a 100-year floodplain. City regulations require that a flood development permit be obtained before developments in the floodplain commence, and the county discourages development in the floodplain. Planning is under way to designate the tailings site as part of the Colorado park system.

The Grand Junction Zoning and Development Code requires that all potable water supply systems, whether individual or public, comply with state and county health departments as well as all city or other applicable regulations. The code also requires that all development be served by the city water treatment and distribution system and empowers the city utilities director to grant exceptions if the requirement is deemed unreasonable or impracticable.

2.4 SURFACE WATER

The former Climax mill site is north of the Colorado River 0.75 mi (1.2 km) upstream from its confluence with the Gunnison River. The Colorado River is braided by several islands from the upstream end of the processing site to a point about 0.5 mi (0.8 km) past its downstream end. The stream bed of the Colorado River is about 4560 feet (ft) (1390 meters [m]) above sea level.

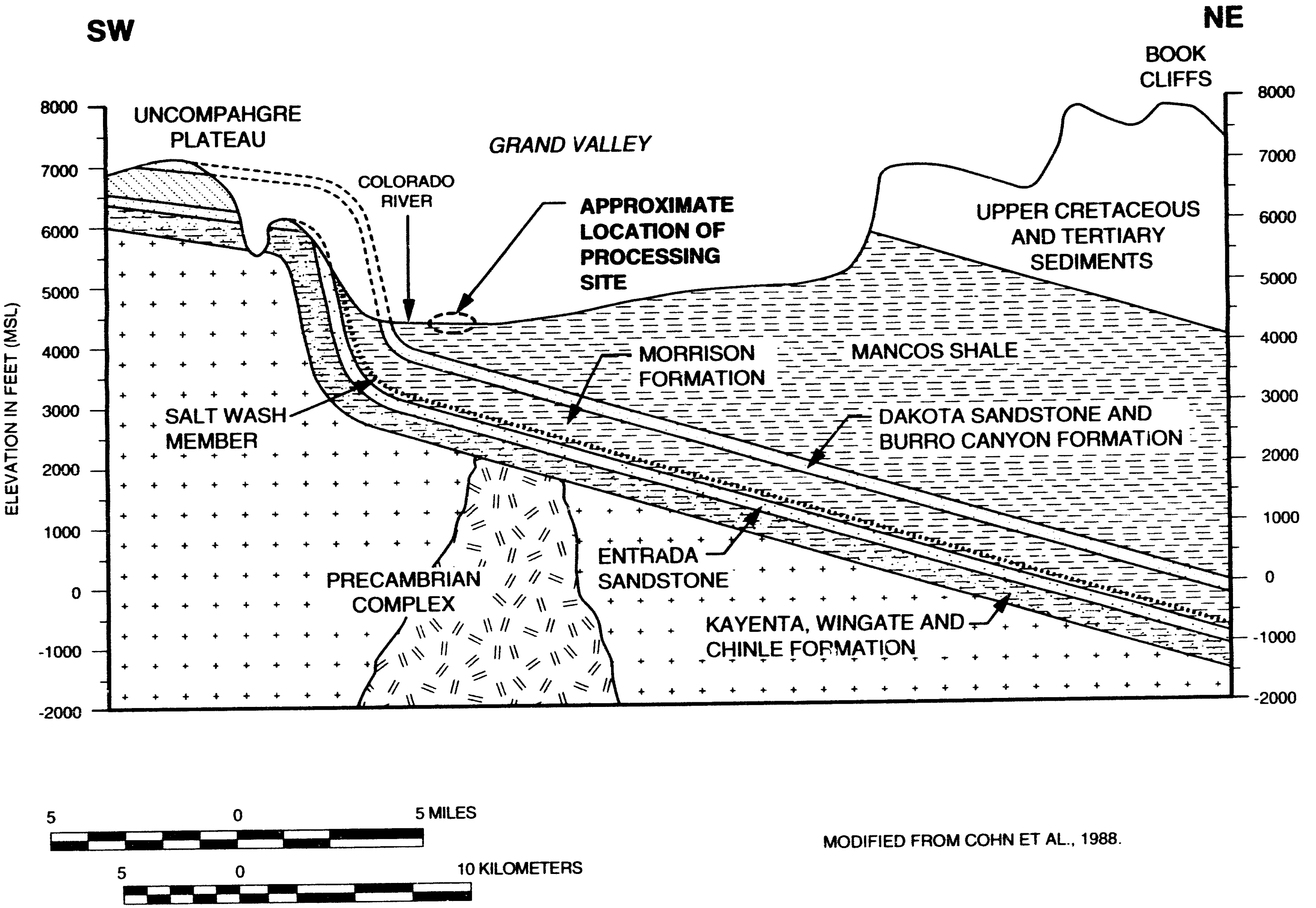
The southern side of the river banks against a steep cliff, approximately 60 ft (20 m) high, with the lower segment composed of Mancos Shale. The northern bank of the river upgradient of the site is stabilized to some degree with riprap, consisting of broken slabs and blocks of concrete and river gravels.

No major shift in location of the Colorado River channel at the site has occurred in the past 100 years. The islands near the site are heavily vegetated, which indicates relative stability over the past 20 years, although some shifts in island locations were noted during floods that occurred in 1984.

Regarding localized erosion, the existing islands indicate that sediment deposition normally occurs near the site. Several factors tend to cause deposition, including a decrease of channel gradient, proximity of the confluence with the Gunnison River, and the broad floodplain along the northern bank. Sediment deposition also would likely occur during the receding phase of a large flood.

2.5 HYDROGEOLOGY

Grand Junction lies in the Colorado Plateau physiographic province. Bedrock consists primarily of marine sedimentary rocks of the Mancos Shale. The site is located in the northeast-southwest trending Grand Valley. The Colorado River is located along the contact between the Dakota Sandstone and the Mancos Shale. Structurally, the sedimentary rocks at this location dip to the northeast at about 2 degrees. Figure 2.4 shows the generalized stratigraphy of the processing site and vicinity.



MODIFIED FROM COHN ET AL., 1988.

FIGURE 2.4
GENERALIZED CROSS SECTION OF THE GRAND VALLEY

The Grand Junction processing site is situated on the floodplain and low-level alluvial terraces immediately north of the Colorado River. In the vicinity of the processing site, the lowlands along the river are capped by variable, but usually thick, deposits of fluvial sand deposited within the last 50,000 years (Lohman, 1965). Ground water occurs in the alluvium under unconfined conditions. The ground water is usually of poor quality and is generally not developed.

Site-specific characteristics of the hydrogeology at the processing site have been determined through borehole drilling and test pit excavation. Figure 2.5 shows monitor well locations at the processing site and vicinity. All of the on-site monitor wells were plugged and abandoned as part of the remedial actions. The shallow stratigraphy near the processing site can be divided into three hydrogeologic zones. From top to bottom, these are as follows:

- A surficial disturbed zone.
- A zone of unconsolidated alluvial sediments.
- A sequence of consolidated sedimentary formations.

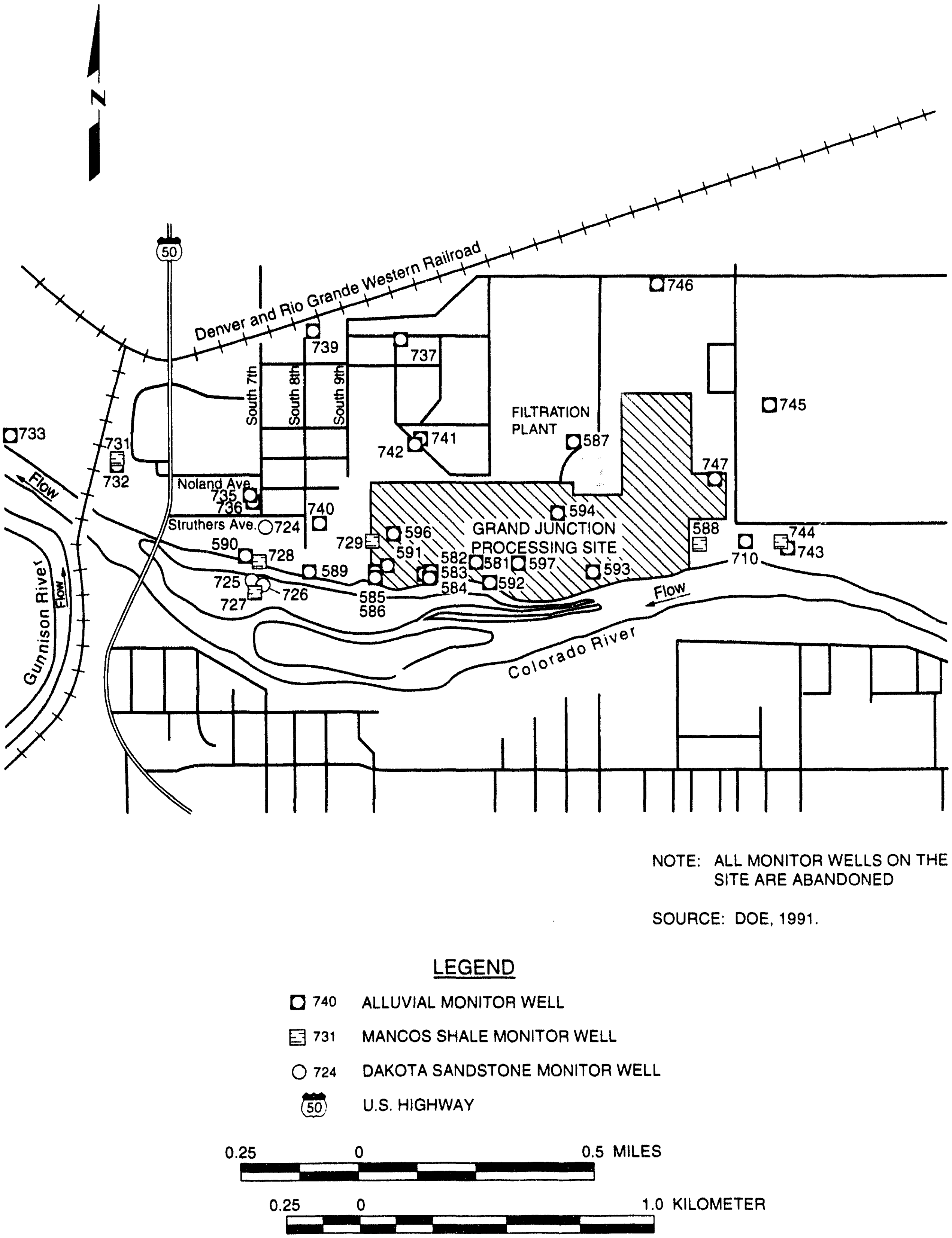
The surficial disturbed zone includes a variety of soil classifications and material types that have been deposited or altered by people. The zone varies in depth from less than 1 ft (0.3 m) to more than 50 ft (15 m).

Underlying, or adjacent to, the surficial disturbed zone is a zone of unconsolidated alluvial sediments. In the vicinity of the processing site, this zone includes mixed gravel, sand and silty layers, ranging in depth from less than 7 ft (2.1 m) to more than 21 ft (6.4 m). In general, the alluvium in this portion of the Grand Valley can be categorized into two types, as described by the U.S. Bureau of Reclamation (1978): "In deeper sections of the Colorado River paleochannel is a stratum of gravel and cobbles overlying the Mancos Shale referred to as the cobble aquifer. Overlying the cobble aquifer is a layer of alluvium that extends over the entire Grand Valley." The bottom of the alluvial aquifer is formed by the erosional surface of the Mancos Shale, which slopes gently (about 5 ft [1.5 m] per mile) to the north (U.S. Bureau of Reclamation, n.d.).

Underlying the unconsolidated alluvial sediments is a sequence of consolidated sedimentary formations. In descending order, these are as follows (Lohman, 1965):

- Mancos Shale.
- Dakota Sandstone.
- Burro Canyon Formation.

Because there is very low probability that ground water in the bedrock formations below the Dakota Sandstone can be influenced by tailings seepage, hydrogeology of formations in aquifers below the Dakota Sandstone will not be discussed further. However, these formations are shown in Figure 2.4.



NOTE: ALL MONITOR WELLS ON THE SITE ARE ABANDONED

SOURCE: DOE, 1991.

FIGURE 2.5
MONITOR WELL LOCATIONS IN THE VICINITY OF THE GRAND JUNCTION, COLORADO, PROCESSING SITE

The Mancos Shale is a thick, relatively extensive sequence of shale that includes some sandy layers and thin sandstone beds. The Mancos Shale is not considered a source of water in the vicinity of Grand Junction. It is an aquitard that behaves as a barrier to downward migration of contamination. The Mancos Shale varies in thickness near the processing site from greater than 100 ft (30 m) near the tailings to almost entirely absent about 0.5 mi (0.8 km) west of the tailings.

The Dakota Sandstone/Burro Canyon Formation aquifer is the uppermost bedrock aquifer that underlies the Mancos Shale. The Dakota Sandstone/Burro Canyon Formation aquifer consists of beds of sandstone, conglomeratic sandstone, shale, and coal. It is the least productive of the four confined aquifers in the Grand Junction area. Lohman (1965) states that "The sandstone beds of the Burro Canyon Formation and Dakota Sandstone are tightly cemented, lenticular and generally thin, hence they yield only small amounts of water, generally under insufficient head to flow at the surface." Depth to the top of the Dakota Sandstone in the vicinity of the processing site ranges from 70 to 170 ft (21 to 52 m) below land surface.

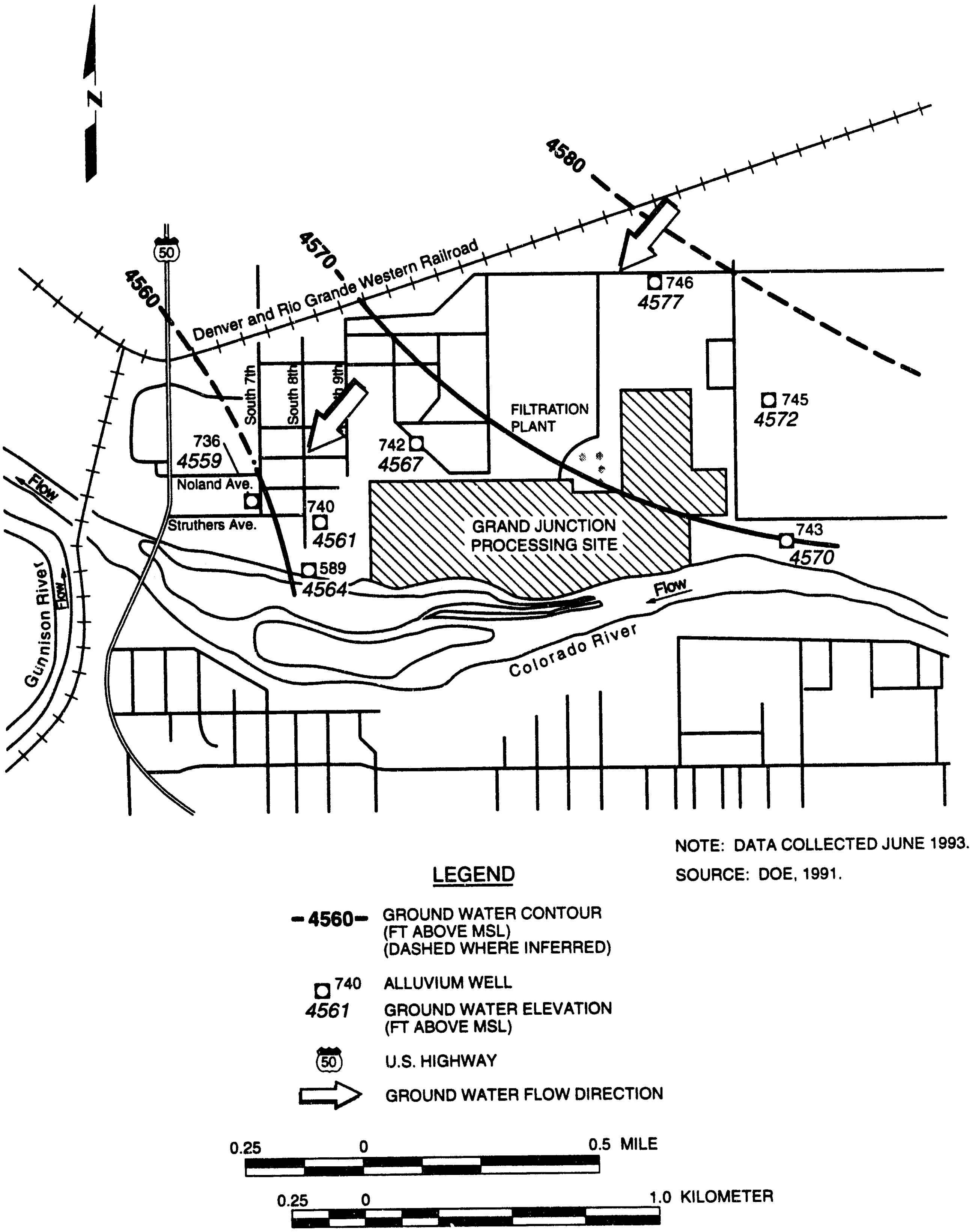
2.6 OCCURRENCE AND MOVEMENT OF GROUND WATER

Ground water beneath the processing site occurs in the alluvium, Mancos Shale, and Dakota Sandstone. The alluvium is the uppermost aquifer. Although the upper, weathered Mancos Shale is saturated beneath the processing site, it functions as an aquitard.

The depth to ground water in the alluvium ranges from approximately 20 ft (6 m) to less than 4 ft (1 m) in areas closest to the Colorado River, based on water level measurements from on-site monitor wells and piezometers. The water table surface in shallow monitor wells at the processing site is shown in Figure 2.6. Historical data indicate that the local hydraulic gradient is predominantly to the southwest.

Ground water levels beneath the site fluctuate from 2 to 5 ft (0.6 to 1.5 m) annually and are lowest during the fall and winter months. These variations in the water table surface are not uniform beneath the site; the largest fluctuations are in wells that are closest to the Colorado River and most affected by river stage fluctuations.

Regionally, the Mancos Shale is a low-permeability formation that is not water-bearing or transmits only very limited quantities of water (Cooley et al., 1969; Lohman, 1965). Slug-withdrawal tests conducted in monitor wells completed in the Mancos Shale confirm that it has a low hydraulic conductivity. *In situ* test results indicate that hydraulic conductivity generally decreases with depth within the Mancos Shale. At the processing site, ground water in the Mancos Shale was encountered in more than 10 monitor wells (DOE, 1986). One well (735) encountered artesian flow that lasted for 10 minutes, but additional drilling downdip from that well encountered nonartesian conditions. These



NOTE: DATA COLLECTED JUNE 1993.

SOURCE: DOE, 1991.

FIGURE 2.6
WATER TABLE CONTOUR MAP OF THE ALLUVIUM IN THE VICINITY OF THE GRAND JUNCTION, COLORADO, PROCESSING SITE

borings indicate that the thin Mancos Shale sandstone units, which are the source of local artesian flow, are discontinuous (DOE, 1986). The general direction of ground water flow within the Mancos Shale appears to be to the northeast. However, due to the discontinuous nature of the ground water flow system (i.e., presence of fractures and discontinuous sandstone beds), a precise flow direction cannot be determined.

Ground water flow directions in the Dakota Sandstone have not been determined because of a lack of borings into the unit. Flow in the unit, however, may be to the north, based on the dip of the beds. The probable recharge area is the outcrop of the Dakota Sandstone south of the site (Lohman, 1965).

2.7 CURRENT GROUND WATER USE

A recent well records search and field reconnaissance were conducted for the Grand Junction processing site and vicinity. The results of this investigation were consistent with past water use surveys. Detailed examination of current data bases and intensive field reconnaissance downgradient and crossgradient from the processing site indicate that there are no known users of alluvial ground water near the processing site that could be influenced by processing site-related contamination.

In the vicinity of the site and the majority of the Grand Valley area, surface water supplies the municipal and industrial needs. Shallow ground water is not used for three reasons: 1) the poor quality of the water, 2) the low yield of the shallow aquifers, and 3) the ready availability of alternate water supplies. Most of the water for the Grand Valley originates as surface water on Grand Mesa.

The Mancos Shale is not considered a source of ground water in the vicinity of Grand Junction. The underlying Dakota Sandstone is the uppermost artesian aquifer in the vicinity of tailings. No registered domestic use or other private wells are known to be completed in the Dakota Sandstone within the potentially affected hydrogeologic environment of the tailings.

The usual water supplies for Grand Junction are obtained from the Juniata and Purdy Mesa reservoirs. During dry spells, Grand Junction sometimes uses Gunnison River water. The intake is approximately 1.0 mi (1.6 km) upstream from the confluence of the Gunnison and Colorado Rivers. The Ute Water District uses Colorado River water during dry spells, but its intake is just upstream from the town of Palisade and therefore upstream from the mill site.

2.8 FUTURE GROUND WATER USE

Future use of shallow ground water in the affected hydrogeologic environment will probably be minimal because of the availability of city water and poor quality of ground water in the alluvial aquifer (discussed in Section 3.0).

3.0 EXTENT OF CONTAMINATION

The uranium mill at Grand Junction was established by the Climax Uranium Company. It began operating in 1951 (Merritt, 1971). According to Merritt (1971) this mill was the first American mill designed primarily for the production of uranium with vanadium as a by-product. The milling process used at this site was somewhat more complex than that at newer mills. The complexity of the milling process is reflected in the relatively large number of chemicals used in the milling process (Table 3.1).

Table 3.1 Major chemicals used at the Grand Junction mill

Inorganics	Organics
Sulfuric acid	Number 2 fuel oil
Hydrochloric acid	Di(2-ethylhexyl) phosphoric acid
Sodium chlorate	Tributyl phosphate
Sodium chloride	Ammonia
Sodium carbonate	Tertiary amines
Hydrogen peroxide	
Powdered iron metal	

The chemicals used in the milling process and the resultant dissolution of many constituents from the raw ore (including uranium and vanadium) generated a large volume of acidic waste material, or "tailings." These tailings contain significant amounts of water-soluble radiologically and chemically hazardous constituents. Ground water was screened for the organic constituents listed in 40 CFR Part 264 (1993), Appendix IX, in 1988. No organic contamination, as represented by the Appendix IX analyte list, was found at the site. However, an organic solvent extraction process was used to recover uranium from the pregnant solution during the milling process at the site. Additional sampling and analyses would be needed to rule out the possibility of organic contamination. Acidic pore water from the tailings has infiltrated into and variably contaminated the subjacent alluvial ground water system at Grand Junction.

As previously discussed, the bedrock unit that underlies the alluvium at Grand Junction is the Mancos Shale. Because water quality data for the Mancos are limited, it is not clear whether the unit has been affected by contaminated alluvial ground water. The Mancos Shale acts as an aquiclude between the overlying alluvium and the underlying Dakota Sandstone/Burro Canyon Formation (Figure 2.4). The Mancos Shale is not a significant water-bearing unit in the Grand Junction area; therefore, the ground water chemistry of the Mancos Shale is not discussed further.

All available DOE water quality data from the wells shown in Figure 2.5 were used to characterize the plume geometry and the geochemical processes active at the site. The remainder of this section will identify and discuss the following parameters: 1) background ground water quality in the alluvial aquifer; 2) the extent and magnitude of contamination of the alluvium by milling-related activities; 3) the milling-related constituents that are of concern to human health and the environment (contaminants of potential concern); and 4) the fate and transport characteristics of the contaminants of potential concern.

3.1 BACKGROUND WATER QUALITY DATA SUMMARY

Background ground water chemistry is defined as the quality of ground water that would exist at this UMTRA Project site if the milling had not taken place. Under this definition, other sources of ground water contamination (for example, industrial or domestic sources) that have affected the water quality of the aquifer(s) at this site would be considered part of the background quality. Upgradient DOE monitor wells 745 and 746 have been selected as the monitor wells that most likely sample background ground water (Figure 2.5). The possibility that contaminants from industrial sites and/or tailings-related vicinity properties in Grand Junction have affected the chemistry of the ground water sampled by monitor wells 745 and 746 cannot be ruled out on the basis of the currently available data.

Table 3.2 summarizes the minimum, median, and maximum values found for chemical parameters in the background ground water and contaminated ground water for the Grand Junction processing site.

The pH of the background ground water ranges from 6.7 to 7.5 and has a median value of 7.0. Available platinum electrode measurements of the reduction-oxidation (redox) state of the background ground water are of questionable reliability. Therefore, the redox state of the alluvial aquifer is not well known. Dissolved oxygen measurements may be more reliable; the limited data available suggest that the background ground waters are oxygen-depleted. Slightly reducing conditions in this alluvial aquifer are consistent with the presence of significant amounts of total organic carbon (TOC) in the ground water from these wells (averaging over 100 milligrams per liter [mg/L] in monitor well 746). However, organics in ground water upgradient of the UMTRA Project site may be from industrial or other anthropogenic sources.

The total dissolved solids (TDS) in background ground water samples from monitor wells 745 and 746 range from approximately 3000 to 7200 mg/L. The background alkalinity (as mg/L $CaCO_3$) ranges from 343 to 439. Despite the relatively high alkalinity of these waters, the dominant anionic species in the background ground water is sulfate (median value = 2800 mg/L). The dominant cations in the background ground waters are sodium, magnesium, and calcium; median concentrations in the background ground water are 659 mg/L sodium, 391 mg/L magnesium, and 532 mg/L calcium. Geochemical modeling of background ground water from monitor wells 745 and 746 with the

Table 3.2 Summary of filtered ground water quality at Grand Junction, Colorado

Constituent	Frequency of detection[a]	Observed Minimum (mg/L)	Observed Median[b] (mg/L)	Observed Maximum (mg/L)
Inorganic constituents				
Aluminum				
Background[c]	9/26	0.048	-	0.38
Plume[d]	5/23	0.004	-	0.51
Ammonium[e]				
Background	23/32	<0.03	0.2	0.6
Plume	23/23	166	357	521
Antimony				
Background	4/22	<0.003	-	0.012
Plume	2/23	<0.003	-	0.012
Arsenic[e]				
Background	7/30	0.001	-	0.04
Plume (584)	6/6	0.007	0.08	0.18
Barium				
Background	10/26	<0.002	-	0.02
Plume	11/21	0.002	-	0.30
Beryllium				
Background	0/16	<0.001	-	<0.01
Plume	0/3	<0.005	-	<0.005
Boron				
Background	16/16	0.36	0.59	0.83
Plume	15/15	0.34	0.57	0.71
Bromide				
Background	4/10	0.1	-	0.6
Plume	1/1	-	-	471
Cadmium[e]				
Background	7/26	<0.001	-	0.04
Plume (584)	4/4	0.073	0.12	0.42
Calcium[e]				
Background	30/30	325	445	595
Plume	33/33	360	545	654

Table 3.2 Summary of filtered ground water quality at Grand Junction, Colorado (Continued)

Constituent	Frequency of detection[a]	Observed Minimum (mg/L)	Observed Median[b] (mg/L)	Observed Maximum (mg/L)
Chloride[e]				
Background	30/30	306	598	2400
Plume	33/33	490	791	970
Chromium				
Background	5/26	<0.003	-	0.15
Plume	20/32	<0.001	0.01	0.03
Cobalt[e]				
Background	2/22	<0.003	-	0.01
Plume (584)	6/6	0.05	0.14	0.66
Copper[e]				
Background	10/26	0.003	-	0.03
Plume	22/33	<0.001	0.02	0.20
Cyanide				
Background	0/20	<0.01	-	<0.01
Plume	0/13	<0.01	-	<0.01
Fluoride[e]				
Background	26/26	0.6	1.0	1.7
Plume (581)	5/5	4.3	4.6	4.8
Iron[e]				
Background	23/30	<0.005	0.4	2.2
Plume (581, 585, 586)	21/21	1.3	11	16
Lead				
Background	2/24	<0.001	-	0.01
Plume	1/17	<0.001	-	0.01
Magnesium				
Background	30/30	210	391	570
Plume	33/33	25	282	620
Manganese[e]				
Background	30/30	0.9	1.3	2.3
Plume (583, 584, 585, 586)	26/26	1.8	4.1	10

Table 3.2 Summary of filtered ground water quality at Grand Junction, Colorado (Continued)

Constituent	Frequency of detection[a]	Observed Minimum (mg/L)	Observed Median[b] (mg/L)	Observed Maximum (mg/L)
Mercury				
Background	1/22	<0.0001	-	0.0002
Plume	3/23	<0.0001	-	0.0004
Molybdenum[e]				
Background	28/28	0.01	0.11	0.23
Plume (583, 584, 585, 586)	26/26	0.13	0.28	0.53
Nickel[e]				
Background	5/26	<0.006	-	0.12
Plume (584)	3/3	0.28	0.32	0.38
Nitrate				
Background	15/32	<0.1	-	16
Plume	11/28	<0.01	-	50
Phosphate				
Background	4/14	<0.05	-	0.1
Plume	0/10	<0.1	-	<0.1
Potassium[e]				
Background	30/30	4.2	8.1	12
Plume	33/33	49	96	120
Selenium				
Background	16/32	<0.001	-	0.19
Plume	13/33	<0.002	-	0.24
Silica				
Background	16/16	8	17	18
Plume	20/20	9	17	29
Silver				
Background	1/20	<0.002	-	0.01
Plume	4/18	<0.002	-	0.004
Sodium[e]				
Background	30/30	345	659	910
Plume	33/33	520	950	1210

Table 3.2 Summary of filtered ground water quality at Grand Junction, Colorado (Continued)

Constituent	Frequency of detection[a]	Observed Minimum (mg/L)	Observed Median[b] (mg/L)	Observed Maximum (mg/L)
Strontium				
Background	32/32	3.2	5.2	7.1
Plume	18/18	3.6	4.7	7.3
Sulfate[e]				
Background	32/32	1450	2800	11,000
Plume (583, 584, 585, 586)	26/26	3100	3945	4,900
Sulfide				
Background	7/12	<0.1	0.4	40
Plume	2/5	<0.1	-	0.2
Thallium				
Background	0/16	<0.005	-	<0.1
Plume	0/3	<0.1	-	<0.1
Tin				
Background	4/22	<0.005	-	0.11
Plume	3/18	<0.005	-	0.008
Uranium[e]				
Background	26/26	0.017	0.046	0.072
Plume (585, 586)	4/4	0.29	0.30	0.45
Vanadium[e]				
Background	8/28	<0.005	-	0.11
Plume (584)	6/6	5.2	7.1	14
Zinc[e]				
Background	7/26	<0.002	-	1.0
Plume (584)	6/6	2.6	4.5	37

Table 3.2 Summary of filtered ground water quality at Grand Junction, Colorado (Concluded)

Constituent	Frequency of detection[a]	Observed Minimum (pCi/L)	Observed Median[b] (pCi/L)	Observed Maximum (pCi/L)
Radionuclides				
Lead-210				
Background	0/4	<1.5	-	<1.5
Plume	4/10	<1.5	-	2.8
Polonium-210				
Background	0/4	<1.0	-	<1.0
Plume	1/10	<1.0	-	1.1
Radium-226[e]				
Background	26/32	0.0	0.1	2.3
Plume	19/22	0.0	2.1	29
Thorium-230				
Background	18/22	0.0	0.1	0.6
Plume	6/17	0.2	-	5.4
Uranium-234[e]				
Background	6/6	17	21	35
Plume	10/10	23	56	118
Uranium-238[e]				
Background	6/6	11	15	27
Plume	10/10	23	58	116

[a] Frequency of detection = Number of measurements above laboratory detection limit/total number of measurements.

[b] Calculation of the median—the 50th percentile of the data—requires that more than 50 percent of the measurements be above detection. A dash "-" in the median column indicates that the median cannot be calculated.

[c] Background concentrations are from DOE monitor wells 745 and 746 (both sampled 1985-1993).

[d] Plume concentrations are from DOE monitor wells 583 and 584 (sampled 1983-1985); 581, 585, and 586 (sampled 1983-1989). Summary statistics are from all five wells unless otherwise noted.

[e] Constituent concentration in plume wells is statistically elevated above background.

numerical code PHREEQE (Parkhurst et al., 1980) indicates that they are at or near saturation with respect to calcite ($CaCO_3$), magnesite ($MgCO_3$), gypsum ($CaSO_4$), and fluorite (CaF_2). Equilibration with these minerals would explain the relatively high concentrations of calcium, magnesium, alkalinity, sulfate, and fluoride (Table 3.2) in these background ground waters.

Trace constituents such as arsenic, uranium, vanadium, and selenium are also present at noteworthy levels in the background ground water (Table 3.2). Uranium concentrations are particularly noteworthy, as the median value present in the background ground water was 0.046 mg/L, slightly above the proposed UMTRA Project maximum concentration limit (MCL) for uranium.

3.2 MAGNITUDE AND EXTENT OF SITE-RELATED GROUND WATER CONTAMINATION

Alluvial ground water at the Grand Junction UMTRA Project site has been contaminated by the influx of acidic leachate from the overlying tailings. Chemical analysis of leachate from lysimeters installed just above or below the tailings/alluvium interface reveals that the tailings leachate was significantly enriched in many major, minor, and trace constituents relative to background alluvial ground water (Table 3.3). The chemical interaction of the tailings leachate with the alluvial system produced contaminated alluvial ground water. This contaminated water, or "plume," is migrating down the ground water flow path, subparallel to the Colorado River (Figure 3.1). Monitor wells downgradient from the site commonly have levels of ammonium, uranium, chloride, and sulfate that are above background levels. Ammonium is strongly adsorbed by clays and is not very mobile, however, and the migration of this constituent away from the site has lagged well behind the more mobile species such as sulfate, chloride, or uranium. The current downgradient extent of the contaminated ground water cannot be precisely defined because of 1) the lack of recent chemical data from on-site and some downgradient monitor wells; and 2) the masking effects of the high background levels of many of the most mobile tracer constituents (for example, uranium, sulfate, and chloride) in the alluvium (Table 3.2).

The water quality data from monitor wells 581, 583, 584, 585, 586, 589, 740, 736, and 733 indicate that the wells farthest downgradient from the site, wells 736 and 733, show lower levels of potential site-related contamination than do the wells closer to the site. For risk assessment purposes, these two wells were removed from further consideration.

Data from the other wells (581, 583, 584, 585, 589, 740, and 590) were analyzed to identify where, at the present time, the worst levels of contamination occur and to quantify those levels. The analysis was complicated by the fact that the wells on the site have not been sampled recently and, in fact, no longer exist. On-site concentrations of milling-related contaminants between 1983 and 1989 were generally higher than off-site levels during the same time period, as well as higher than subsequent off-site levels measured

Table 3.3 Typical Grand Junction tailings lysimeter and background water quality data

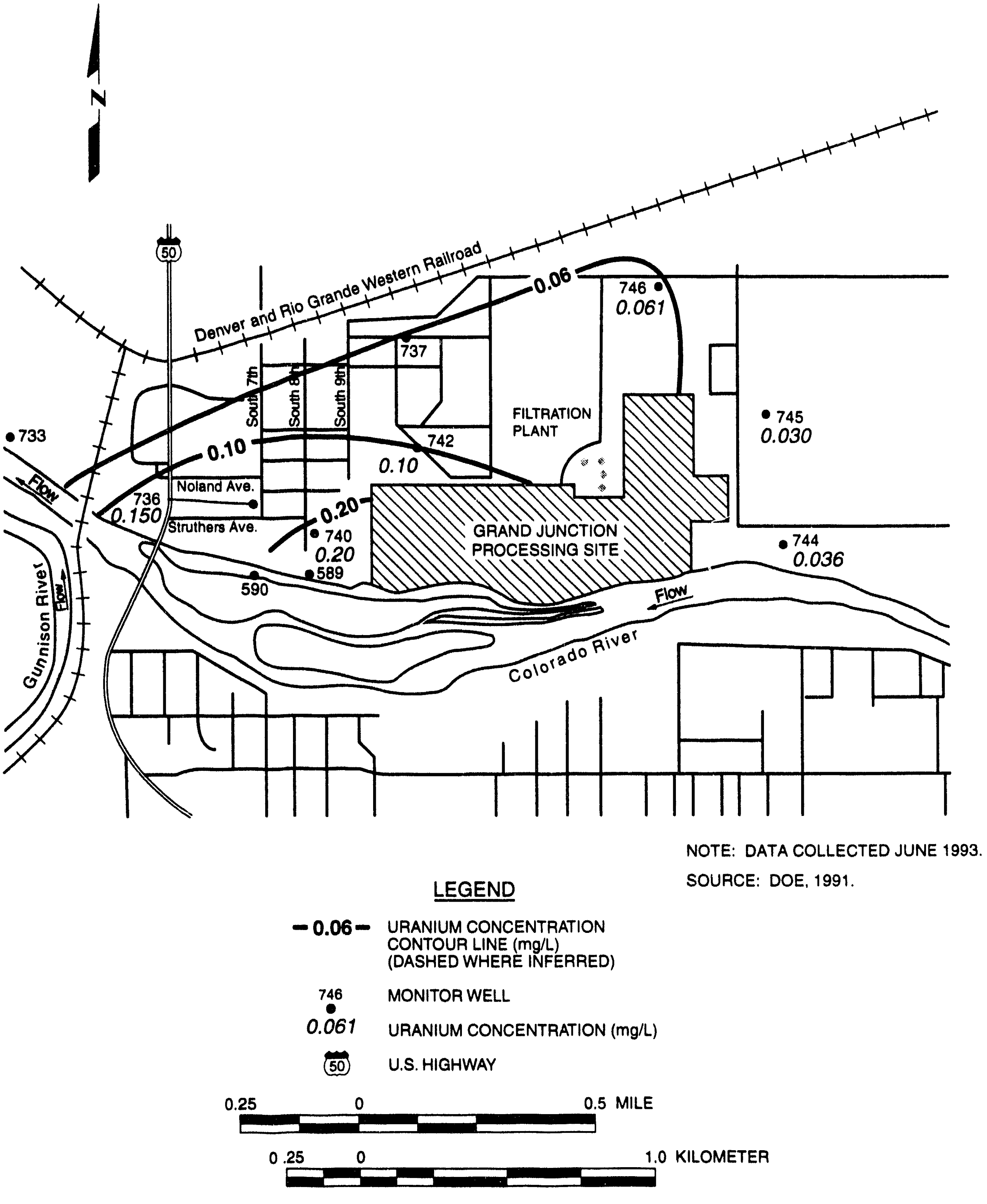
Parameter	Lysimeter 550[a]	Lysimeter 552[a]	Lysimeter 553[a]	Background 745[b]	Background 746[c]
pH	6.77	NA	4.25	7.32	7.06
Ec	11,500	NA	10,000	3,080	7,340
Temperature (°C)	26.0	NA	21.0	12.2	14.9
Alkalinity	770	NA		485	425
TDS	10,156	NA	8,274	3,550	6,820
Aluminum	<0.50	82.9	25.3	NA	<0.05
Ammonium	897	NA	780	0.4	0.2
Antimony	0.020	<0.010	0.013	NA	0.004
Arsenic	0.090	0.050	0.640	<0.005	<0.05
Boron	0.60	NA	1.00	NA	0.68
Cadmium	0.0071	0.3500	0.2700	NA	<0.001
Calcium	470	NA	670	330	488
Chloride	1,450	NA	1,100	404	689
Chromium	0.10	0.10	0.10	NA	<0.01
Cobalt	0.20	0.70	0.60	NA	<0.03
Copper	0.10	2.70	2.90	NA	<0.01
Fluoride	4.1	NA	12.8	0.8	1.7
Iron	7.60	1.50	53.0	0.52	<0.03
Lead	0.016	0.080	0.033	<0.003	<0.03
Magnesium	410	NA	275	221.0	495
Manganese	7.50	8.90	7.60	0.96	1.32
Mercury	<0.0001	<0.0001	<0.0001	NA	<0.0002
Molybdenum	0.90	<0.50	<0.50	0.04	0.15
Nickel	0.80	4.20	1.80	NA	<0.04
Nitrate	<0.1	NA	0.2	<1	9.7
Phosphate	0.1	NA	0.3	NA	0.1
Potassium	180	NA	140	4.6	9.19
Selenium	0.21	0.8	0.08	<0.005	0.11
Silica	27.5	NA	82.5	16.7	18.3

Table 3.3 Typical Grand Junction tailings lysimeter and background water quality data (Concluded)

Parameter	Lysimeter 550[a]	Lysimeter 552[a]	Lysimeter 553[a]	Background 745[b]	Background 746[c]
Silver	<0.0001	0.0016	0.0004	NA	<0.01
Sodium	1,145	NA	670	401.0	844
Strontium	4.20	5.50	4.80	3.41	6.57
Sulfate	2,307	NA	2,540	1,790	3,600
Tin	0.010	<0.010	<0.010	NA	<0.03
Uranium	2.897	3.050	0.180	0.030	0.060
Vanadium	1.00	0.40	2.90	NA	<0.01
Zinc	0.50	6.70	7.00	NA	<0.05

[a] Lysimeter water quality data are a composite of August 1989 and October 1989 data.
[b] Background water quality data from February 1993.
[c] Background water quality data from February 1992.

NA = Not analyzed.



NOTE: DATA COLLECTED JUNE 1993.

SOURCE: DOE, 1991.

FIGURE 3.1
DISTRIBUTION OF URANIUM IN ALLUVIAL GROUND WATER IN THE VICINITY OF THE GRAND JUNCTION, COLORADO, PROCESSING SITE

between 1991 and 1993. Trend analyses of five constituents associated with uranium milling—chloride, sulfate, ammonium, uranium, and molybdenum—suggest that molybdenum concentrations on the site decreased by about half between 1983 and 1989. On-site sulfate concentrations may also be decreasing, but only slightly. Chloride, ammonium, and uranium concentrations were steady. Except for molybdenum, the on-site data do not indicate that water quality under the site was improving at the time that sampling ceased.

The off-site wells located near, but downgradient from, the site (589, 590, and 740) were also studied for possible time trends in concentration levels. Between 1983 and 1993, chloride and sulfate levels in well 589 appear to have peaked and may be starting to decline. Uranium levels in this well are clearly decreasing with time. In the two other off-site wells, 590 and 740, located slightly farther from the site, chloride and sulfate concentrations appear to be increasing, but uranium is fairly steady with time. Although wells located off the site show trends over time between 1983 and 1993, these trends do not necessarily agree in magnitude or direction. Based on these analyses, it was concluded that currently the highest concentrations of milling-related contaminants in the alluvial aquifer are probably still under the site itself, at levels comparable to those last measured in 1989. Ground water quality in on-site wells 581 and 583 through 586 from 1983 to 1989 is summarized in Table 3.2.

3.3 CONTAMINANTS OF CONCERN

The data presented in Sections 3.1 and 3.2 are summarized in Table 3.2. These data were used to compile a list of contaminants of potential concern for the assessment of human health at the Grand Junction site. A constituent was placed on the list of contaminants of potential concern (Table 3.4) if concentrations of the contaminant in on-site monitor wells were, on average, higher than those in the off-site upgradient wells at the 0.05 level of significance. The statistical comparison was made using a nonparametric Mann-Whitney test.

The constituents identified in column 1 of Table 3.4 were screened for their impact on human health using the criteria discussed below to develop a final list of contaminants of potential concern for human health. Because environmental effects differ from effects on human health, the complete column 1 list of contaminants will be evaluated in the ecological assessment presented in Section 7.0. Several constituents that were detected above background levels were deleted from the final list of contaminants of potential concern for human health because they are essential nutrients and the levels at which they were detected are within nutritional ranges. These chemicals are calcium, chloride, and potassium.

Table 3.4 Contaminants of potential concern for human health evaluation

Contaminants that exceed background levels	Contaminants in nutritional range	Contaminants of low toxicity and/or high dietary range	Contaminants of potential concern
Ammonium		Ammonium	
Arsenic			Arsenic
Calcium	Calcium		
Cadmium			Cadmium
Chloride	Chloride		
Cobalt			Cobalt
Copper		Copper	
Fluoride			Fluoride
Iron			Iron
Manganese			Manganese
Molybdenum			Molybdenum
Nickel			Nickel
Potassium	Potassium		
Radium-226			Radium-226
Sodium		Sodium	
Sulfate			Sulfate
Uranium			Uranium
Vanadium			Vanadium
Zinc			Zinc

Bromide has been analyzed only once in the plume wells. Although the detected concentration is elevated above background concentrations, bromide was not included for evaluation as a contaminant of potential concern because of the significant uncertainty (i.e., only one data point from one monitor well) associated with the bromide data base.

Final screening of the remaining contaminants was based on very low toxicity and relatively high normal dietary intake compared to the values detected. These criteria were used to screen out ammonium, copper, and sodium.

After screening based on the criteria described above, the contaminants remaining as contaminants of potential concern were arsenic, cadmium, cobalt, fluoride, iron, manganese, molybdenum, nickel, radium-226, sulfate, uranium, vanadium, and zinc. These constituents form the basis of the human health portion of the risk assessment for Grand Junction.

3.4 CONTAMINANT FATE AND TRANSPORT

Although the aqueous speciation of a constituent in solution is one of the major determinants of its mobility in an aquifer, speciation can also influence the toxicity of some constituents. For example, trivalent arsenic species are more toxic to humans than arsenic in the pentavalent state. To determine the probable predominant species for the contaminants of potential concern, the geochemical code PHREEQE (Parkhurst et al., 1980) was used to model the ground water chemistry of plume-affected alluvial ground water. Although the redox state of the ground water at Grand Junction is not well defined, the dominant solution species for the contaminants of potential concern at an Eh of 150 millivolts (mV) are listed in Table 3.5. An Eh of 150 mV is a realistic estimate given the overall chemistry of the alluvial aquifer.

3.4.1 Fate and transport of metallic contaminants of potential concern

The solubility and mobility of metals and metalloids (for example, iron, chromium, arsenic, and selenium) vary dramatically as a function of water chemistry (for example, pH, Eh, and ionic strength), aquifer matrix composition, and the chemical characteristics of the contaminant. Nevertheless, some basic similarities in the aqueous chemistry of many metallic contaminants allow us to group the contaminants of potential concern at Grand Junction into constituent groups that have similar fate and transport characteristics.

All of the metallic and semimetallic contaminants are pH-sensitive and can be solubilized and transported by ground water under acidic, low-pH conditions. Some of these metals (for example, zinc, cadmium, and nickel) are relatively insensitive to aqueous Eh conditions but form soluble cationic species under acidic (low-pH) ground water conditions. These contaminants will generally reprecipitate (for example, as hydroxide or carbonate compounds) or be readsorbed by the aquifer matrix once the pH and/or alkalinity of the affected water is increased to near background levels by reacting with the aquifer matrix or by mixing with uncontaminated water.

Other metals and metalloids of concern are sensitive to pH and Eh conditions (for example, iron, manganese, arsenic, uranium, and vanadium), and once adsorbed or precipitated they can be remobilized by a significant change in the ambient state of either of these important ground water/aquifer parameters. At the Grand Junction site, the dominant factor on-site controlling these constituents appears to be the effects of contaminant-rich acid tailings leachate.

Iron and manganese

Iron and manganese were solubilized from the tailings and subpile aquifer matrix by the acidic tailings pore solutions that interacted with them. Arsenic,

Table 3.5 Aqueous species of contaminants of potential concern in the alluvial aquifer at Grand Junction, Colorado[a]

Contaminant of potential concern	Nomenclature	Aqueous species	Valence state	Molar percent
Arsenic	Arsenate	$HAsO_4^{2-}$	As(V)	81
	Arsenate	$H_2AsO_4^-$	As(V)	19
Cadmium	Cadmium	Cd^{2+}	Cd(II)	29
	Cadmium sulfate	$CdSO_{4AQ}$	Cd(II)	23
	Cadmium chloride	$CdCl^+$	Cd(II)	22
	Cadmium bicarbonate	$CdHCO_3^+$	Cd(II)	9
	Cadmium carbonate	$CdCO_{3AQ}$	Cd(II)	8
	Cadmium disulfate	$Cd(SO_4)_2^{2-}$	Cd(II)	8
	Cadmium dichloride	$CdCL_{2AQ}$	Cd(II)	1
Cobalt[b]	Cobalt carbonate[b]	$CoCO_{3AQ}$	Co(II)	80
	Cobalt[b]	Co^{2+}	Co(II)	20
Fluoride	Fluoride	F^-	F(I)	86
	Magnesium fluoride	MgF^+	F(I)	12
	Calcium fluoride	CaF^+	F(I)	2
Iron	Ferrous iron	Fe^{2+}	Fe(II)	72
	Ferrous sulfate	$FeSO_{4AQ}$	Fe(II)	28
Manganese	Manganese	Mn^{2+}	Mn(II)	66
	Manganese sulfate	$MnSO_{4AQ}$	Mn(II)	29
	Manganese bicarbonate	$MnHCO_3^+$	Mn(II)	3
	Manganese chloride	$MnCL^+$	Mn(II)	2
Molybdenum[c]	Molybdate	MoO_4^{2-}	Mo(VI)	100
Nickel	Nickel carbonate	$NiCO_{3AQ}$	Ni(II)	79
	Nickel	Ni_{2+}	Ni(II)	10
	Nickel sulfate	$NiSO_{4AQ}$	Ni(II)	5
	Nickel bicarbonate	$NiHCO_3^+$	Ni(II)	4
	Nickel dicarbonate	$Ni(CO_3)_2^{2-}$	Ni(II)	2

Table 3.5 Aqueous species of contaminants of potential concern in the alluvial aquifer at Grand Junction, Colorado[a] (Concluded)

Contaminant of potential concern	Nomenclature	Aqueous species	Valence state	Molar percent
Sulfate	Sulfate	SO_4^{2-}	S(VI)	68
	Calcium sulfate	$CaSO_{4AQ}$	S(VI)	12
	Magnesium sulfate	$MgSO_{4AQ}$	S(VI)	12
	Sodium sulfate	$NaSO_4^-$	S(VI)	4
	Ammonium sulfate	$NH_4SO_4^-$	S(VI)	4
Uranium	Uranyl tricarbonate	$UO_2(CO_3)_3^{4-}$	U(VI)	87
	Uranyl dicarbonate	$UO_2(CO_3)_2^{2-}$	U(VI)	13
Vanadium	Vanadium trihydroxide	$V(OH)_3^+$	V(IV)	80
	Vanadium oxide	VO^{2+}	V(IV)	8
	Vanadium oxide	$H_2V_2O_4^{2+}$	V(IV)	7
	Vanadium sulfate	$VOSO_{4AQ}$	V(IV)	4
	Vanadium fluoride	VOF^+	V(IV)	1
	Vanadate	$HV_2O_7^{3-}$	V(V)	77
	Vanadate	$H_2VO_4^-$	V(V)	20
	Vanadate	HVO_4^{2-}	V(V)	3
Zinc	Zinc	Zn^{2+}	Zn(II)	43
	Zinc sulfate	$ZnSO_{4AQ}$	Zn(II)	26
	Zinc bicarbonate	$ZnHCO_3^+$	Zn(II)	13
	Zinc carbonate	$ZnCO_{3AQ}$	Zn(II)	9
	Zinc disulfate	$Zn(SO_4)_2^{2-}$	Zn(II)	7
	Zinc dicarbonate	$Zn(CO_3)_2^{2-}$	Zn(II)	2

[a] Ground water quality analysis from well 586 (1989 sampling round) along with maximum concentrations of contaminants of potential concern (excluding sulfate) (Table 3.2) were used as input for the model. Aqueous species were calculated using the geochemical code MINTEQA2/PRODEFA2 (Allison et al., 1991). Select speciation information was taken from Brookins (1988).

[b] Estimated from Eh-pH diagram (Brookins, 1988) and the similar behavior of nickel and cobalt in an aqueous environment.

[c] Estimated from Eh-pH diagram (Brookins, 1988).

uranium, and vanadium can also be solubilized from the tailings and the aquifer matrix by low-pH conditions but will be discussed separately. Although the redox state of the alluvial aquifer as a whole is not well defined, the conditions in the shallow ground water on-site are probably relatively oxidizing. Under oxidizing conditions, iron and manganese will hydrolyze and precipitate as oxyhydroxides once the pH approaches neutral values. Although this process can occur at lower values of pH (5.0 to about 6.5), the kinetics of oxidation and hydrolysis are slower. The oxidation of manganese in particular is very sensitive to pH, and this element can persist in solution (as Mn^{2+}) under somewhat oxidizing ground water conditions if the pH is much below about 7.5 to 8.0.

Nickel, cobalt, and zinc

The precipitation of iron and manganese as hydroxides as acidity is neutralized by reacting with the aquifer matrix and by mixing with alkaline ground water can be of critical importance for the fate of many other contaminant trace elements. Iron and manganese hydroxides have a high affinity for many trace constituents, and the precipitation of their hydroxides can sweep these other, potentially toxic, metals from ground water (for example, cobalt, thorium, nickel, and zinc). Under the typical pH range (6.0 to 8.0) observed for the alkaline ground water in the alluvial aquifer, these trace constituents should rapidly approach background levels in downgradient ground water.

Cadmium

Cadmium will be rapidly removed by the precipitation of octavite ($CdCO_3$) and by hydrolysis reactions as the low pH of the tailings leachate is neutralized by alkaline ground water and calcite in the aquifer matrix. Dilution with background water will produce cadmium concentrations in downgradient ground water that are typically below detection limits. Elevated levels of cadmium should be restricted to the areas underneath or immediately adjacent to the tailings pile.

Radium

Radium solubility and mobility at the Grand Junction site should be extremely low. Radium forms an extremely insoluble sulfate compound and commonly coprecipitates with barite ($BaSO_4$). Radium also has a high sorption affinity for clays and for iron and manganese hydroxides.

Arsenic, uranium, molybdenum, vanadium

Some other contaminants of potential concern (for example, arsenic, uranium, molybdenum, and vanadium) commonly form stable anionic species under near neutral to high pH or alkaline conditions, and they will not precipitate immediately or be completely swept by the precipitation of iron and manganese hydroxides. These contaminants can be transported for significant distances under oxidizing, near neutral to high pH or alkaline ground water conditions before they are eventually absorbed by the matrix and/or diluted to background levels by mixing with uncontaminated ground water.

3.4.2 Fate and transport of nonmetallic contaminants of potential concern

Fluoride

Fluoride (F^-) is elevated relative to background levels in the tailings leachate and leachate-contaminated ground water at the Grand Junction site (Tables 3.2 and 3.3). Geochemical modeling of background ground waters, plume-affected ground water, and acidic tailings leachate indicated that all of these waters approached or slightly exceeded saturation with CaF_2. This suggests that the upper limit on fluoride concentrations in the alkaline, plume-contaminated, on-site and near-downgradient ground water will be set by the precipitation of fluorite. As the plume migrates farther downgradient, dilution with river water and adsorption of fluoride on aquifer sediments could reduce the concentration of this constituent below levels required to maintain equilibrium with fluoride.

Sulfate

Sulfate concentrations in the highly contaminated parts of the plume immediately adjacent to the tailings are controlled primarily by gypsum solubility. Farther downgradient, sulfate concentrations are controlled largely by physical processes such as dispersion and dilution. Reduction of sulfate to sulfide is also possible if the plume interacts with strongly reducing sediment or ground water. Adsorption reactions are not likely to have a significant effect on the sulfate concentrations in the shallow ground water because of the relatively high concentrations involved.

Currently, gypsum precipitation predominantly controls sulfate concentrations because the shallow ground water in and around the tailings piles is oversaturated with gypsum. After the tailings piles are removed, however, the ground water sulfate concentrations in and around the former piles will decrease. Eventually, as sulfate levels drop below gypsum saturation, the gypsum that has precipitated previously will begin to redissolve. The dissolution of gypsum will buffer the sulfate concentrations at fairly high levels at Grand Junction until the supply of gypsum is exhausted. At this point, natural flushing with background waters will substantially lower the sulfate concentrations in this area.

3.5 SURFACE WATER MONITORING

Surface water quality has been monitored for several years in the Colorado River in the vicinity of the site. The locations of the surface water sampling points are shown in Figure 3.2. Five locations have been sampled: one location upstream of the site, three locations adjacent to the site, and one location downstream of the site. Filtered samples were collected once, in 1991, from upstream of the site (location 423), adjacent to the site (locations 424 and

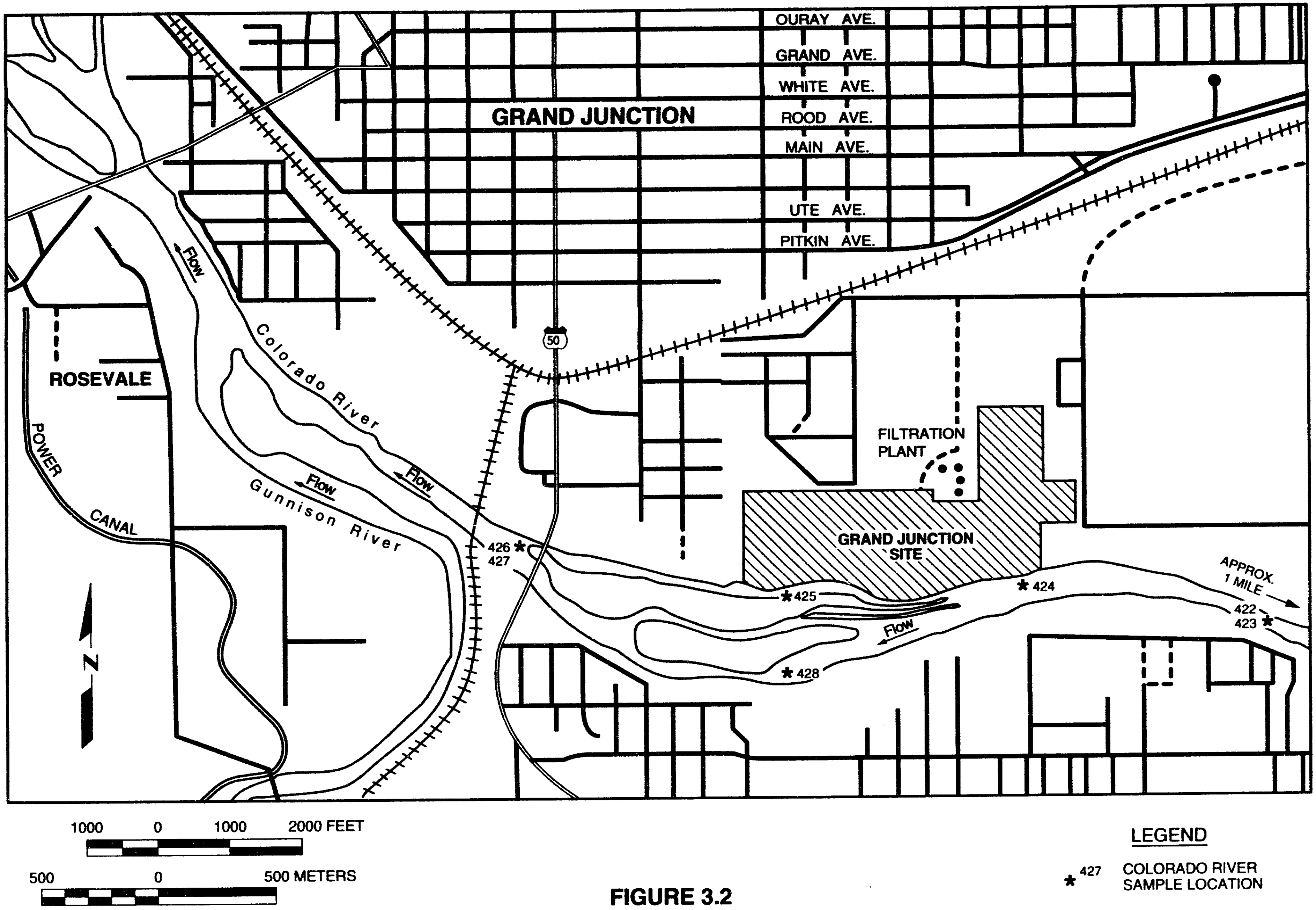


FIGURE 3.2
SURFACE WATER AND SEDIMENT SAMPLE LOCATIONS
NEAR THE PROCESSING SITE
GRAND JUNCTION, COLORADO

MAC: SITE/GRJ/BRA/BASE2-LAYERS/SURFACESAMPLOCS

425), and downstream of the site (location 427). Between 1991 and 1993, six rounds of unfiltered samples were collected from these four locations. At the upstream and downstream locations, the unfiltered samples were designated with location identifications of 422 and 426, respectively. One unfiltered sample has been collected to date from location 428, in 1993. The samples collected from 1991 through 1992 were analyzed for a full suite of analytes. The samples collected in September 1993 were analyzed for a select list of analytes (molybdenum, selenium, strontium, sulfate, and uranium). This list was selected prior to the development of this risk assessment. Sediment samples were also collected in September 1993 from the same five surface water locations; the results are discussed in Section 7.0.

Surface water data from the Colorado River show that most of the constituents detected at the adjacent and downstream locations were not greater than their respective background (upstream) concentrations. From the list of ground water constituents that are identified as exceeding background ground water quality (Table 3.4), only ammonium, copper, iron, radium-226, uranium, and vanadium were detected at concentrations above background at the adjacent locations and/or the downstream location. However, the concentrations were only slightly higher, and there is no trend in the data that suggests a relationship with the site.

Precipitation and snowmelt may have carried both dissolved and suspended constituents along surface drainages to the Colorado River. Metal constituents transported as dissolved species would have become diluted after discharging to these water bodies. Alternatively, dissolved species could have precipitated, becoming adsorbed to sediments or absorbed into biota with varying biochemical and geochemical conditions. Constituents transported from the processing site that were sorbed onto soil particles would have been deposited as sediments. Variations in geochemical conditions or biological action could release constituents adsorbed onto sediments into surface waters. Thus, deposited sediments could act as a source of site-related surface water contamination. However, as discussed further in Section 7.0, there is no compelling evidence suggesting that this is occurring now.

4.0 EXPOSURE ASSESSMENT

This section discusses and quantifies the potential exposures that could be incurred by current or future residents, recreational users, and others who use ground water or surface water contaminated by the Grand Junction processing site. The methodology used here is consistent with the latest EPA guidance on exposure assessments (EPA, 1989a), which recommends an analysis based on the reasonable maximum exposure under both current and future land-use conditions. The reasonable maximum exposure is defined as the highest exposure that can be reasonably expected to occur at the site.

4.1 POTENTIALLY EXPOSED POPULATION

Exposure can occur only if there is both a source of contamination and a mechanism of transport to a receptor population or individual. As discussed in Section 2.7, there is no current use of ground water contaminated by uranium processing at the Grand Junction site. Information obtained during searches of well records and field water well surveys indicates that no domestic, agricultural, or industrial wells are located within the alluvium, Mancos Shale, or Dakota Sandstone in the site vicinity.

Because there are no current human receptors of contaminated ground water, a future ground water use scenario must be assumed. This scenario evaluates domestic ground water use consistent with current household water use by the population in the region. The potentially exposed population includes individuals of the following age groups: infants (birth to 1 year old), children (1 to 10 years old), and adults (11 to 64 years old). These age groups were selected for the following reasons:

- Survey data for population variables such as age, weight, and daily water intake are available for these age groups.
- Toxicological variables are similar within these age groups, including responsiveness of sensitive subgroups (infants and children) to the contaminants of potential concern, toxicant intake to body weight ratios, and toxicokinetics.

4.2 EXPOSURE PATHWAYS

There is no current use of ground water in the site vicinity. It is unlikely that ground water will be used in the future for household purposes because of the existing public water supply system. However, consistent with EPA guidance, this risk assessment will assume hypothetical future use of ground water resources, including the use of ground water from the alluvium for drinking, cooking, and bathing. In addition to drinking water use, the potential exists that water from a future hypothetical well could be used to irrigate garden plants or to water livestock. Plants with roots in the alluvial aquifer or irrigated with water from this zone could take up and concentrate contaminants, forming a pathway to humans through plant consumption. The land use in the vicinity of the site is a mixture of residential, industrial/commercial, and agricultural. Thus, the potential exists that at some point in the future a resident could have a limited number of livestock to use as a food source. Using the livestock for food would create an exposure pathway to humans. In addition to exposure to ground water, the potential for exposure to surface water through recreational use (e.g., sportfishing and swimming) of the Colorado River also exists. Figure 4.1 provides a conceptual model for potential future ground water exposure pathways that could result from these uses.

4.2.1 Drinking water ingestion

Drinking water ingestion is generally the most significant exposure pathway for ground water contaminated with metals and other nonvolatile compounds. For this evaluation, drinking water consumption includes water consumed for drinking, as well as water used for food preparation (for example, reconstituted juices, soup, rice, and beans). For comparison of relative pathway significance, a screening level assessment of drinking water intake is shown in Table 4.1. These calculations are based on estimates of the maximum concentrations of contaminants within the plume (that is, the maximum detected concentration from the most contaminated plume wells).

4.2.2 Dermal absorption

Dermal absorption is the process by which chemicals coming into contact with skin are absorbed into blood vessels near the surface of the skin. Some compounds are absorbed easily in this manner, though metals do not possess the chemical properties that are conducive to skin absorption.

To evaluate this exposure route, a screening calculation was performed to determine whether a dermal absorption pathway would be significant compared to the drinking water pathway for the contaminants of potential concern. Since chemical-specific absorption factors are not available for these contaminants, it was assumed that they are absorbed across the skin at the same rate as water. This assumption will probably overestimate any potential contribution from dermal absorption. Additionally, the concentration in water was assumed to be the maximum detected concentration from the most contaminated plume wells, which also will overestimate exposure.

The results of the screening are given in Table 4.1. Based on these results, dermal absorption was eliminated from more detailed evaluation at this time because it contributed less than 1 percent of the total intake from drinking water for all constituents.

4.2.3 Ingestion of ground water-irrigated produce

This exposure route was also evaluated for its relative significance to the drinking water ingestion route. The results of the screening calculation are

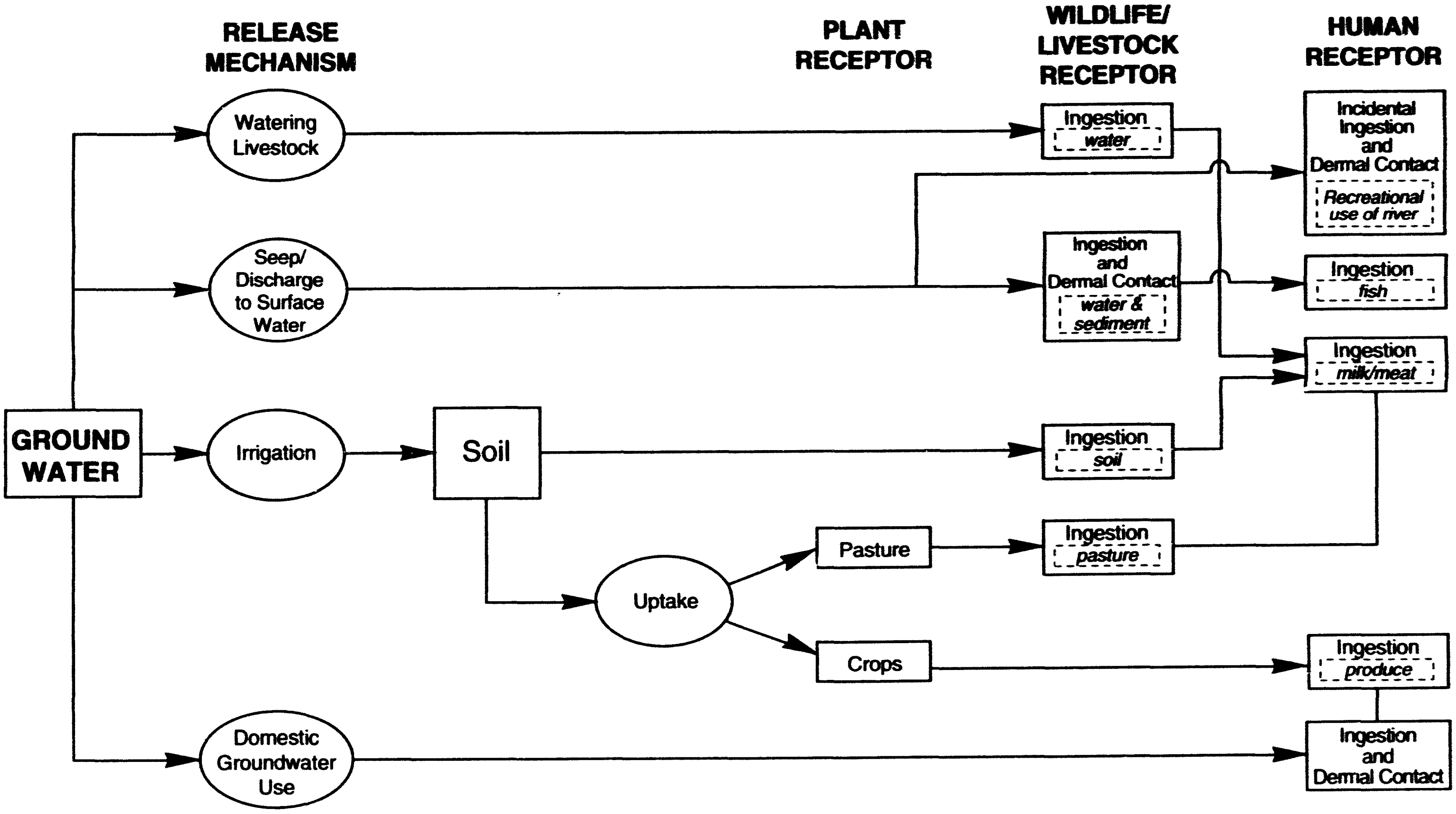


FIGURE 4.1
GRAND JUNCTION CONCEPTUAL MODEL

PREPARATION DATE: 6/25/94

MAC: SITE/GRJ/BRA/CONCEPTMOD

Table 4.1 Exposure dose calculations and equation definitions for ground water ingestion and dermal contact, Grand Junction UMTRA Project site, Grand Junction, Colorado

Contaminant of potential concern	Cw	Ground water exposure doses (mg/kg-day) Ingestion	Dermal contact	Ratio of dermal:ingestion[a]
Noncarcinogenic effects	**(mg/L)**			
Arsenic	0.18	4.9E-03	9.6E-06	0.002
Cadmium	0.42	1.2E-02	2.2E-05	0.002
Cobalt	0.66	1.8E-02	3.5E-05	0.002
Fluoride	4.8	1.3E-01	2.6E-04	0.002
Iron	16	4.4E-01	8.5E-04	0.002
Manganese	10	2.7E-01	5.3E-04	0.002
Molybdenum	0.53	1.5E-02	2.8E-05	0.002
Nickel	0.38	1.0E-02	2.0E-05	0.002
Sulfate	4900	1.3E+02	2.6E-01	0.002
Uranium	0.45	1.2E-02	2.4E-05	0.002
Vanadium	14	3.8E-01	7.4E-04	0.002
Zinc	37	1.0E+00	1.9E-03	0.002
Carcinogenic effects	**(pCi/L)**			
Arsenic	0.18	2.1E-03	4.1E-06	0.002
Radium-226	29	6.1E+05[b]	1.2E+03[b]	0.002
Uranium[c]	234	4.9E+06[b]	9.5E+03[b]	0.002

Equation definitions for exposure dose calculations

Ingestion of ground water

Chemicals

$$\text{Chronic daily intake (mg/kg-day)} = \frac{Cw \times IRw \times EF \times ED}{BW \times AT}$$

Radionuclides

$$\text{Lifetime intake (pCi/lifetime)} = Cw \times IRw \times EF \times ED$$

Dermal contact with ground water

Chemicals

$$\text{Chronic daily intake (mg/kg-day)} = \frac{(Cw \times SA \times Pc \times Cf) \times ET \times EF \times ED}{BW \times AT}$$

Radionuclides

$$\text{Lifetime intake (pCi/lifetime)} = Cw \times SA \times Pc \times Cf \times ET \times EF \times ED$$

Table 4.1 Exposure dose calculations and equation definitions for ground water ingestion and dermal contact, Grand Junction UMTRA Project site, Grand Junction, Colorado (Concluded)

Where:

Cw = Contaminant concentration in ground water (maximum concentration detected) (mg/L or pCi/L).
IRw = Ingestion rate for water (L/day) (2 L/day for an adult).
EF = Exposure frequency (350 days/year).
ED = Exposure duration (30 years for an adult).
BW = Body weight (70 kg for an adult).
AT = Averaging time (365 days x ED for noncarcinogens; 365 days x 70 years for carcinogens).
SA = Skin surface area [19,400 square centimeters (cm^2)].
Pc = Dermal permeability constant (0.001 cm/hour).
Cf = Conversion factor (0.001 L/cm^3).
ET = Exposure time (0.2 hour/day).

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[a] This value is calculated by dividing the dermal contact exposure dose by the ground water ingestion exposure dose.
[b] Picocuries per lifetime.
[c] Uranium-234 and uranium-238 combined.

shown in Table 4.2. The assumptions for this evaluation will probably overestimate the potential for exposure from this route, because it is assumed that this garden would be the source of all garden produce in the diet. The results of this screening show that for the contaminants of potential concern at this site, ingesting garden vegetables and fruit irrigated with contaminated ground water would lead to potential exposures of approximately 4 percent or less of that associated with drinking water ingestion. Thus, this pathway is eliminated from further evaluation, although the contribution of this additional source to site-related risks is discussed in Sections 6.1 and 6.2.

4.2.4 Ingestion of milk or meat from ground water-fed livestock

These pathways were eliminated from further consideration, because the concentrations of sulfate and TDS were so high that livestock would not likely survive chronic ingestion of the water. If the livestock cannot consume the water, there is no potential for bioaccumulation or transfer of contaminants ingested to meat tissue or milk. Further evaluation of the direct toxicity to livestock is presented in Section 7.0.

4.2.5 Ingestion of fish

Sportfishing occurs in the reaches of the Colorado River near the site. Ingestion of fish that may have accumulated site-related constituents is a potential exposure pathway.

To evaluate the fish ingestion exposure route, a screening calculation was performed to determine whether the contribution from fish ingestion would be significant compared to the drinking water pathway. Since no fish tissue samples have been collected from the Colorado River near the site, contaminant-specific bioconcentration factors (BCF) for freshwater fish tissue were used (EPA, 1992a; NUREG, 1986). Fish tissue concentrations were estimated for the contaminants of potential ecological concern identified for the Colorado River (refer to Section 7.3). These contaminants are ammonium, copper, fluoride, iron, radium-226, uranium, and vanadium. No fish BCFs are available for ammonium, fluoride, iron, and vanadium. Thus, no fish tissue concentrations can be estimated. The ingestion rate of fish is an annualized average for consumption of fin fish from recreational fishing (EPA, 1991). It assumes that half of the recreationally caught fish would be from the river. This is approximately one 8-ounce serving per week.

The results of the screening are given in Table 4.3. Based on the results, the fish ingestion exposure route is eliminated from more detailed evaluation, since it contributed to less than 4 percent of the total dose from drinking water.

4.2.6 Recreational use of the river

The potential exists for people to be exposed to the Colorado River in the vicinity of the site during recreational activities (for example, swimming,

Table 4.2 Exposure dose calculations and equation definitions for ground water-irrigated garden produce ingestion, Grand Junction UMTRA Project site, Grand Junction, Colorado

Contaminant of potential concern	Cw (mg/L)	Kd (L/kg)	Bv	Br	Garden produce ingestion exposures[a] (mg/kg-day)	Ratio of produce ingestion:water ingestion[b]
Noncarcinogenic effects						
Arsenic	0.18	5.86	0.04	0.006	2.1E-06	4.3E-04
Cadmium	0.42	14.9	0.55	0.15	1.8E-04	1.6E-02
Cobalt	0.66	1.94	0.02	0.007	1.4E-06	7.8E-05
Fluoride	4.8	0.0	0.06	6.0×10^{-3}	--	--
Iron	16	15.0	4.0×10^{-3}	1.0×10^{-3}	5.0E-05	1.1E-04
Manganese	10	16.5	0.25	0.05	2.1E-03	7.9E-03
Molybdenum	0.53	40.0	0.25	0.06	2.7E-04	1.9E-02
Nickel	0.38	12.2	0.06	0.06	2.0E-05	1.9E-03
Sulfate	4900	0	0.5	0.5	0.0E+00	0.0E+00
Uranium	0.45	0	0.0085	0.004	--[c]	--
Vanadium	14	50	0.0055	0.003	2.3E-04	5.9E-04
Zinc	37	12.7	1.5	0.9	4.3E-02	4.3E-02
Carcinogenic effects						
Arsenic	0.18	5.86	0.04	0.006	8.9E-07	4.2E-04
Radium-226	29	24.3	0.015	0.0015	3.9E+02[d]	6.4E-04
Uranium	234	0	0.0085	0.004	--	--

Equation definitions for exposure dose calculations

Ingestion of garden produce irrigated with ground water

Chemicals

$$\text{Chronic daily intake (mg/kg-day)} = \frac{Cw \times Kd \times Bv \text{ or } Br^{e} \times DF \times IRp \times FI \times EF \times ED}{BW \times AT}$$

Radionuclides

$$\text{Lifetime intake (pCi/lifetime)} = Cw \times Kd \times Bv \text{ or } Br^{e} \times DF \times IRp \times FI \times EF \times ED$$

Table 4.2 Exposure dose calculations and equation definitions for ground water-irrigated garden produce ingestion, Grand Junction UMTRA Project site, Grand Junction, Colorado (Concluded)

Where:

Cw = Contaminant concentration in ground water (maximum concentration detected) (mg/L or pCi/L).
Kd = Soil-water partition coefficient (L/kg); from Pacific Northwest Laboratory (PNL), 1989.
Bv = Soil-to-plant concentration ratio for vegetative portions of plants (unitless).
Br = Soil-to-plant concentration ratio for reproductive portions (fruits, tubers) of plants (unitless).
DF = Dry weight fraction of plant (0.066 unitless).
IRp = Ingestion rate for garden produce (0.05 kg/day for vegetative parts; 0.03 kg/day for reproductive parts).
Fi = Fraction of garden produce ingested from contaminated source (1.0 unitless).
EF = Exposure frequency (350 days/year).
ED = Exposure duration (30 years for an adult).
BW = Body weight (70 kg for an adult).
AT = Averaging time (365 days x ED for noncarcinogens; 365 days x 70 years for carcinogens).

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[a] Exposure doses shown are the sum of the vegetative parts plus the reproductive parts.

[b] This value is calculated by dividing the garden produce ingestion exposure dose by the ground water ingestion exposure dose.

[c] Value cannot be calculated because Kd is equal to zero.

[d] Picocuries per lifetime.

[e] Exposure doses due to vegetative parts and reproductive parts of garden produce are calculated separately, then summed for total intake.

Table 4.3 Exposure dose calculations and equation definitions for ingestion of fish from the Colorado River in the site vicinity, Grand Junction UMTRA Project site, Grand Junction, Colorado

	Colorado River upstream		Colorado River adjacent to site		Colorado River downstream		
	Estimated fish concentration	Estimated intake	Estimated fish concentration	Estimated intake	Estimated fish concentration	Estimated intake	Ratio of fish ingestion:ground water ingestion[a]
Constituent	mg/kg	mg/kg-day	mg/kg	mg/kg-day	mg/kg	mg/kg-day	
Copper	0.35	1.3E-04	0.47	1.7E-04	0.22	8.1E-05	NA[b]
Radium-226	47	1.3E+04	70	2.0E+04	80	2.3E+04	0.038
Uranium	0.0094	3.5E-06	0.0098	3.6E-06	0.012	4.4E-06	0.00037

Equation definitions for exposure dose calculations

Ingestion of fish

Chemicals

$$\text{Chronic daily intake (mg/kg-day)} = \frac{Cf \times IR \times EF \times ED \times Fi}{BW \times AT}$$

Radionuclides

$$\text{Lifetime intake (pCi/lifetime)} = Cf \times IR \times EF \times ED \times Fi$$

Where:

Cf = Contaminant concentration in fish (mg/kg; pCi/kg).
IR = Ingestion rate of fish (0.054 kg/day for an adult).
EF = Exposure frequency (350 days/year).
ED = Exposure duration (30 years for an adult).
Fi = Fraction ingested from contaminated source (0.5 unitless).
BW = Body weight (70 kg for an adult).
AT = Averaging time (365 days x ED for noncarcinogens).

No fish BCFs were available for ammonium, fluoride, and vanadium. Therefore, no estimated fish concentrations were calculated.
[a] The maximum fish ingestion exposure dose was used in calculating this ratio.
[b] Not applicable; constituent was not identified as a contaminant of potential concern in ground water.

wading, rafting, etc.). Some activities, such as swimming in the river, are not encouraged by the state; however, they are not specifically prohibited. Although exposure to constituents in the river is possible, an evaluation of the surface water data indicates that there are no significant differences between the upstream (background) concentrations and the concentrations detected adjacent and downstream of the site. Thus, further evaluation of potential exposure due to recreational use of the river is not warranted.

Summary

In summary, the results from all of the screening pathways (water ingestion, dermal contact with water, garden produce ingestion, fish ingestion, and recreational use of the river) indicate that drinking water ingestion is the dominant pathway. Therefore, only the drinking water pathway is further evaluated probabilistically in Section 4.4. The total contribution for any contaminant from all other sources is less than 5 percent, and is less than 1 percent for most. The potential for other pathways to increase site-related risk will be discussed qualitatively in Section 6.0.

4.3 EXPOSURE CONCENTRATIONS

The exposure concentration of a contaminant in ground water is defined as the concentration contacted by an individual over a specific period. In this evaluation, the contaminant concentrations are assumed to be in a steady state, although actual contaminant concentrations (and therefore exposures) are expected to decrease with time after the tailings are removed. Nonetheless, these estimates are reasonable for chronic exposure soon after surface remediation. Chronic exposure for noncarcinogens is considered to be exposure for any period longer than 7 years.

For noncarcinogens, exposure concentrations are evaluated as a probability of occurrence based on ground water data collected from monitor wells 581, 583, 584, 585, and 586 for the contaminants of potential concern. These wells have consistently shown the highest concentrations of most contaminants during the monitoring period (1983 to 1989). All of these wells are located on the processing site.

For constituents that show no net trend over time, distributions were derived from actual recorded water quality measurements, using data from the subset of wells that consistently had the highest concentrations of the constituent. The theoretical distribution reflects, to the extent possible, the same average concentration, standard deviation (spread), and pattern of occurrences (shape) as occurred in the actual water quality data. For constituents that show an obvious trend over time, the theoretical distribution is centered around the more recently observed concentrations. An increasing trend was incorporated for arsenic, using well 584 data. A decreasing trend in molybdenum was observed in all on-site wells but not incorporated. As a result, possible molybdenum exposure may have been overestimated. Stable distributions were used to model cadmium, cobalt, fluoride, iron, manganese, nickel, sulfate, uranium, vanadium, and zinc.

The distributions are all truncated; values below 0 mg/L or above the 99th percentile are disallowed. The lower limit of 0 mg/L reflects the impossibility of negative exposures. The truncation of the upper end of the distribution places a reasonable limit on possible exposures. For every contaminant, this highest allowable concentration was higher than the maximum observed concentration in the historical water quality data. The software package @RISK (Palisade Corporation, 1992) was used to generate the probability curves for the nonradioactive contaminants of potential concern. The results are shown in Figures 4.2 through 4.13.

The concentrations of radionuclides were represented by the same wells used to represent the inorganic constituents.

4.4 ESTIMATION OF INTAKE

Within the population of future residents, individuals are expected to vary with respect to water consumption habits, stable body weight, and length of time they reside in the potential contamination zone. Consequently, health risks associated with ground water consumption will vary among members of this population. To adequately describe the range of potential risks to the future population, naturally occurring variability in daily water intake, body weight, and residency time were incorporated in this assessment through probability distributions; these distributions were generated from United States public health and census documents. All distributions were truncated at the upper and lower 0.01 percentile. Within the hypothetical population, values disallowed through this truncation may occur with a probability of less than 1 in 10,000.

The potential toxicity of noncarcinogenic contaminants in drinking water depends primarily on long-term average daily consumption of the contaminant per kilogram of body weight. For probability distributions of noncarcinogens, chronic daily intake is calculated as follows:

$$\underset{\text{(mg/kg-day)}}{\text{Intake}} = \frac{\underset{\text{(mg/L)}}{\text{Concentration}} \times \underset{\text{(L/day)}}{\text{ingestion rate}} \times \underset{\text{(days/year)}}{\text{exposure frequency}} \times \underset{\text{(years)}}{\text{exposure duration}}}{\underset{\text{(kg)}}{\text{Body weight}} \times \underset{\text{(days/year)}}{365} \times \underset{\text{(years)}}{\text{exposure duration}}}$$

Potential carcinogenicity is thought to increase with *total* intake over time, instead of with *average daily* intake as for noncarcinogens. Also, body weight is relatively insignificant in determining carcinogenic risk from exposure to radionuclides. The only carcinogens elevated above background at the Grand

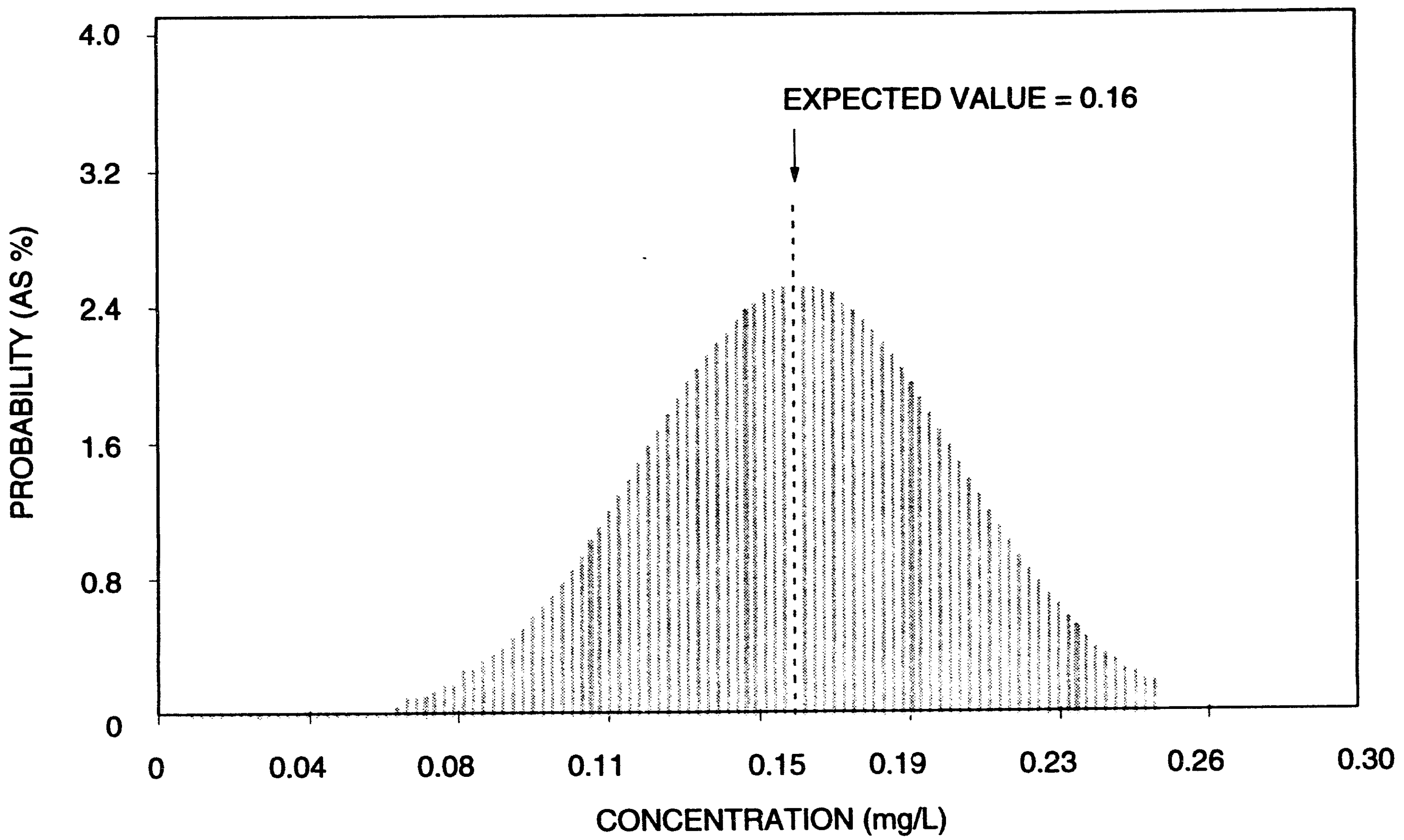

FIGURE 4.2
SIMULATED DISTRIBUTION OF ARSENIC CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

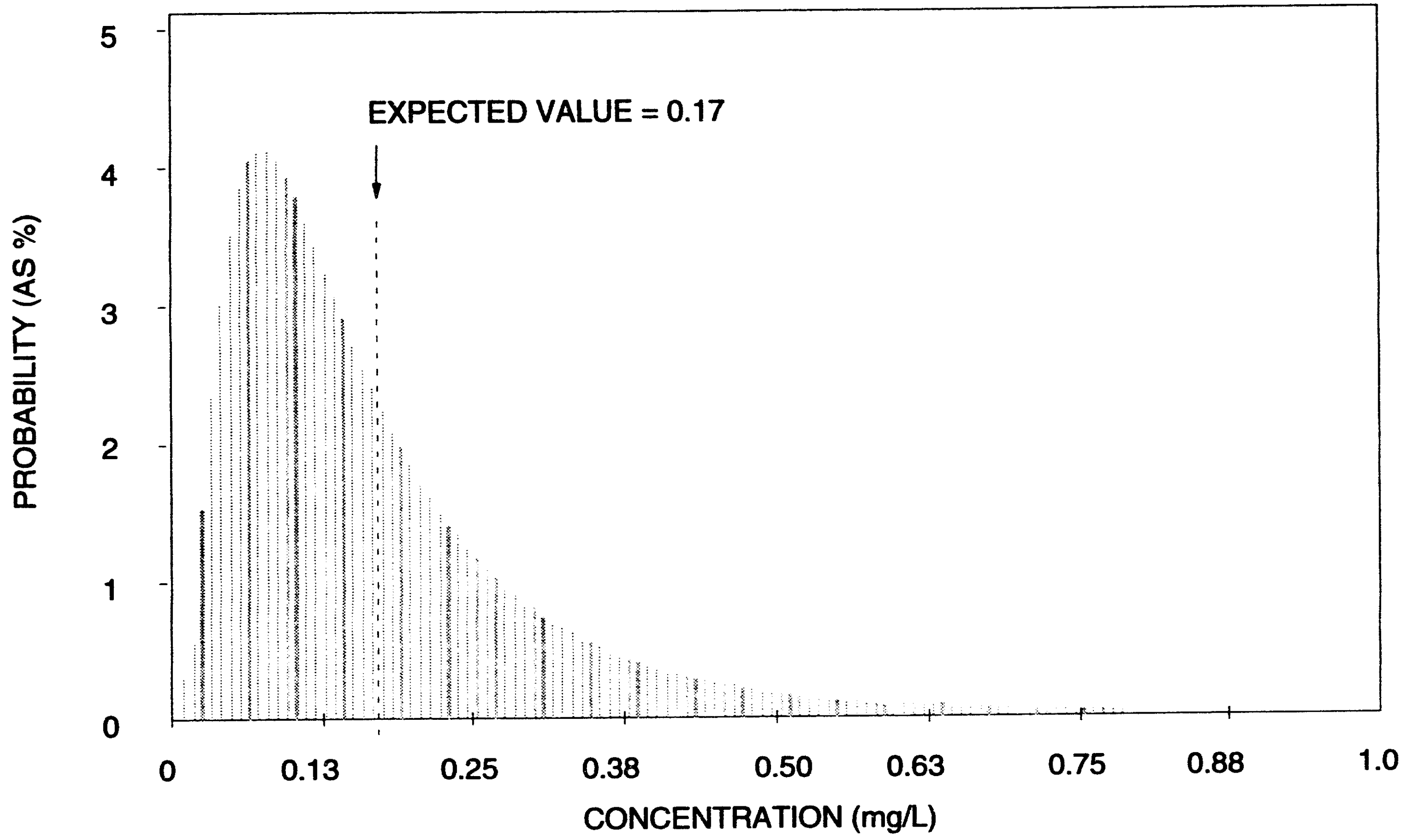

FIGURE 4.3
SIMULATED DISTRIBUTION OF CADMIUM CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

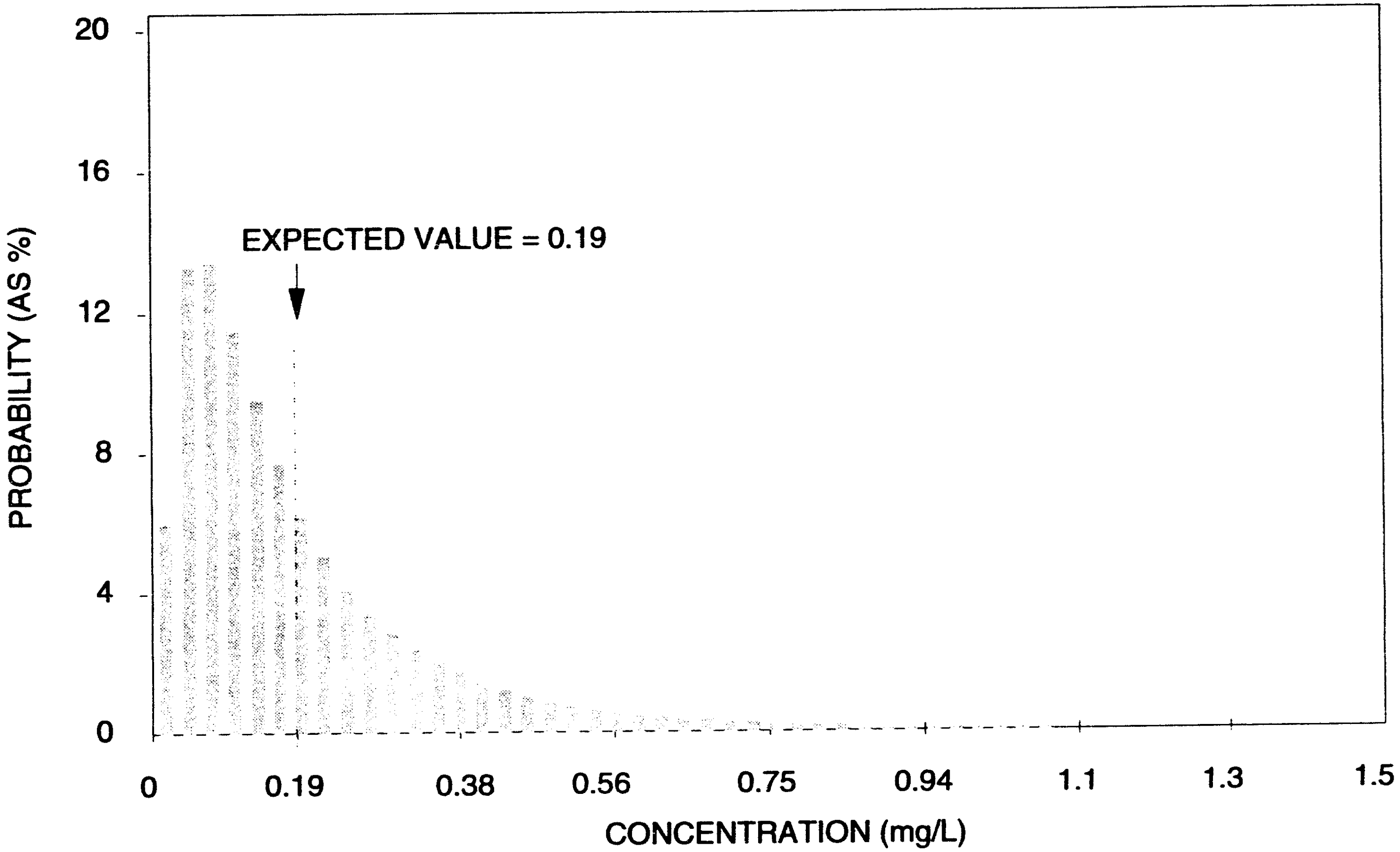

FIGURE 4.4
SIMULATED DISTRIBUTION OF COBALT CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

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COBCONC.DRW

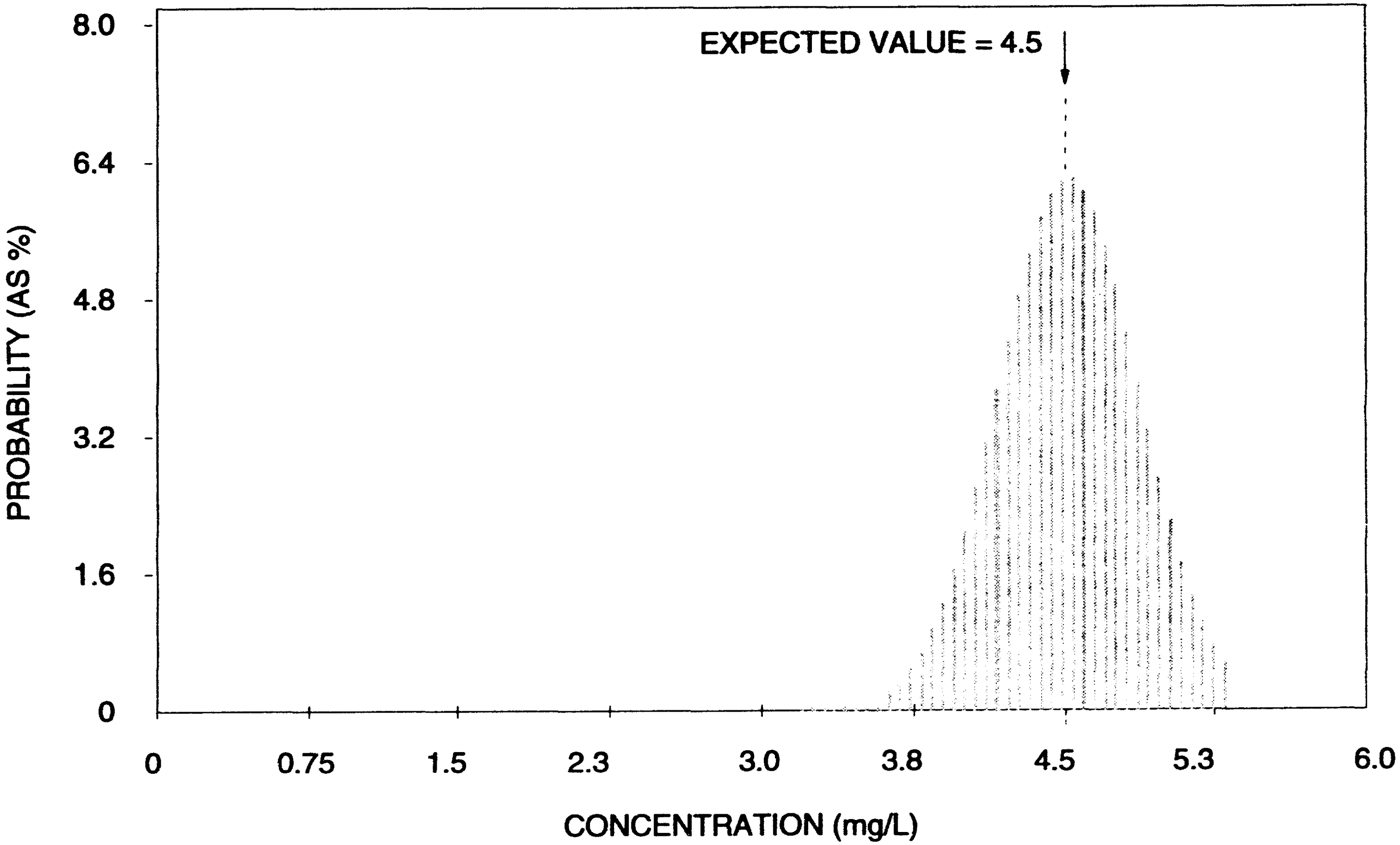

FIGURE 4.5
SIMULATED DISTRIBUTION OF FLUORIDE CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

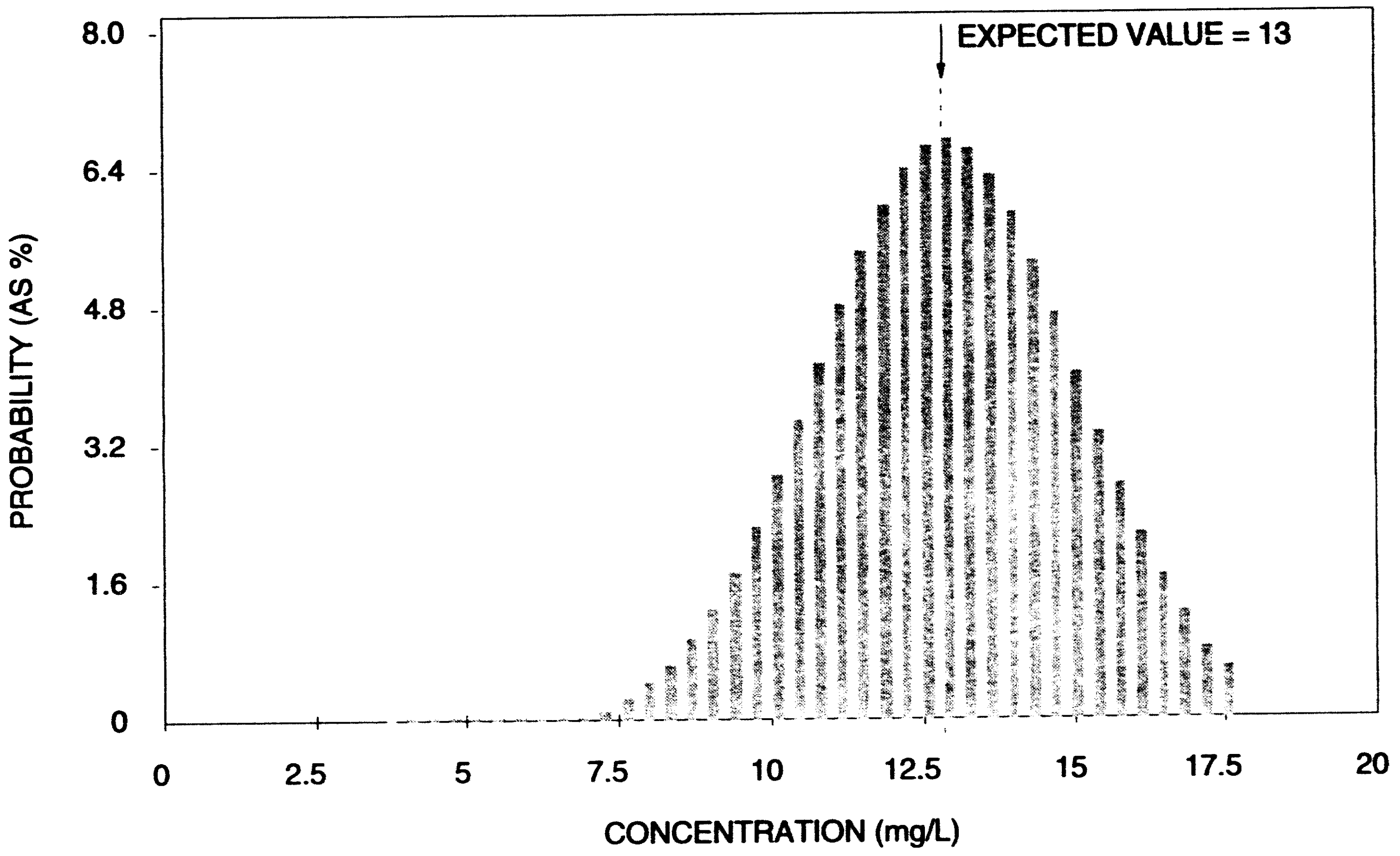

FIGURE 4.6
SIMULATED DISTRIBUTION OF IRON CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

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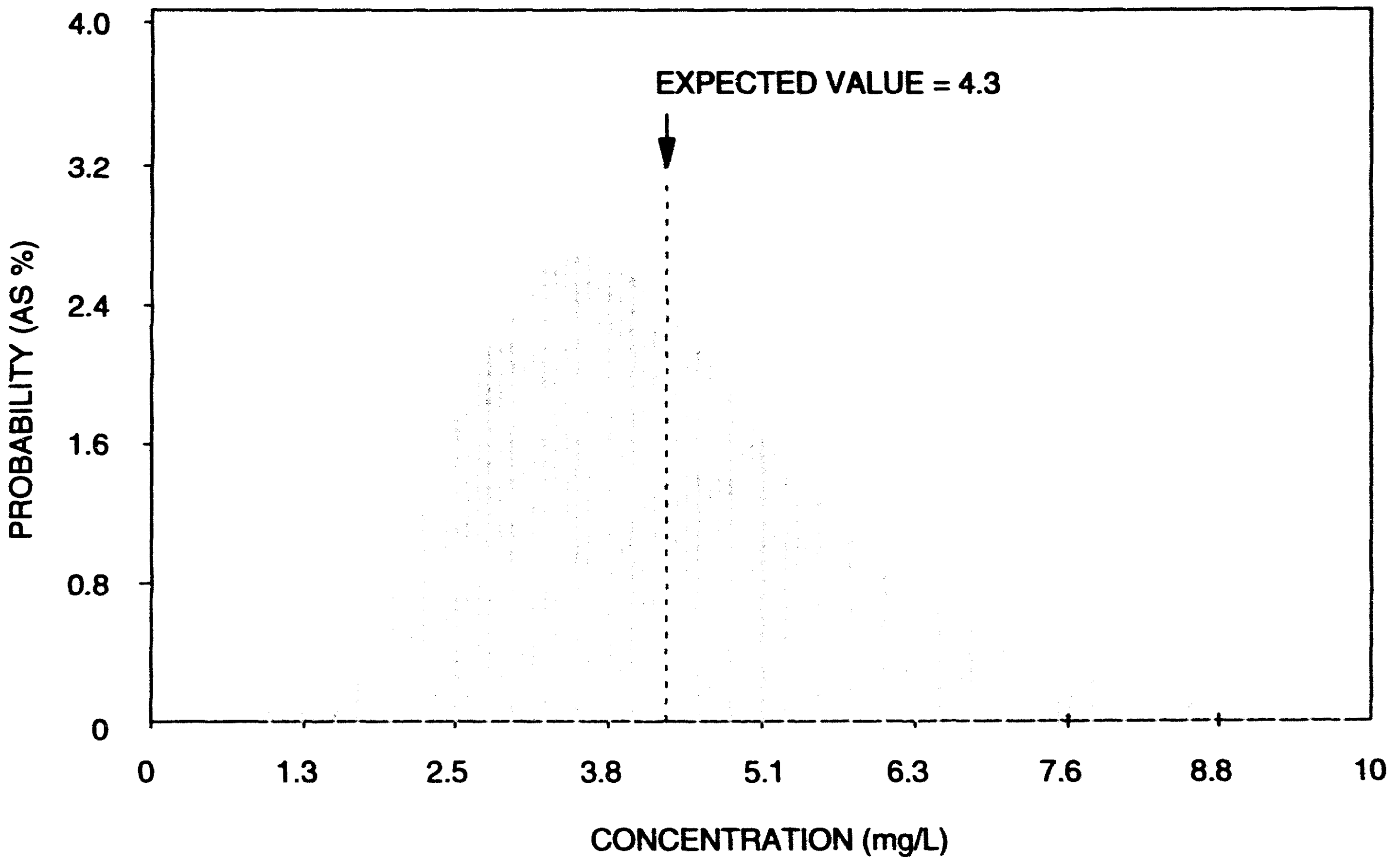


FIGURE 4.7
SIMULATED DISTRIBUTION OF MANGANESE CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

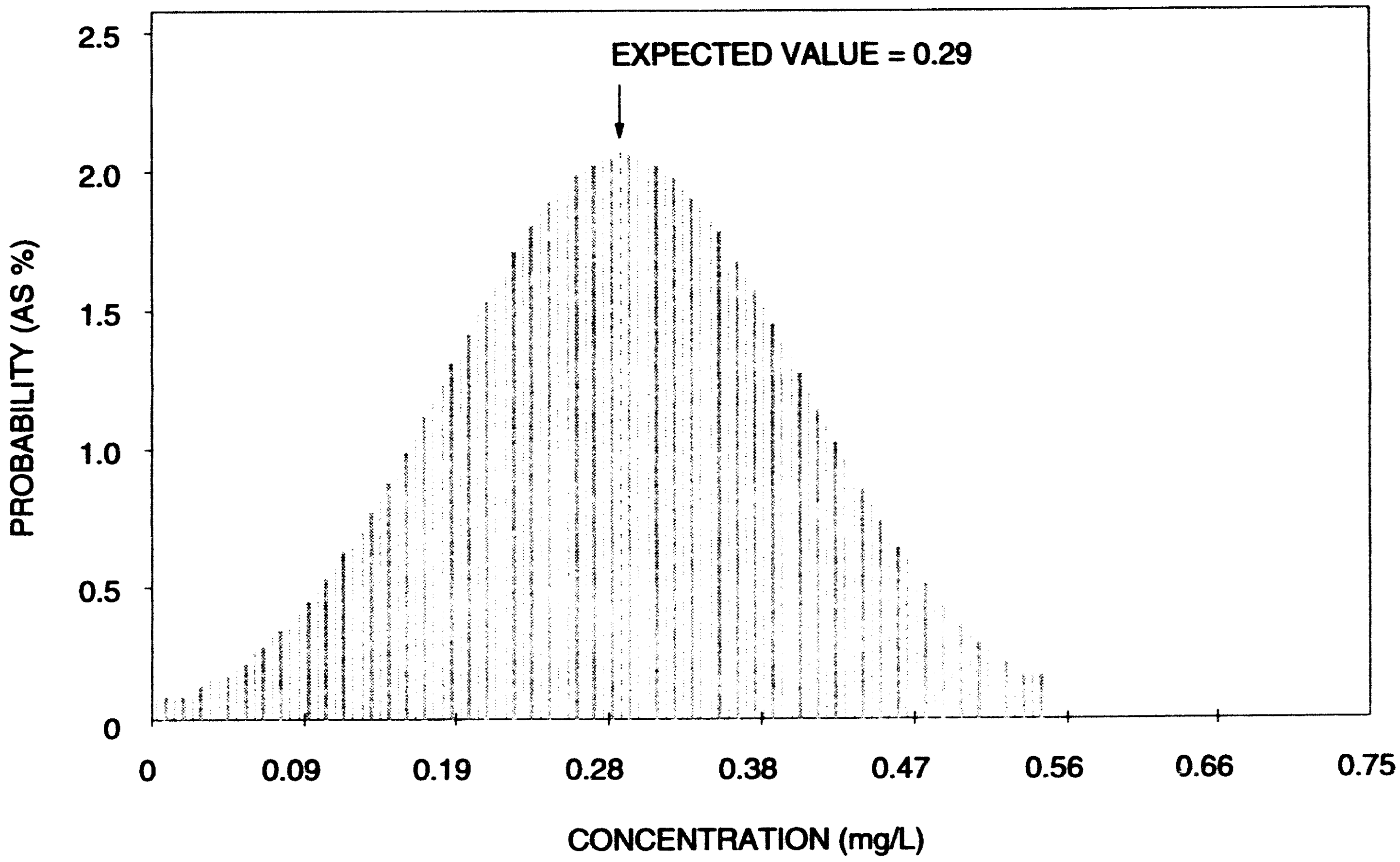


FIGURE 4.8
SIMULATED DISTRIBUTION OF MOLYBDENUM CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

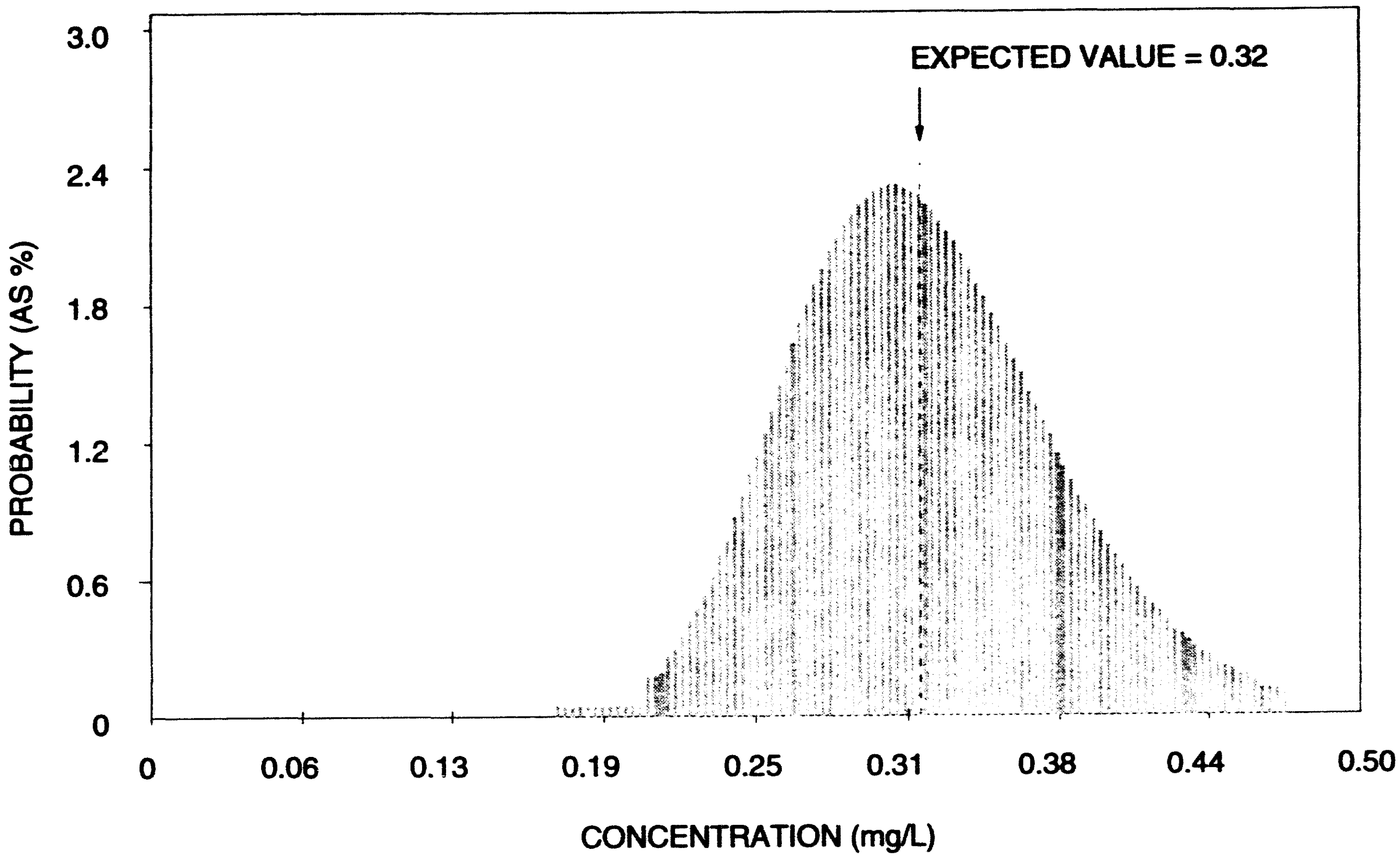


FIGURE 4.9
SIMULATED DISTRIBUTION OF NICKEL CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

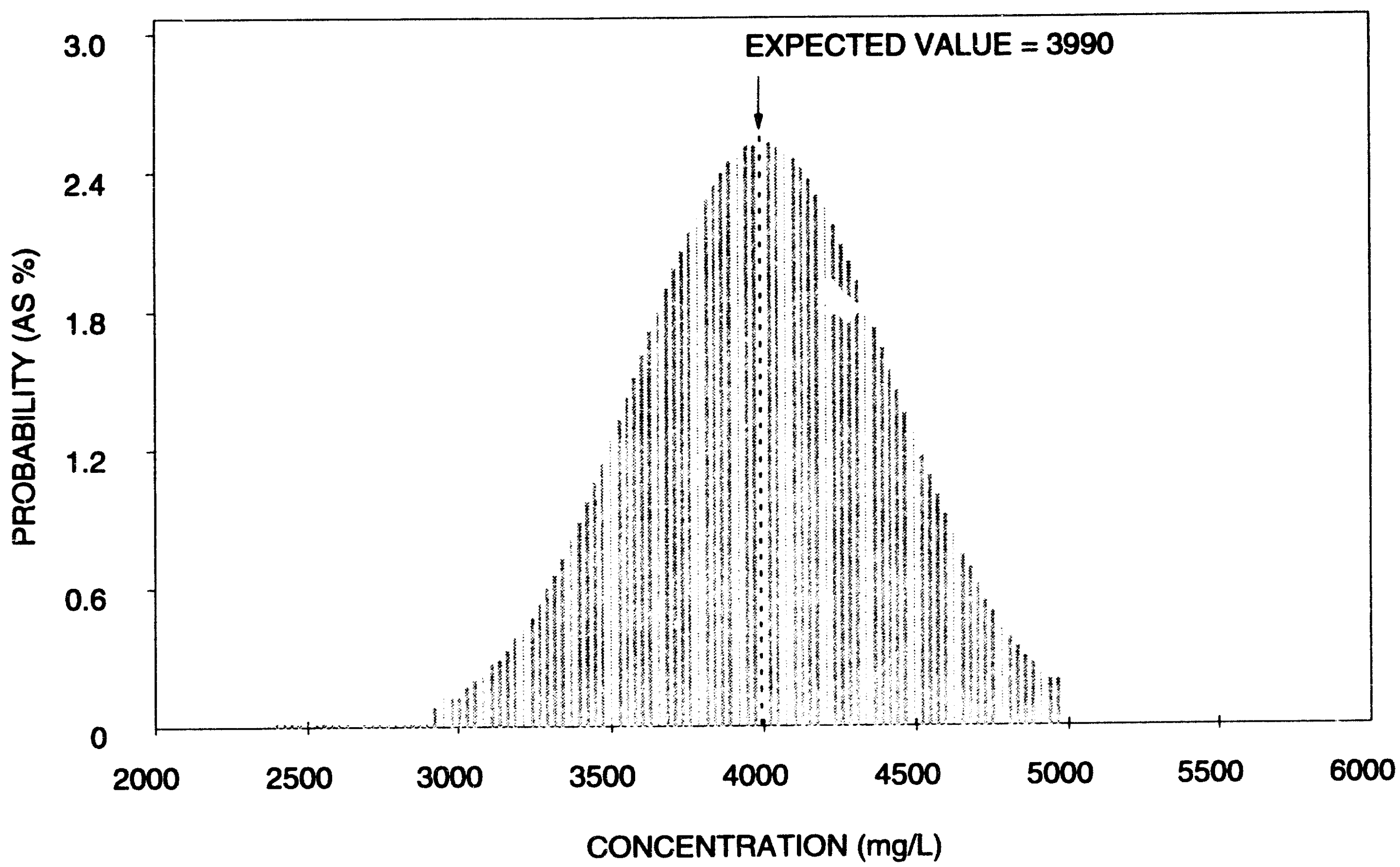


FIGURE 4.10
SIMULATED DISTRIBUTION OF SULFATE CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

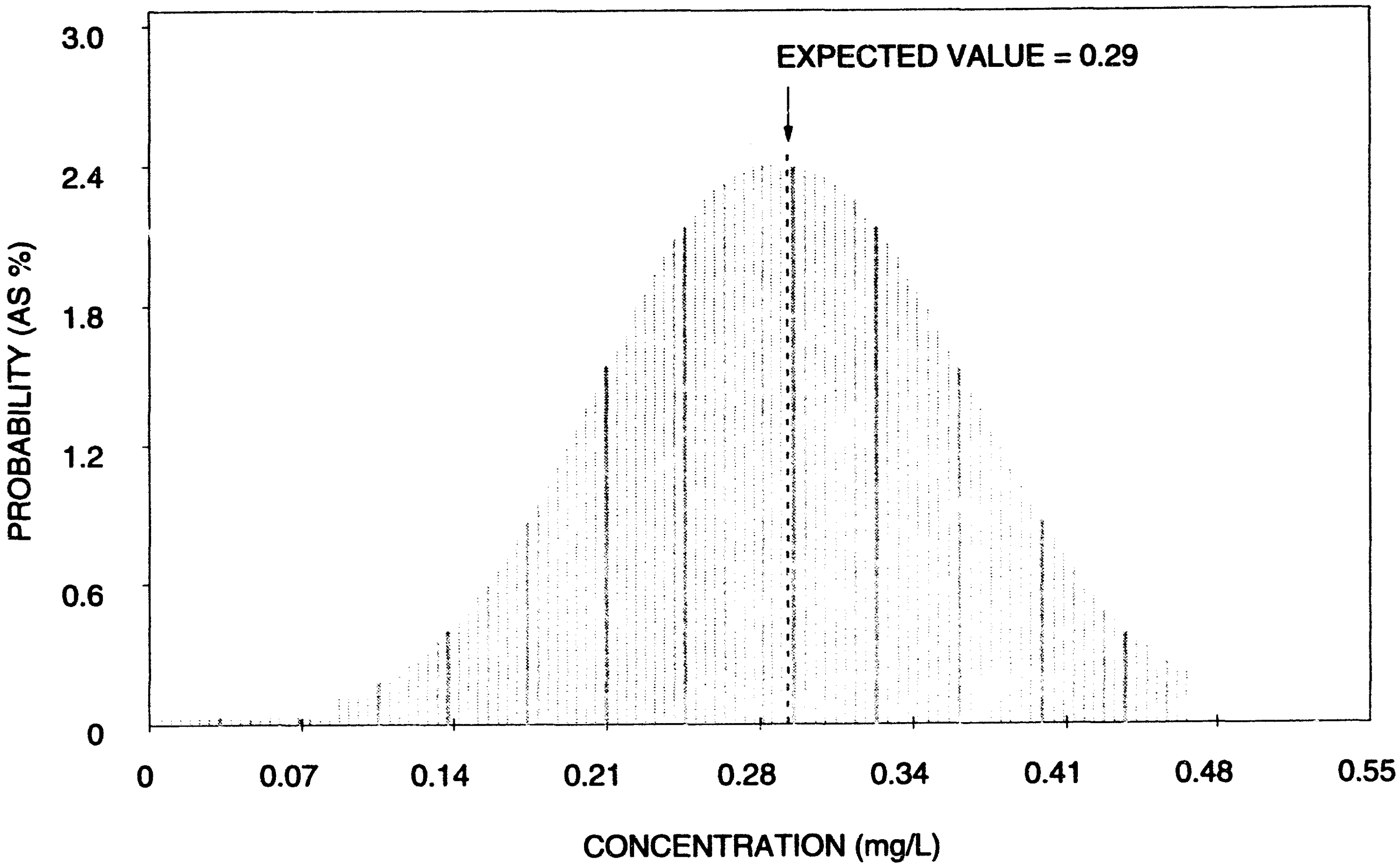


FIGURE 4.11
SIMULATED DISTRIBUTION OF URANIUM CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

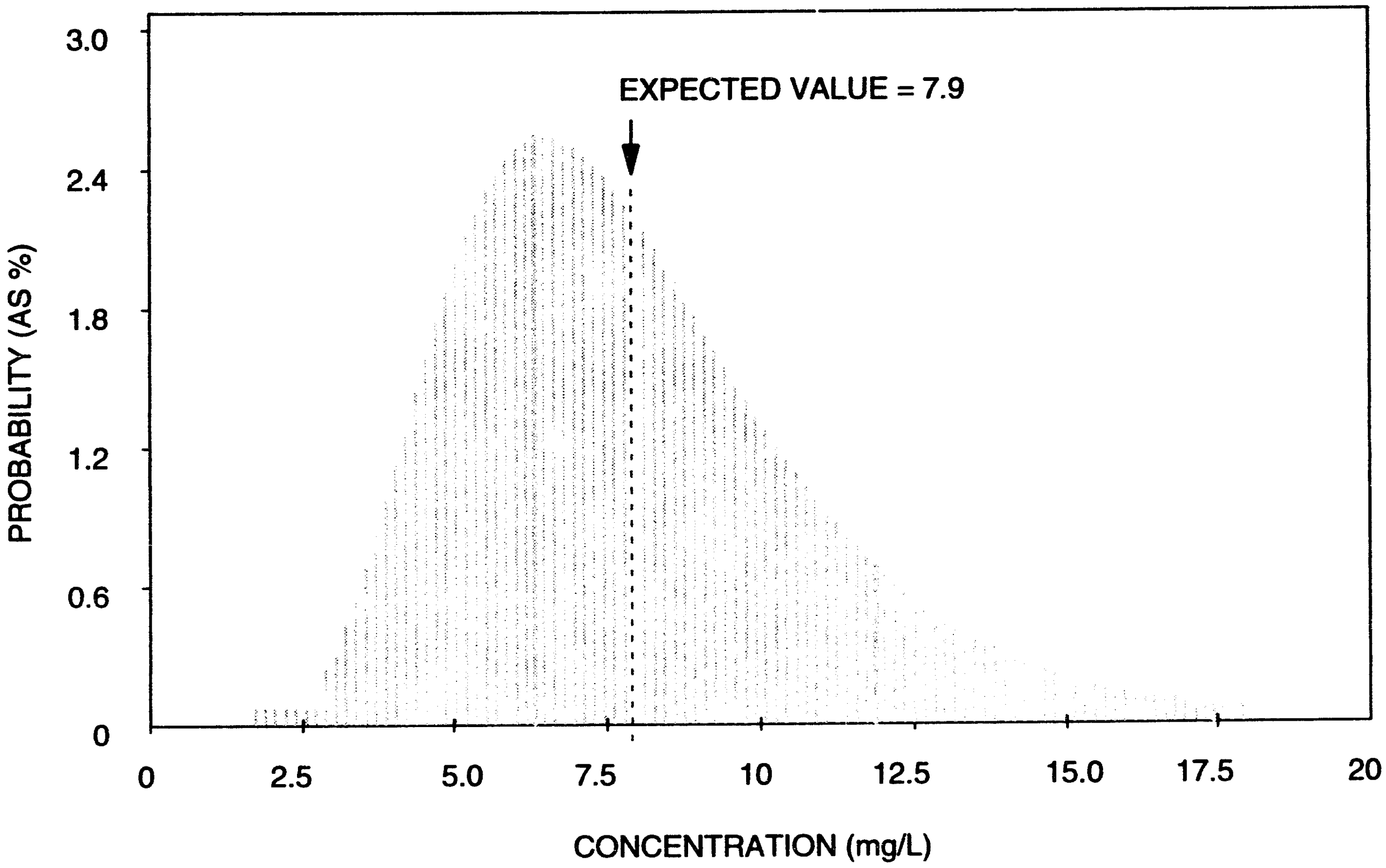


FIGURE 4.12
SIMULATED DISTRIBUTION OF VANADIUM CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

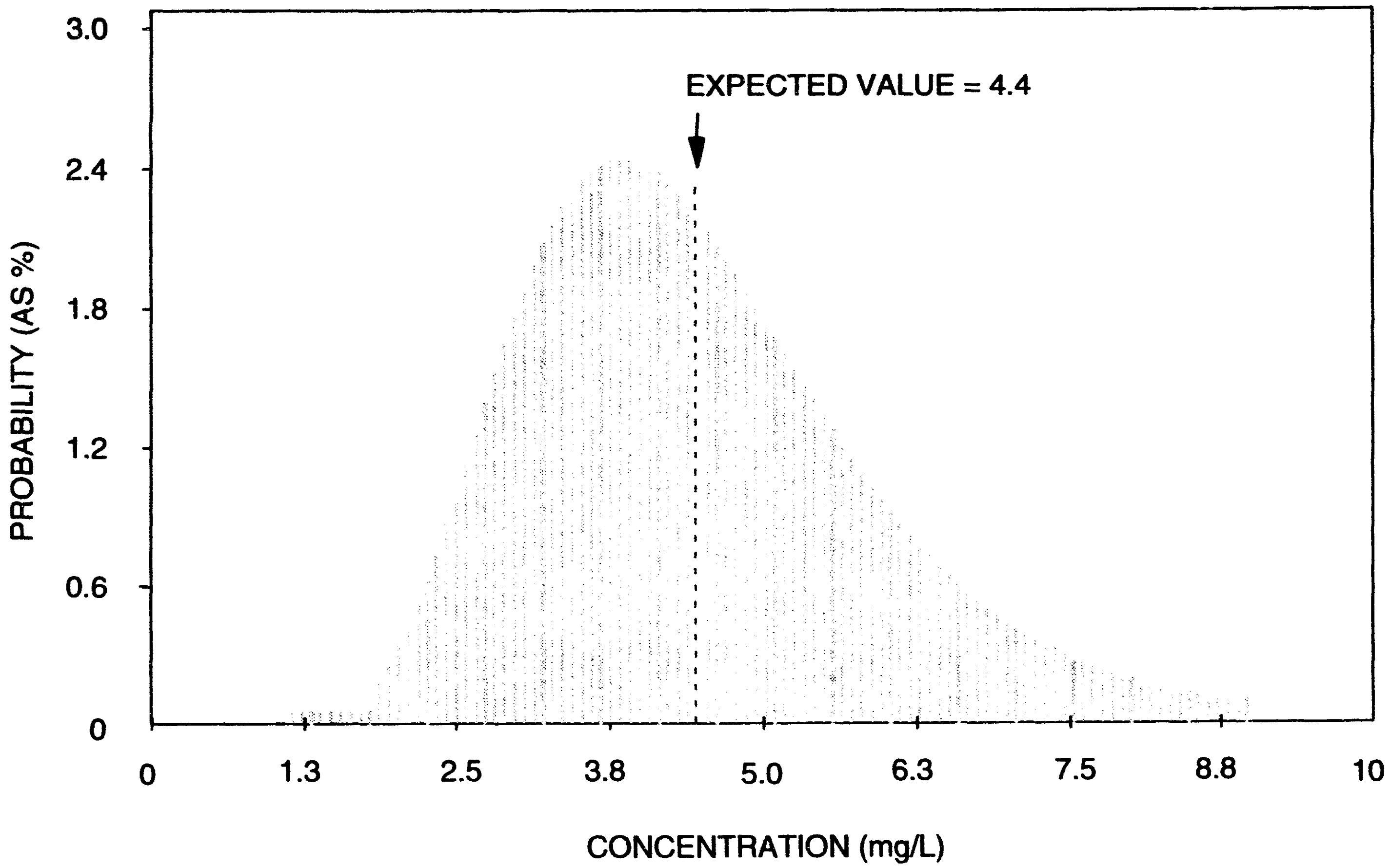


FIGURE 4.13
SIMULATED DISTRIBUTION OF ZINC CONCENTRATIONS
GRAND JUNCTION, COLORADO, SITE

Junction site are the radionuclides radium-226 and uranium and the metal arsenic. Intake of a radioactive carcinogen is therefore quantified as total exposure to radioactivity throughout the residency period of an individual:

$$\underset{(\text{pCi})}{\text{Intake}} = \underset{(\text{pCi/L})}{\text{Concentration}} \times \underset{(\text{L/day})}{\text{ingestion rate}} \times \underset{(\text{days/year})}{\text{exposure frequency}} \times \underset{(\text{years})}{\text{exposure duration}}$$

Intake of a chemical carcinogen, such as arsenic, is quantified using the following equation:

$$\underset{(\text{mg/kg-day})}{\text{Intake}} = \frac{\underset{(\text{mg/L})}{\text{Concentration}} \times \underset{(\text{L/day})}{\text{ingestion rate}} \times \underset{(\text{days/year})}{\text{exposure frequency}} \times \underset{(\text{years})}{\text{exposure duration}}}{\underset{(\text{kg})}{\text{Body weight}} \times \underset{(\text{days/year})}{365} \times \underset{(\text{years})}{\text{lifetime}}}$$

Average daily intake (L/day)

Lognormal probability distributions were used to describe variations in average daily tap water intake among members of the population (Roseberry and Burmaster, 1992). These distributions were developed from data collected during the 1977-78 National Food Consumption Survey conducted by the U.S. Department of Agriculture. During the survey, total tap water consumption during a 3-day period was recorded for 26,081 survey participants nationwide (Figure 4.14).

Body weight (kg)

Extensive national data on weights of males and females, by age, were collected by the National Health and Nutrition Survey between 1976 and 1980. These data were used to develop lognormal probability distributions for body weight by age, separately by sex. The distributions for males and females were then combined using census data on the national ratio of males to females within each age group (Figure 4.15).

Exposure duration (years)

Distributions of total residence time (or exposure duration) were developed by Israeli and Nelson (1992) using data collected by the U.S. Department of Commerce, the Bureau of the Census, and the U.S. Department of Housing and Urban Development in 1985 and 1987. However, a fixed exposure duration of 30 years was assumed reasonable for the population in the Grand Junction risk assessment, because it is consistent with EPA exposure duration parameters for urban residents (EPA, 1991). Because the concentrations of arsenic and the radionuclides in ground water at the Grand Junction site are decreasing with time, evaluations of risk based on this exposure duration combined with historic and current median and maximum concentrations of arsenic and the

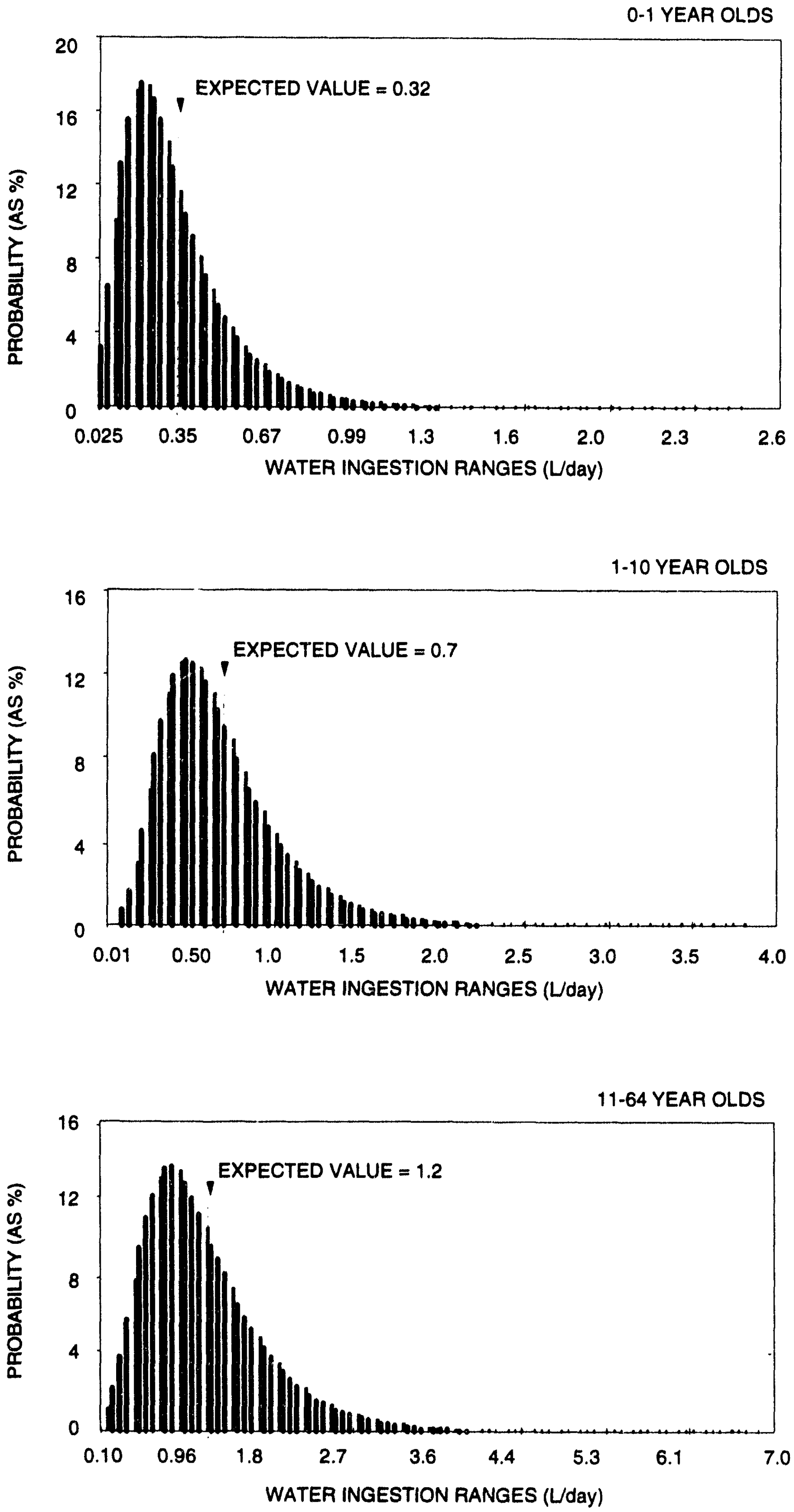


FIGURE 4.14
PROBABILITY DISTRIBUTIONS FOR TAP WATER INGESTION RATES

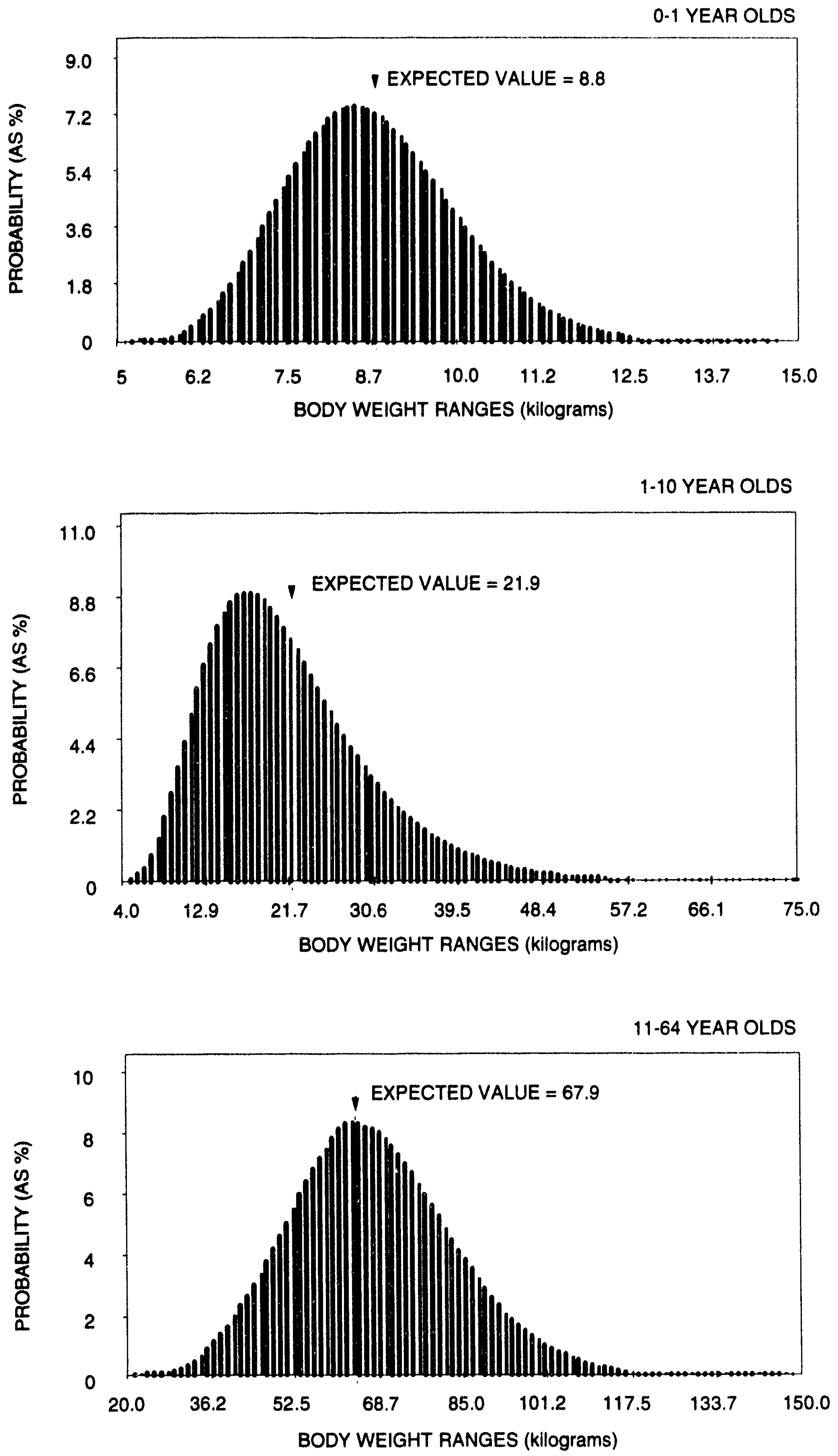


FIGURE 4.15
PROBABILITY DISTRIBUTIONS FOR BODY WEIGHT

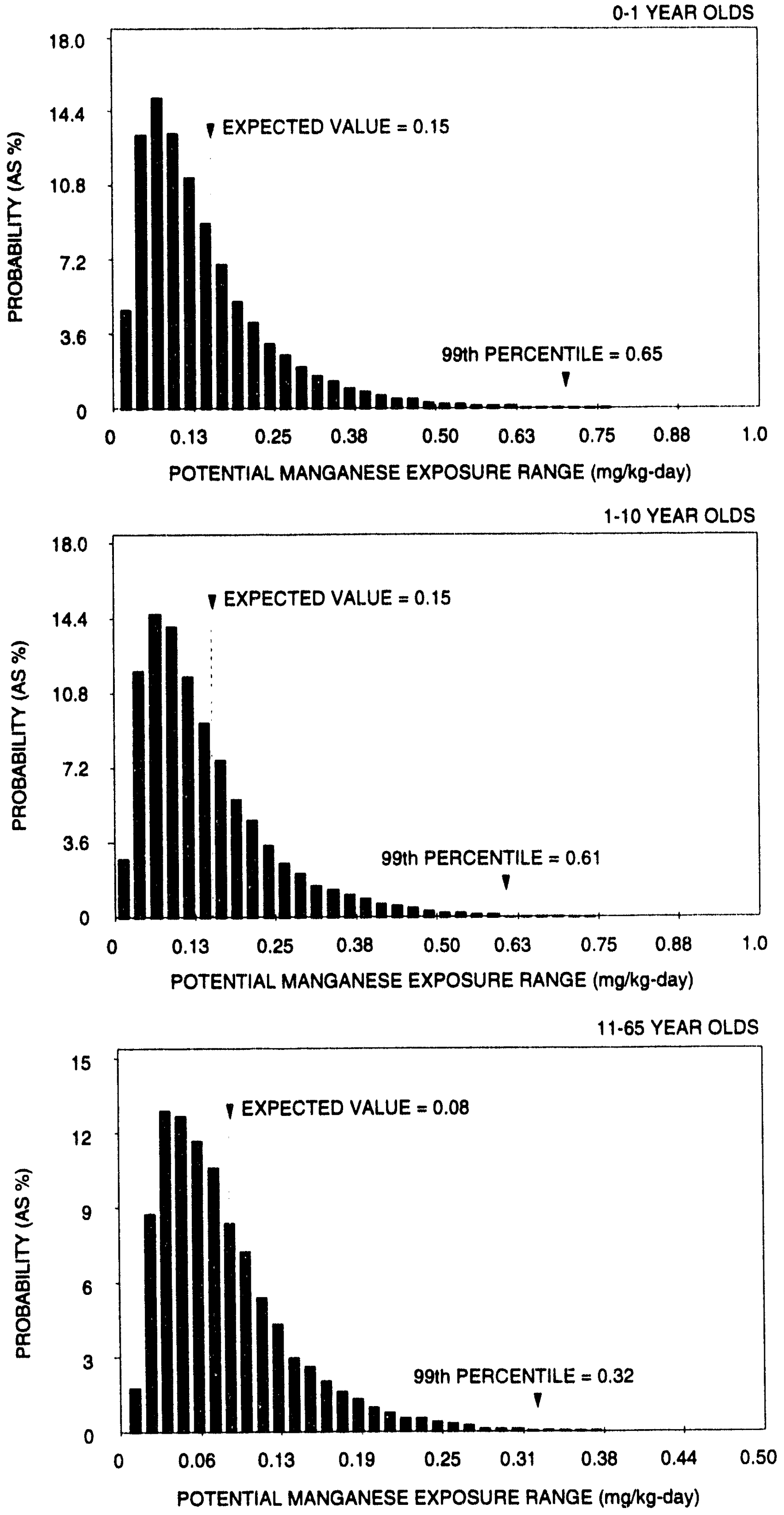
radionuclides should yield conservative estimates of carcinogenic risks for this site.

Using exposure concentration distributions discussed in Section 4.3 and the intake parameter distributions described in this section, total intake distributions derived for the three age groups were generated for manganese. These results are presented in Figure 4.16 to illustrate the effect of the different age group characterizations on daily intake of noncarcinogens. This figure shows that intake is greatest in the 1- to 10-year age group, although the intake for the 0- to 1-year age group is very similar. The 1- to 10-year old age group consistently showed the highest intake-to-body-weight distributions and therefore is the most conservative age group to evaluate. However, since infants are the most susceptible receptors to sulfate toxicity, the intake distribution for this age group is used for sulfate. The simulated intake distributions for 1- to 10-year old children and for infants (sulfate only) for the contaminants at this site are presented in Figures 4.17 through 4.28.

4.5 EXPOSURE ASSESSMENT UNCERTAINTIES

A number of potential sources of error may arise in all phases of the exposure assessment, including the following more significant sources of uncertainty:

- Uncertainties resulting from the lack of thorough environmental sampling data (ground water, surface water, sediment, and biota), which could lead to an underestimate or overestimate in the exposure analysis.
- Uncertainties arising from the assumption that the ground water contaminant source term at the site has reached a steady state and that contaminant concentrations at the exposure point will remain constant for chronic periods of exposure (generally greater than 7 years). Because the source of contamination at Grand Junction has been removed, the assumption of a constant source will probably lead to an overestimation of risk.
- Uncertainties associated with the model used to estimate uptake of contaminants into plants for the irrigated garden produce pathway. Site-specific plant uptake factors could vary substantially from the default literature estimates. As with environmental sampling, the net effect on risk estimates of this uncertainty cannot be predicted.
- Uncertainties with fish BCFs. Site-specific BCFs could vary substantially from the default literature values.
- Uncertainties associated with the relationship of an applied dose (used in this assessment) and absorbed dose or effective toxic dose.



NOTE: HIGHEST POTENTIAL EXPOSURE OCCURS IN CHILDREN AGE 1-10 YEARS (AVERAGE POTENTIAL EXPOSURE OF 0.15 mg/kg-day).

FIGURE 4.16

COMPARISON OF DISTRIBUTIONS OF POTENTIAL MANGANESE INTAKES AS A RESULT OF DRINKING CONTAMINATED GROUND WATER FOR THE THREE AGE GROUPS EXAMINED GRAND JUNCTION, COLORADO, SITE

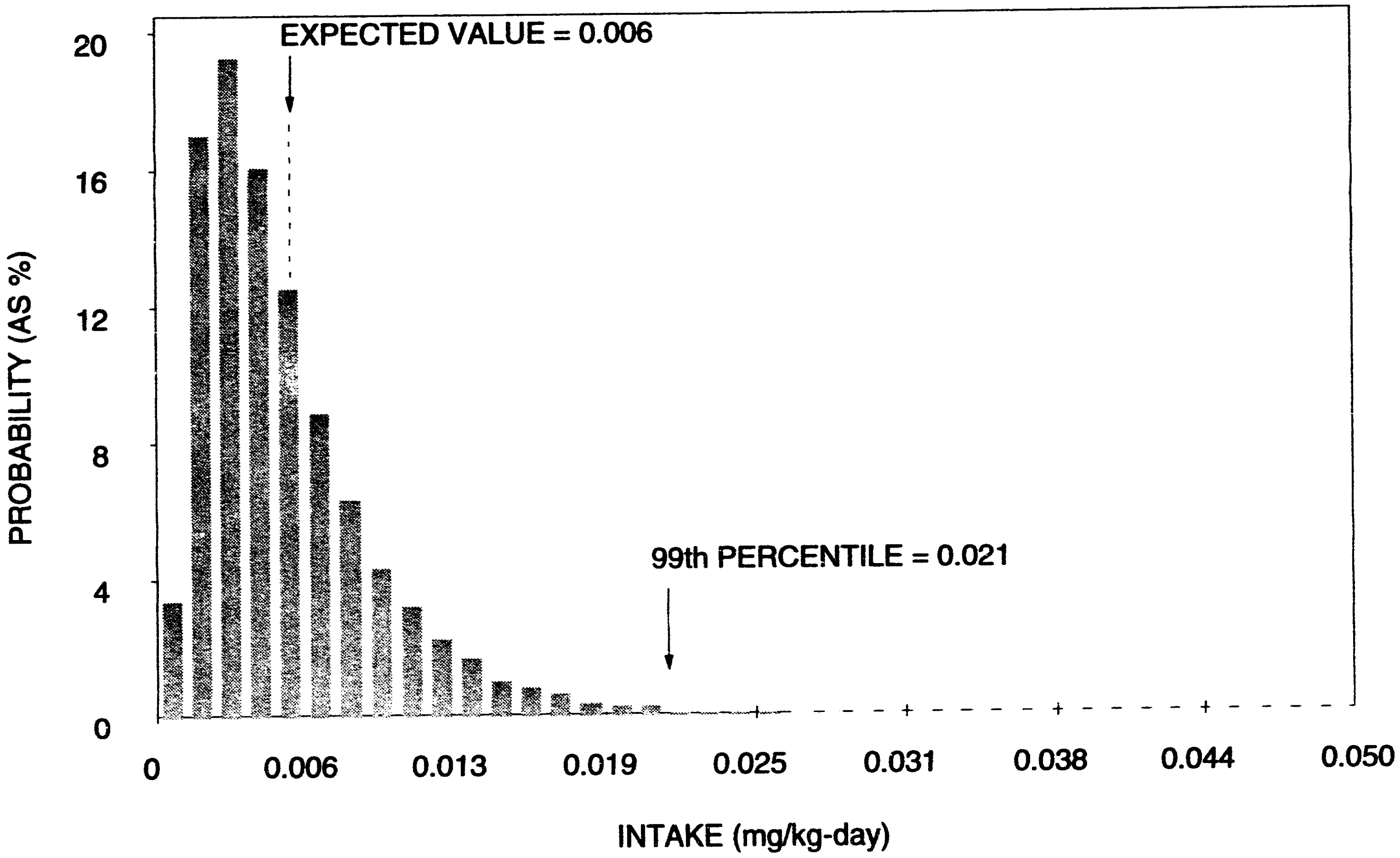


FIGURE 4.17
DISTRIBUTION OF POTENTIAL ARSENIC INTAKES FOR CHILDREN AS A RESULT OF GROUND WATER INGESTION GRAND JUNCTION, COLORADO, SITE

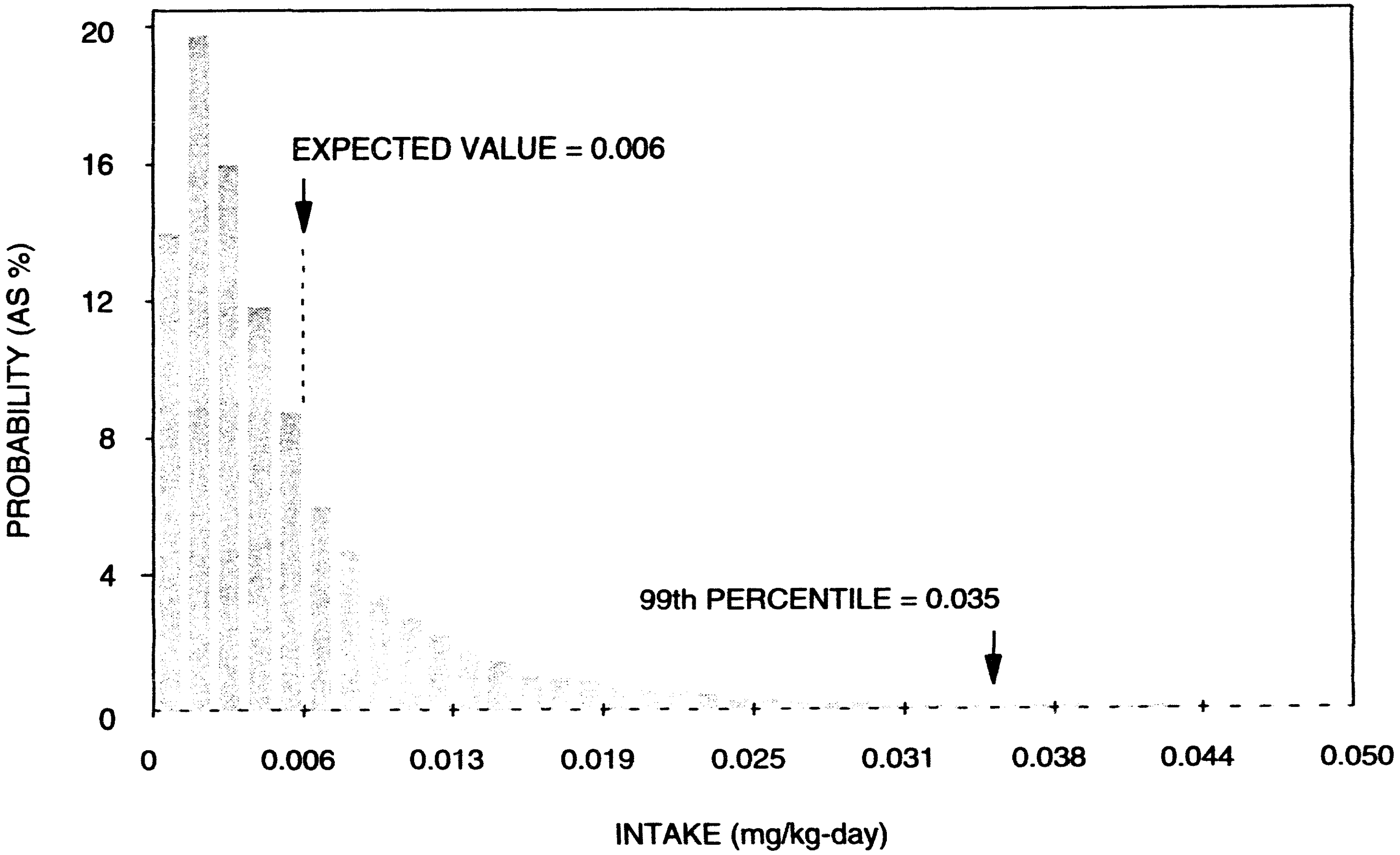


FIGURE 4.18
DISTRIBUTION OF POTENTIAL CADMIUM INTAKES FOR CHILDREN
AS A RESULT OF GROUND WATER INGESTION
GRAND JUNCTION, COLORADO, SITE

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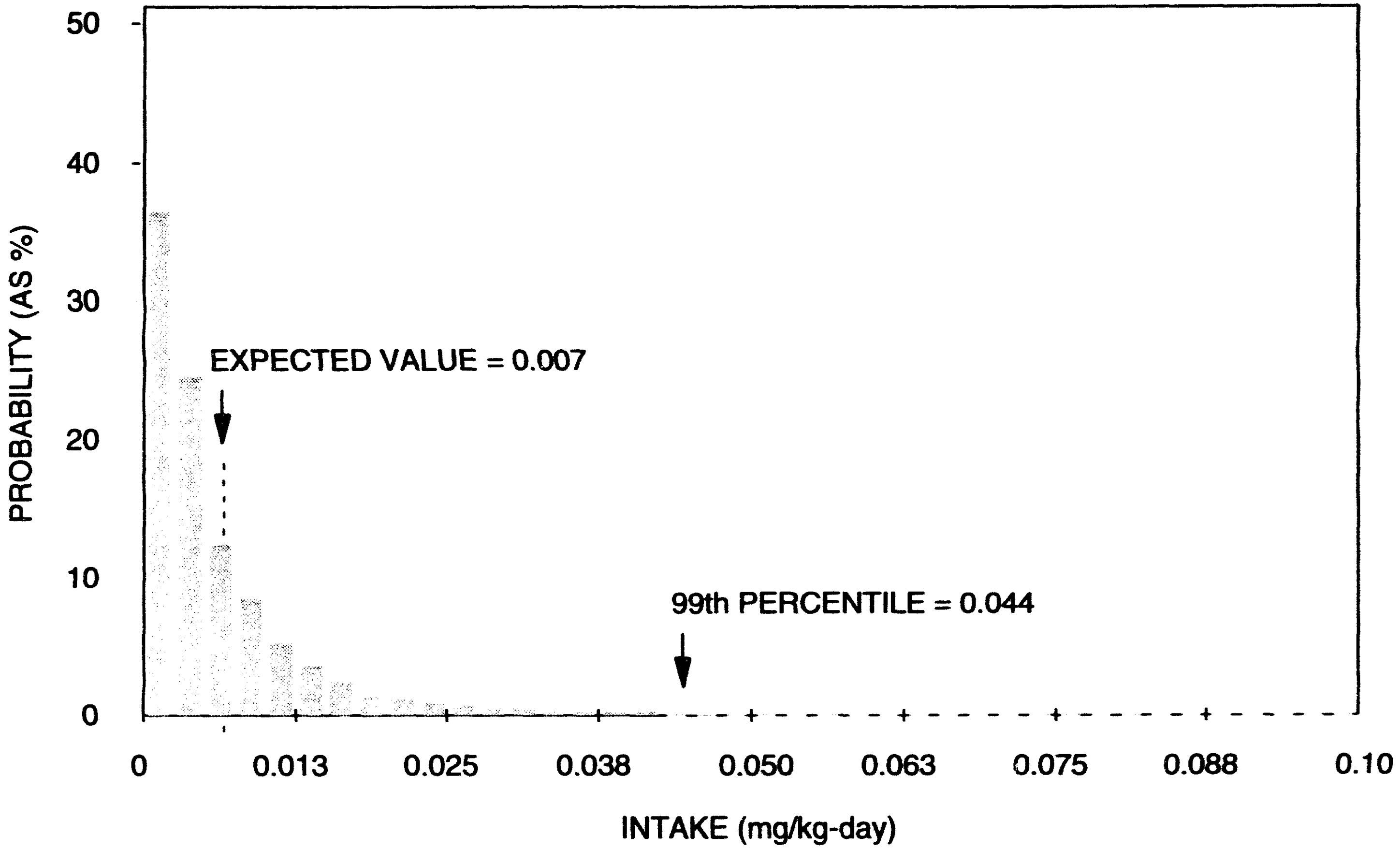

FIGURE 4.19
DISTRIBUTION OF POTENTIAL COBALT INTAKES FOR CHILDREN
AS A RESULT OF GROUND WATER INGESTION
GRAND JUNCTION, COLORADO, SITE

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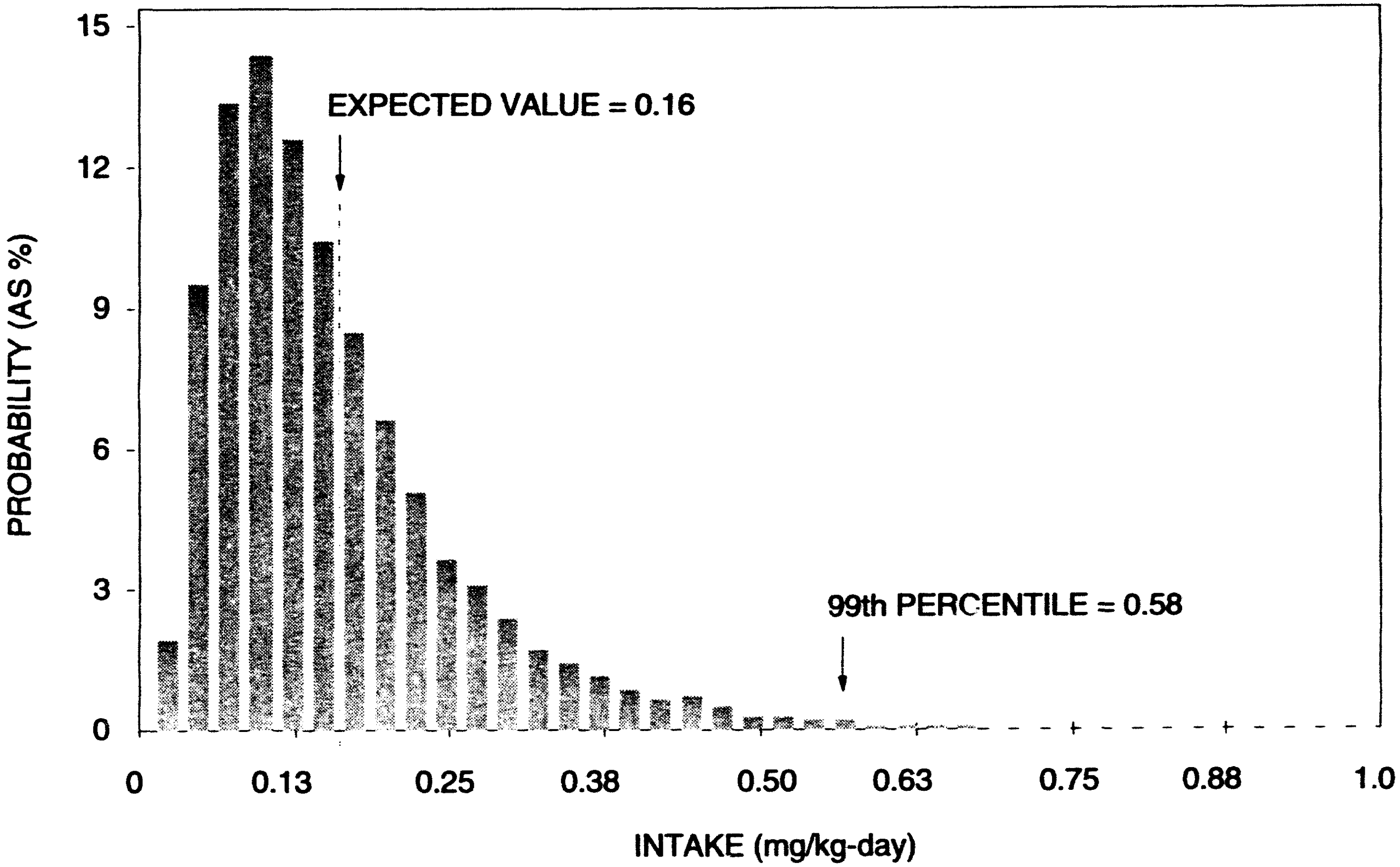

FIGURE 4.20
DISTRIBUTION OF POTENTIAL FLUORIDE INTAKES FOR CHILDREN
AS A RESULT OF GROUND WATER INGESTION
GRAND JUNCTION, COLORADO, SITE

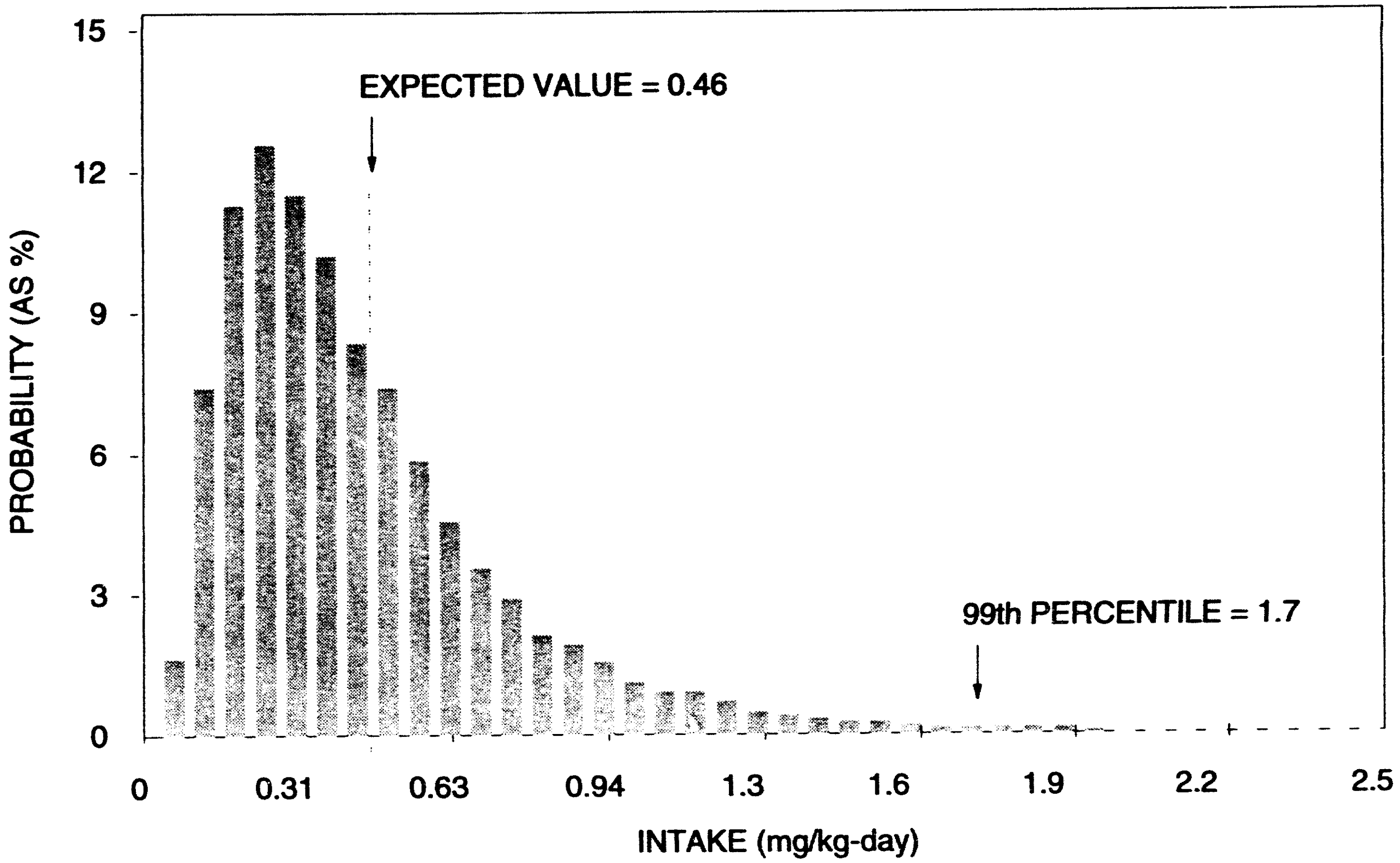

FIGURE 4.21
DISTRIBUTION OF POTENTIAL IRON INTAKES FOR CHILDREN
AS A RESULT OF GROUND WATER INGESTION
GRAND JUNCTION, COLORADO, SITE

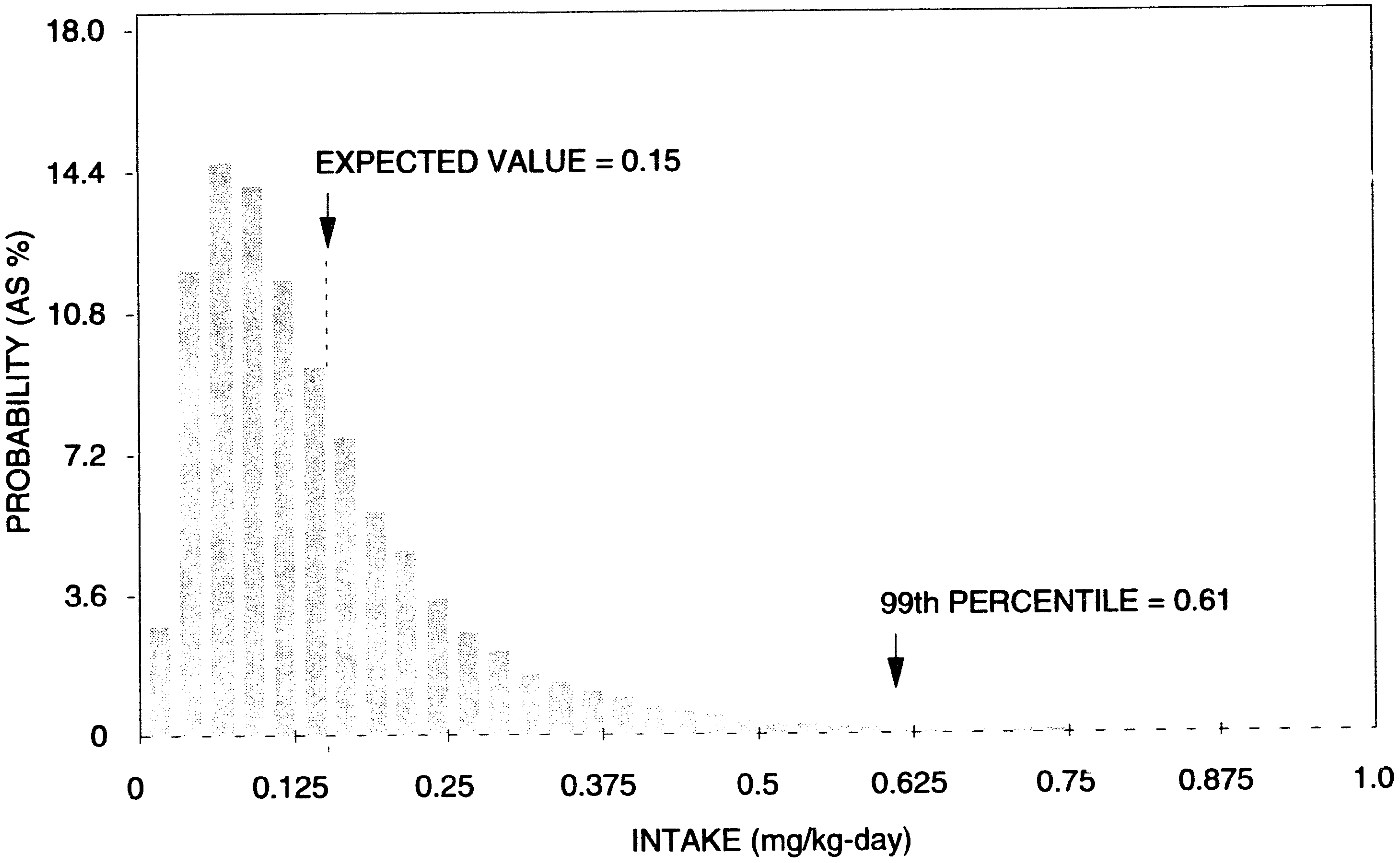

FIGURE 4.22
DISTRIBUTION OF POTENTIAL MANGANESE INTAKES FOR CHILDREN
AS A RESULT OF GROUND WATER INGESTION
GRAND JUNCTION, COLORADO, SITE

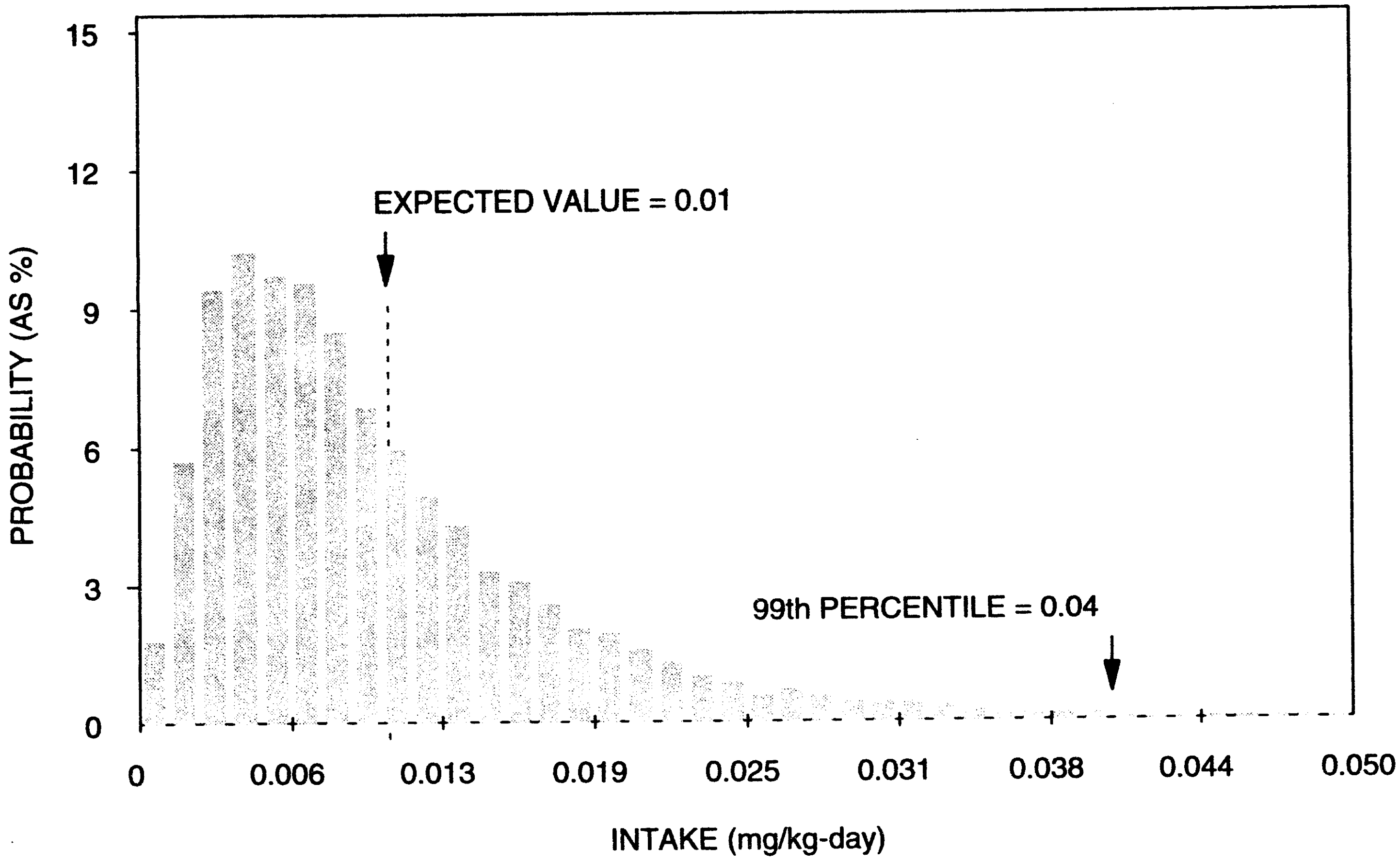

FIGURE 4.23
DISTRIBUTION OF POTENTIAL MOLYBDENUM INTAKES FOR CHILDREN
AS A RESULT OF GROUND WATER INGESTION
GRAND JUNCTION, COLORADO, SITE

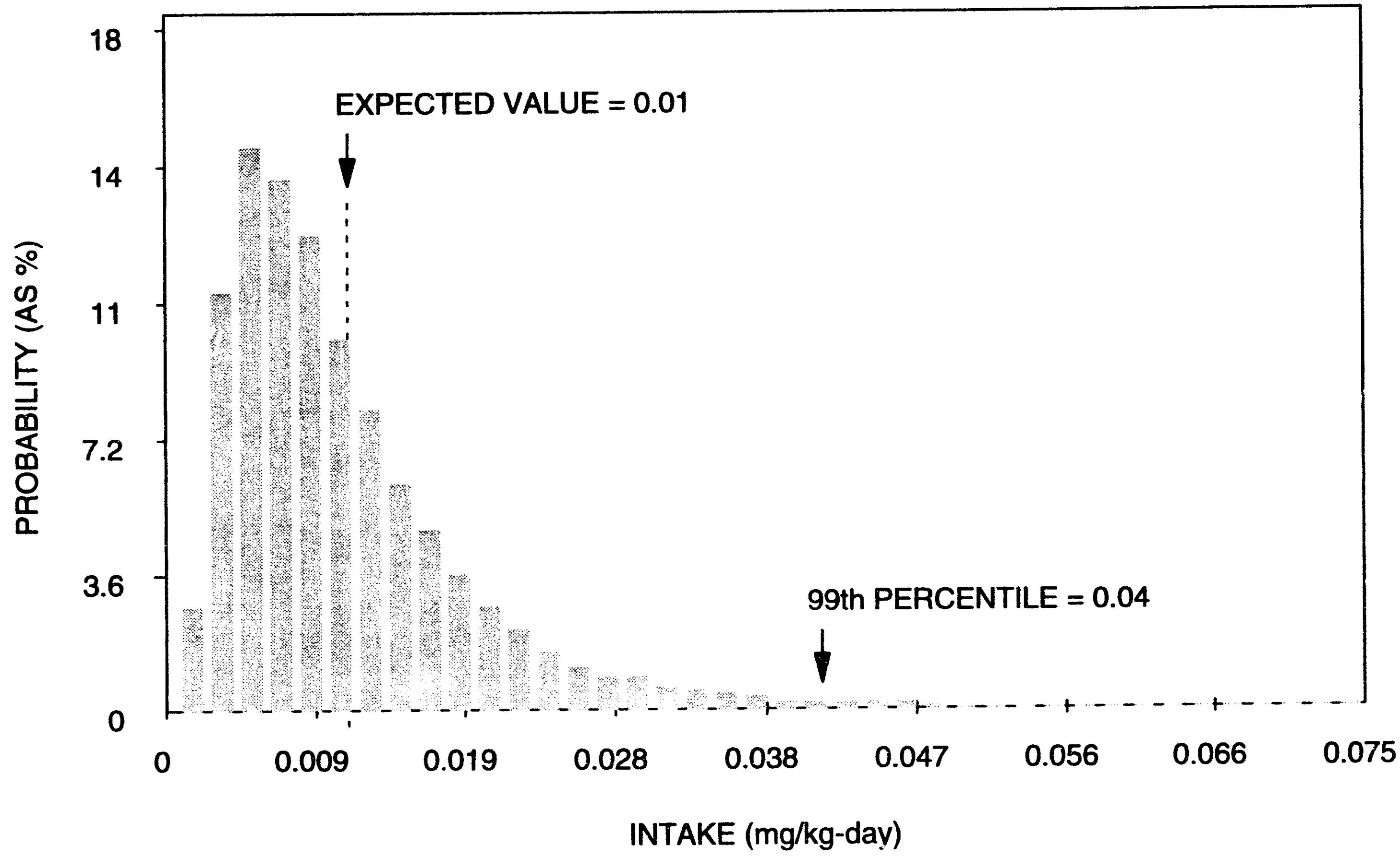

FIGURE 4.24
DISTRIBUTION OF POTENTIAL NICKEL INTAKES FOR CHILDREN AS A RESULT OF GROUND WATER INGESTION GRAND JUNCTION, COLORADO, SITE

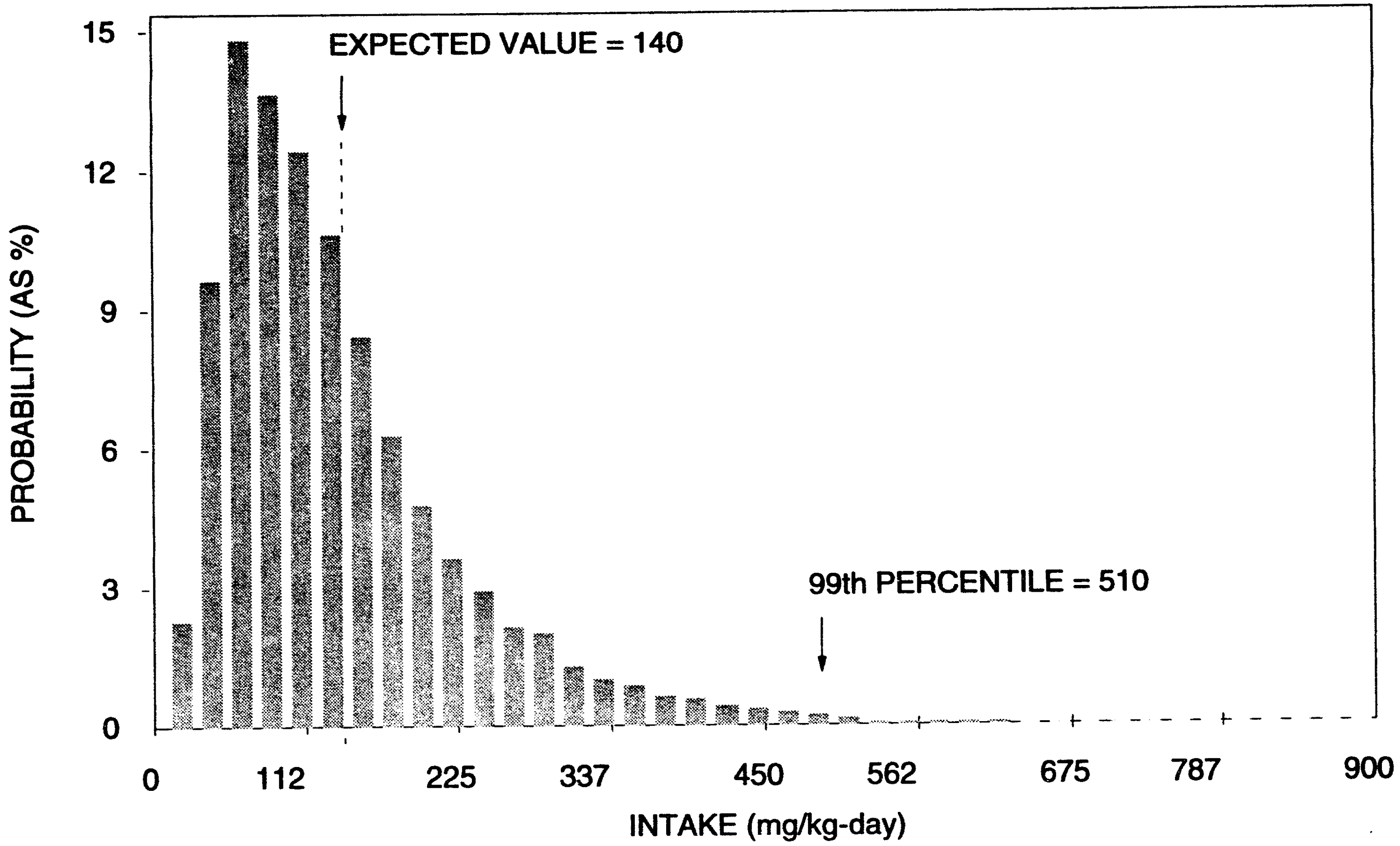

FIGURE 4.25
DISTRIBUTION OF POTENTIAL SULFATE INTAKES FOR CHILDREN
AS A RESULT OF GROUND WATER INGESTION
GRAND JUNCTION, COLORADO, SITE

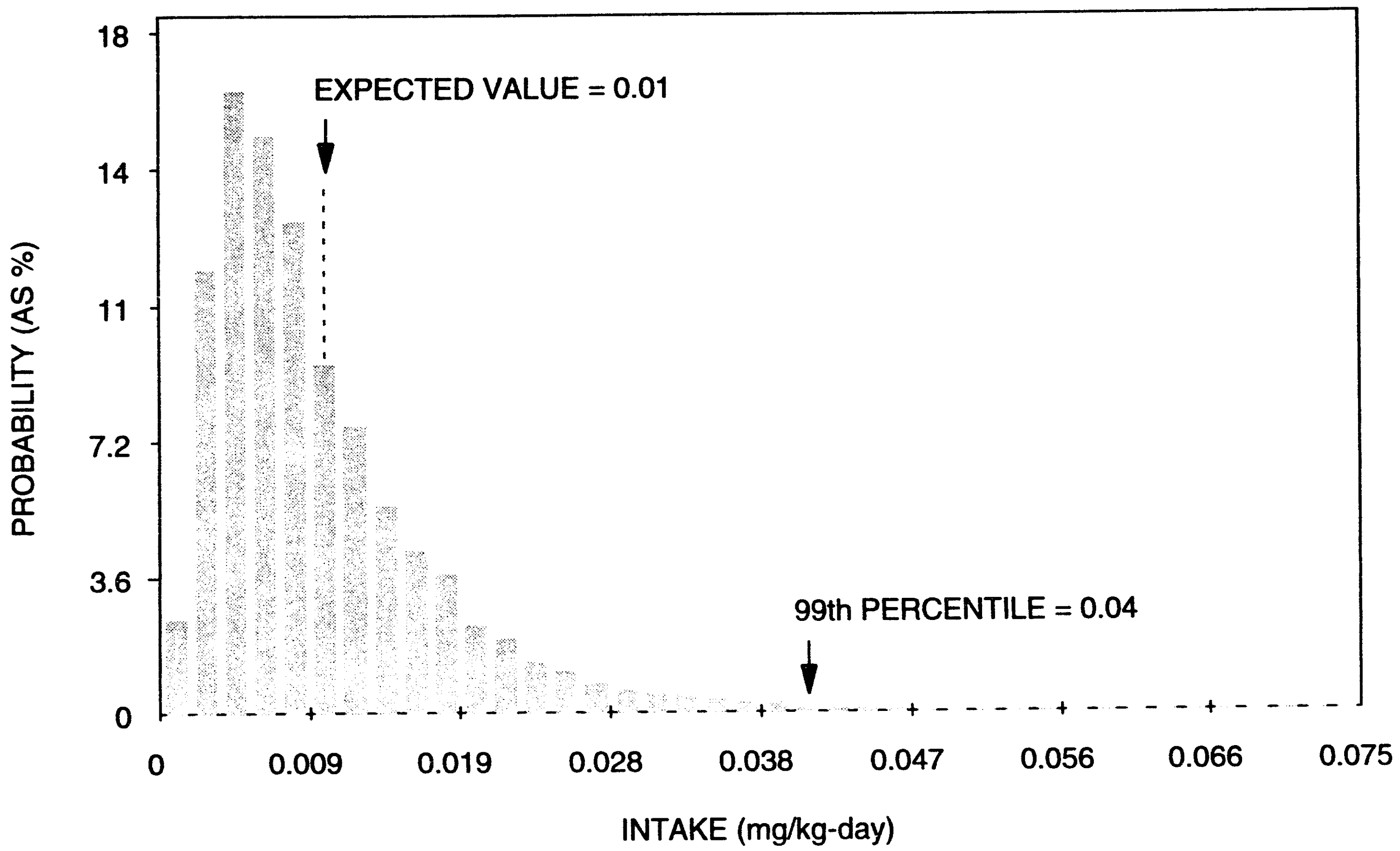

FIGURE 4.26
DISTRIBUTION OF POTENTIAL URANIUM INTAKES FOR CHILDREN
AS A RESULT OF GROUND WATER INGESTION
GRAND JUNCTION, COLORADO, SITE

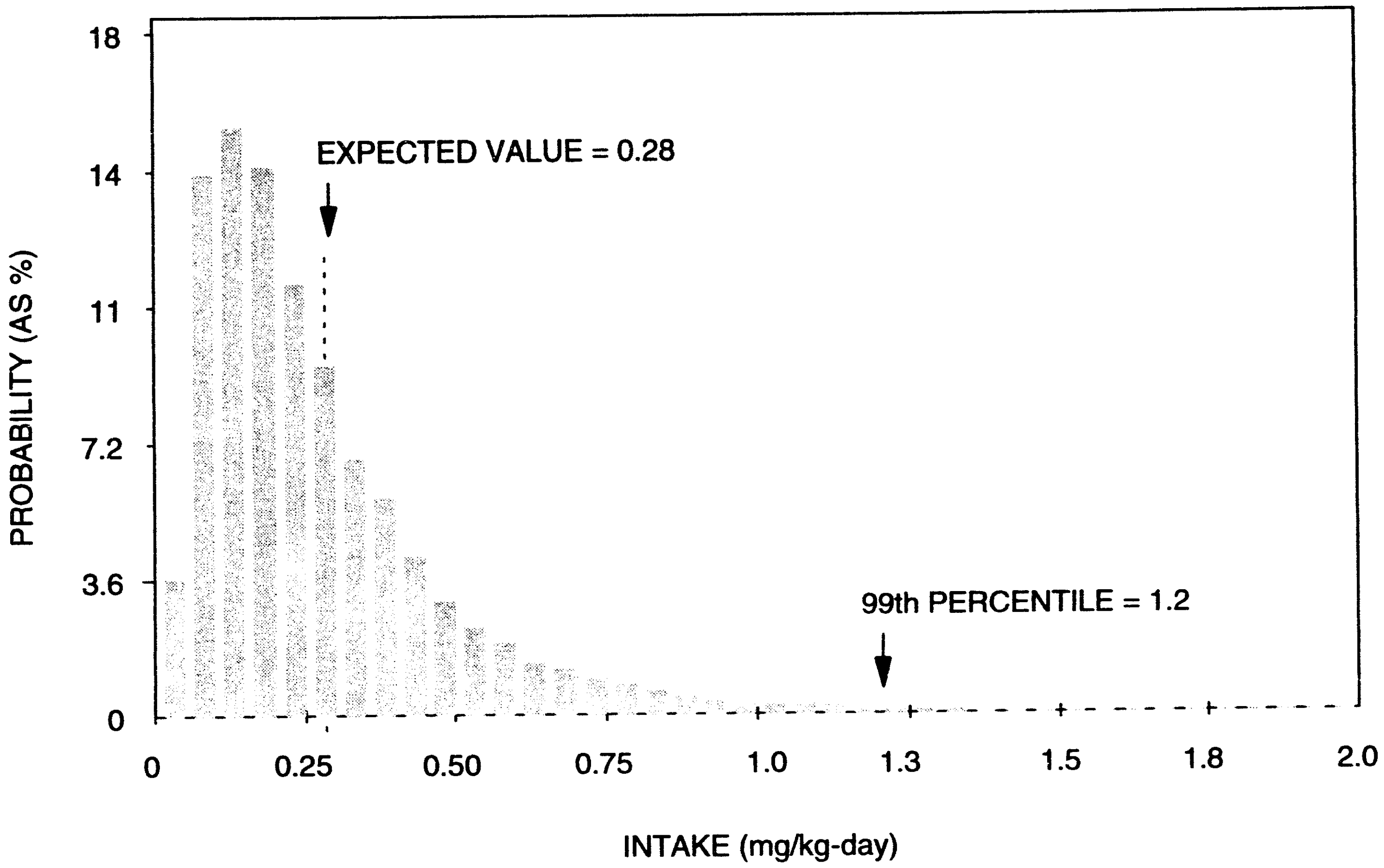

FIGURE 4.27
DISTRIBUTION OF POTENTIAL VANADIUM INTAKES FOR CHILDREN
AS A RESULT OF GROUND WATER INGESTION
GRAND JUNCTION, COLORADO, SITE

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VA1_10.DRW

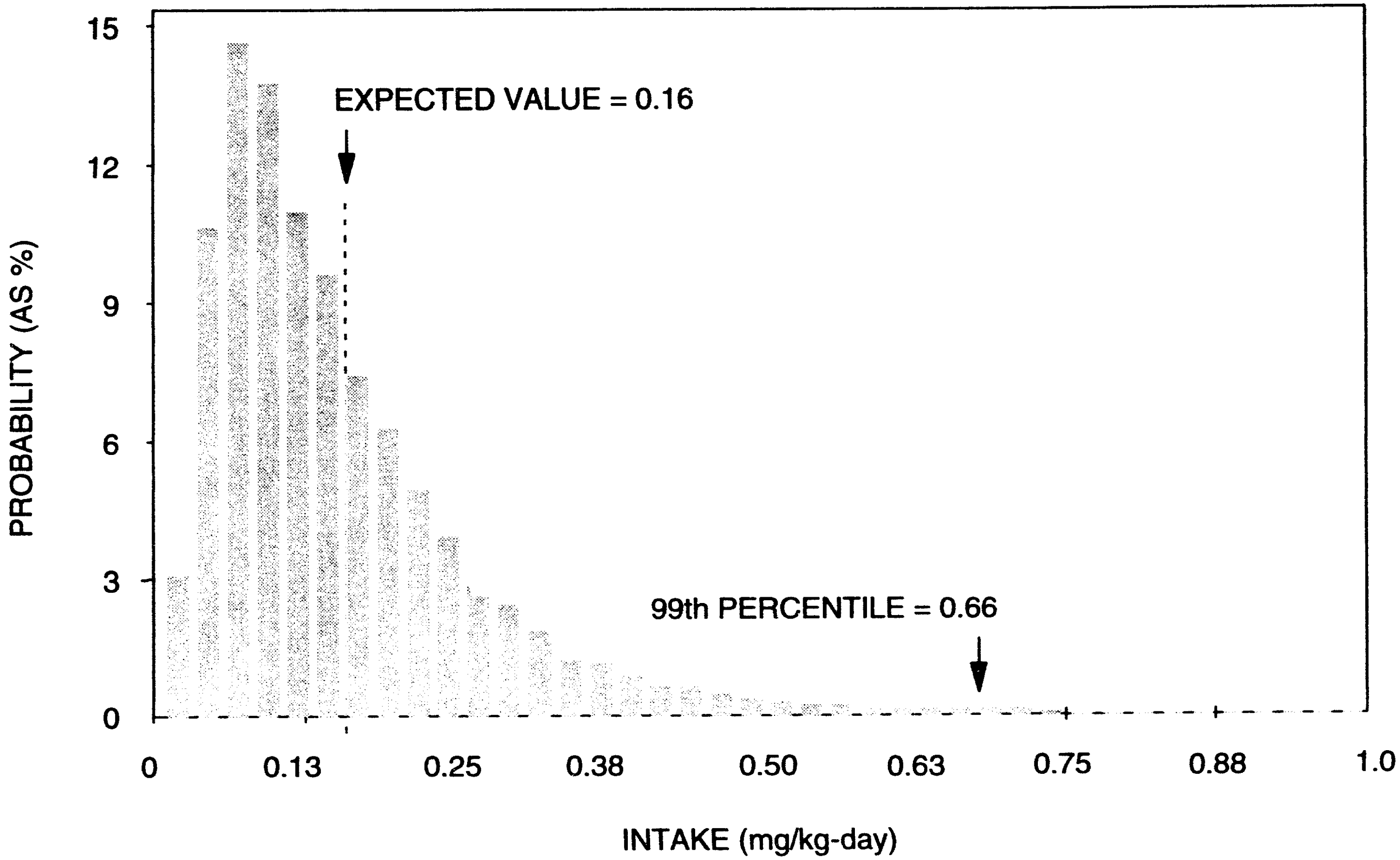


FIGURE 4.28
DISTRIBUTION OF POTENTIAL ZINC INTAKES FOR CHILDREN
AS A RESULT OF GROUND WATER INGESTION
GRAND JUNCTION, COLORADO, SITE

- Uncertainties associated with differing sensitivities of subpopulations, such as individuals with chronic illnesses, that could alter predicted responses to contaminants.

Despite these uncertainties, the use of probability distributions that incorporate all definable sources of variability should provide a representative picture of the potential range of exposures.

5.0 TOXICITY ASSESSMENT

Several contaminants that have the potential for causing adverse human health effects have been detected in ground water at the site. This section summarizes the toxicological effects of the chemical contaminants and carcinogenic potentials of arsenic and the radionuclides. Source materials used in developing these toxicological profiles include, when available, the EPA's Integrated Risk Information System (IRIS); the Agency for Toxic Substances and Disease Registry Toxicological Profiles, published by the Department of Health and Human Services (DHHS); the *Handbook on the Toxicology of Metals* (Friberg et al., 1986); and peer-reviewed scientific literature as cited when these review documents were not available. By basing toxicity information on the standardized review documents cited above, the evaluation of risks at UMTRA Project sites should be consistent with evaluations at other sites.

The toxicity profiles presented in this section focus on drinking water source material in humans whenever available. Animal information is used only if human data are not available. Animal information on the toxicity range graphs is represented by the use of widely spaced dotted lines. When uncertainty exists about the beginning or ending points of a range of exposures that produces specific toxic effects, closely spaced dots will be used at the appropriate end of the line denoting range.

5.1 CONTAMINANT TOXICITY SUMMARIES

The following summaries address the basic toxicokinetics and toxicity of the contaminants of potential concern at Grand Junction based on the preliminary screening discussed previously. These are arsenic, cadmium, cobalt, fluoride, iron, manganese, molybdenum, nickel, sulfate, uranium, vanadium, zinc, and radium-226. Although these contaminants have a wide range of toxic effects, depending on the exposure levels, the following discussions focus most heavily on toxic effects observed in the exposure range most relevant to contamination at Grand Junction.

5.1.1 Arsenic

Absorption

Arsenic is effectively absorbed through the gastrointestinal tract and via inhalation. Dermal absorption is negligible. In humans, approximately 80 percent of an ingested amount of dissolved inorganic trivalent (arsenite) or pentavalent arsenic (arsenate) is absorbed from the gastrointestinal tract (Pershagen and Vahter, 1979; Marafante and Vahter, 1987).

Tissue accumulation and clearance

After absorption by the gastrointestinal tract, arsenic is transported via the blood to most tissues. In humans as well as in most animal species, exposure to either arsenite or arsenate leads to an initial accumulation in the liver, kidneys, and lungs. The clearance from these tissues is very rapid, and a long-term retention of arsenic is seen in organs rich in sulfhydryl containing proteins, such as the hair, skin, squamous epithelium of the upper gastrointestinal tract, epididymis, thyroid, lens, and skeleton (Lindgren et al., 1982). Specific target tissue is dependent on the form of arsenic. Higher retention of arsenic occurs after exposure to trivalent arsenic than to pentavalent form and tissue distribution is altered (Webb, 1966; Casarett and Doull, 1991).

In humans and rats, inorganic arsenic passes through the placental barrier. It has also been demonstrated to enter both cow and human milk (Marcus and Rispin, 1988).

In the human body, where methylcobalamine acts as a major methyl group donor in the biotransformation process, inorganic arsenic is converted to methylated compounds. It has been demonstrated that the major site of arsenic methylation is the liver (Marcus and Rispin, 1988). Trivalent arsenic is the substrate for methylation, and pentavalent arsenic must be reduced to trivalent arsenic before methylation can occur. Dimethylarsenic acid is a major metabolite found in animals and humans. Methylation results in a detoxification of inorganic arsenic (about one order of magnitude per methyl group) and increases the rate of arsenic excretion from the body.

The major route of excretion following human exposure to inorganic arsenic is via the kidneys (Ishinishi et al., 1986). Only a few percent is excreted in feces. The rate of excretion in urine varies depending on the chemical form of arsenic, the duration of exposure, and the species exposed. In humans exposed to a single low dose of arsenite, about 35 percent was excreted in urine over a period of 48 hours (Buchet et al., 1980; 1981). In the case of continuous human intake over a few days, 60 to 70 percent of the daily dose is excreted in urine (Buchet et al., 1981). Following exposure to arsenate, the limited human data available indicate a rate of excretion similar to that of arsenite. Other, less important routes of elimination of inorganic arsenic include skin, hair, nails, and sweat.

After oral intake of radiolabeled pentavalent arsenic, 66 percent was excreted with a half-time of 2.1 days, 30 percent with a half-time of 9.5 days, and 3.7 percent with a half-time of 38 days (Marcus and Rispin, 1988).

Environmental sources of arsenic

Arsenic is ubiquitous in nature in both inorganic and organic compounds. Water is the major means of transport of arsenic under natural conditions. In oxygenated water, arsenic occurs in a pentavalent form; but under reducing conditions, the trivalent form predominates. Sedimentation of arsenic in association with iron and aluminum represents a considerable factor in environmental transport and deposition of this element (Marcus and Rispin, 1988).

As a result of arsenic's widespread occurrence, the general human population is exposed to it primarily from drinking water and foodstuffs. Certain target groups are exposed to arsenic from industrial and agricultural uses. Medicinal use has also been a significant means of human exposure.

Drinking water usually contains a few micrograms of arsenic, predominantly as inorganic salts in the trivalent and pentavalent states (WHO, 1981). However, concentrations of up to 1.1 mg/L in drinking water have been reported in Chile, Argentina, Taiwan, the United States, and the United Kingdom (WHO, 1981).

Certain foodstuffs contain appreciable amounts of arsenic. The concentration of arsenic in fish and seafood, particularly shellfish, is generally one or two orders of magnitude higher than in other foods. Wine and mineral waters can contain several hundred micrograms of arsenic per liter (Crecelius, 1977; WHO, 1981).

Toxicity of arsenic

Levels of exposure associated with acute arsenic toxicity vary with the valency form of the element. Trivalent arsenicals (arsenites) are generally more toxic than pentavalent (arsenates) (Morrison et al., 1989), and inorganic arsenic compounds are more toxic than organic (Shannon and Strayer, 1989). Based on geochemical models for the Grand Junction site, arsenic exists primarily in the pentavalent form in ground water (Table 3.5). For arsenic trioxide, the reported estimated acute oral lethal dose in humans ranges from 70 to 300 mg (1 to 4 milligrams per kilogram [mg/kg]) (EPA, 1984). Acute exposure to inorganic arsenic compounds may lead to a severe inflammation of the gastrointestinal tract, encephalopathy, and an acute renal failure after ingestion.

Teratogenic effects of arsenic compounds administered intravenously or intraperitoneally at high doses have been demonstrated in laboratory animals only (Ferm, 1971; Hood, 1972; EPA, 1984). Teratogenic effects, also referred to as birth defects, can be defined as effects resulting in structural or functional anomalies in live offspring.

Increasing chronic oral ingestion doses of arsenic progressively produce systemic effects including 1) arterial thickening in children and adults (0.02 milligrams per kilogram [of body weight] per day [mg/kg-day]); 2) neurological symptoms including peripheral neuropathy (0.04 mg/kg-day); 3) fibrosis of the liver (0.05 mg/kg-day); and 4) cirrhosis of the liver (0.08 mg/kg-day) (DHHS, 1993).

Chronic arsenic intoxications result from exposure to even small doses of arsenic over a long period of time. These intoxications are frequently caused by arsenic content in drinking water and in food. Changes of the skin leading to skin cancer are commonly seen in populations exposed to high concentrations of arsenic in drinking water. Endemic arsenic poisoning is seen in Cordoba, Argentina, where the concentration of arsenic in drinking water ranges from 0.9 to 3.4 mg/L (equivalent to 0.026 to 0.097 mg/kg-day). Certain areas in Taiwan also have high natural arsenic concentrations in drinking water that cause blackfoot disease (a peripheral extremity vascular disorder resulting in gangrene). A dose-response relationship between the incidence of blackfoot disease and the duration of exposure to arsenic has been documented (Tseng, 1977).

Hyperpigmentation, hyperkeratoses, and skin cancer with prevalence of 7.1 percent, 18.4 percent, and 1.1 percent, respectively, were reported in Taiwanese studies of more than 40,000 people exposed to arsenic in drinking water at daily intakes ranging from 1.4 to 6.3 mg/L.

Certain characteristics of exposed human populations may influence arsenic toxicity at high exposure levels. Genetic dispositions (rapid versus poor acetylators) and protein-deficient diet may decrease the methylation of arsenic. This can result in an increased deposition of the element in the target organs (e.g., lung or skin).

The EPA has classified inorganic arsenic as a Group A (human) carcinogen (EPA, 1994), based on the occurrence of increased lung cancer mortality in populations exposed primarily via inhalation and of increased skin cancer prevalence in populations exposed through consumption of drinking water containing high concentrations of arsenic. The current slope factor (SF) for oral exposure to arsenic is given in Table 5.1. This SF is currently under review by EPA with respect to recent data suggesting arsenic ingestion may result in increased cancers in internal organs as well as skin cancers. The health effects from exposure to arsenic as a function of dose are summarized in Figure 5.1.

5.1.2 Cadmium

Humans absorb approximately 5 percent of the cadmium ingested through drinking water, but this figure can increase substantially with exposure to other metals (such as calcium or iron) or with increased protein intake (Friberg et al., 1986). The amount of cadmium absorbed from food sources is about half the amount absorbed from water. Absorption is also substantially increased in individuals with low iron stores (Flanagan et al., 1978). Once absorbed, cadmium is bound to protein, primarily metallothionein. The ability of many metals to increase the concentration of metallothionein is the likely basis for interactions in absorption, tissue concentrations, and toxicity of combined exposures to metals.

Tissue accumulation and clearance

Humans with low-level exposure to cadmium show approximately 50 percent of the body burden in the kidneys, 15 percent in the liver, and 20 percent in muscle (Kjellström, 1979). Kidney concentration increases with continued exposure only to about age 50, but concentration in muscle increases throughout life. When high exposure results in kidney damage, kidney concentrations can be quite low, but liver concentrations can be 100 times

Table 5.1 Toxicity values: carcinogenic effects

Parameter	Oral SF $(pCi)^{-1}$	Weight of evidence classification	Type of cancer	SF basis/SF source
Arsenic (inorganic)	1.8E+0[a]	A	Skin	Water/HEAST
Lead-210	5.1E-10	A	Bone	Water/HEAST
Polonium-210	1.5E-10	A	Liver, kidneys, spleen	Water/HEAST
Radium-226	1.2E-10	A	Bone	Water/HEAST
Thorium-230	1.3E-11	A	Bone	Water/HEAST
Uranium-238	1.6E-11	A	Note[b]	Water/HEAST
Uranium-234	1.6E-11	A	Note[b]	Water/HEAST

[a] In $(mg/kg\text{-}day)^{-1}$.

[b] No human or animal studies have shown a definite association between oral exposure to uranium and development of cancer.

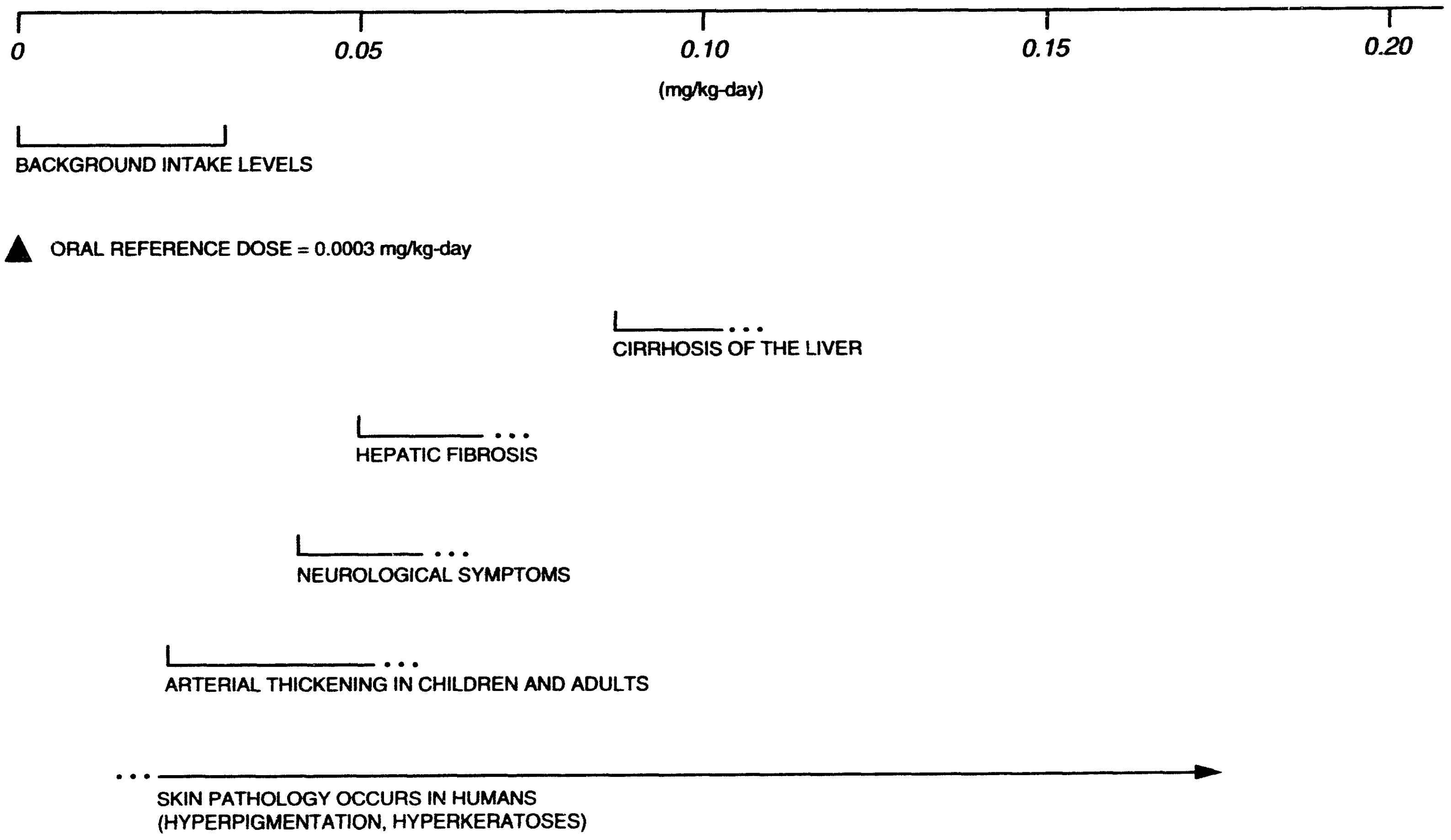
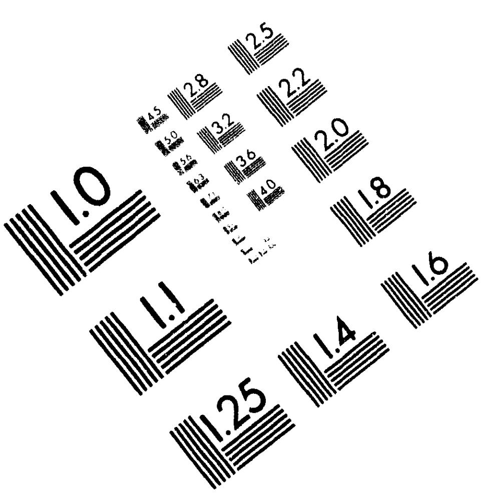


FIGURE 5.1
ARSENIC TOXICITY RANGES



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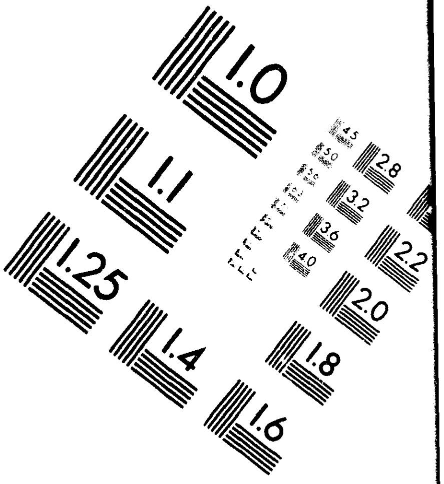

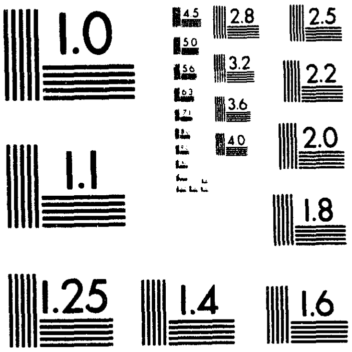

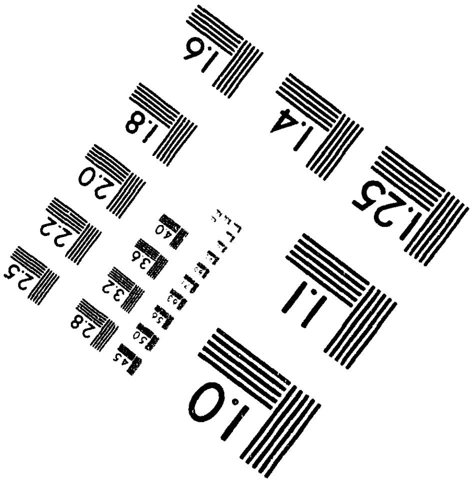

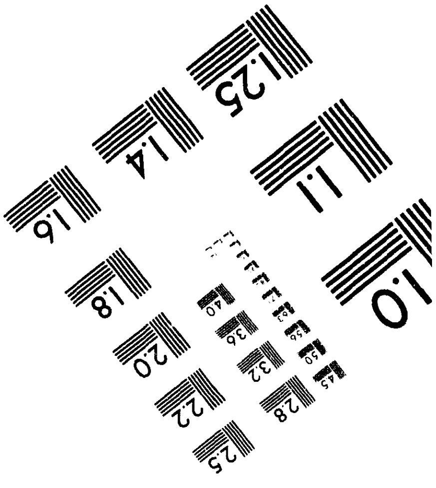

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higher than normal. Only 0.01 to 0.02 percent of the total body burden of cadmium is excreted daily, resulting in continuously increasing body burdens with continuous exposure. The biological half-time of cadmium, or the time needed to eliminate 50 percent of the cadmium in the body at a given time, is 10 to 30 years in humans (Nordberg et al., 1985).

Environmental sources of cadmium

The normal cadmium content of food and water in nonpolluted areas results in 0.01 to 0.06 mg/day intake of cadmium (0.0001 to 0.0009 mg/kg-day). Cadmium occurs naturally with zinc and lead; therefore, cadmium is often present as an impurity in products using these metals (for example, solders and galvanized metals). These products can lead to contact with water supplies (water heaters and coolers, some pipes, and taps).

Toxicity of cadmium

Acute exposure to high concentrations of cadmium (15 mg/L in water) results in acute gastrointestinal effects, including abdominal cramps, diarrhea, and vomiting (at 0.07 mg/kg). These gastrointestinal effects have not been reported in any chronic environmental exposure.

The primary toxic effect of long-term exposure to cadmium is disturbance of reabsorption in the proximal tubules of the kidney. This effect is first observed by an increase of low molecular-weight proteins in the urine. This initial effect is observed following a daily intake of 0.0075 mg/kg-day. Progressive disruption of kidney function will lead to an increase in amino acids, glucose, phosphate, and protein in urine. Long-term exposures can also disturb calcium metabolism, leading to osteoporosis and osteomalacia. A combination of these two effects is referred to as *Itai-itai* disease and was seen in epidemic proportions in a cadmium-contaminated region in Japan in the 1950s (Friberg et al., 1986). These health effects are summarized in Figure 5.2 as a function of dose.

5.1.3 Cobalt

Gastrointestinal absorption of soluble cobalt compounds is estimated to be about 25 percent with wide individual variation; the gastrointestinal absorption in individuals reportedly varies from 5 to 45 percent (Friberg et al., 1986).

Cobalt is an integral component of vitamin B_{12}. The total vitamin B_{12} content of the body in a normal (that is, nondeficient) adult human is about 5 mg, which is equivalent to about 0.2 mg of cobalt (Friberg et al., 1986).

Tissue accumulation and clearance

In humans exposed to cobalt, the liver exhibits the highest concentration, followed by the kidneys. Excretion occurs mainly through the urinary tract.

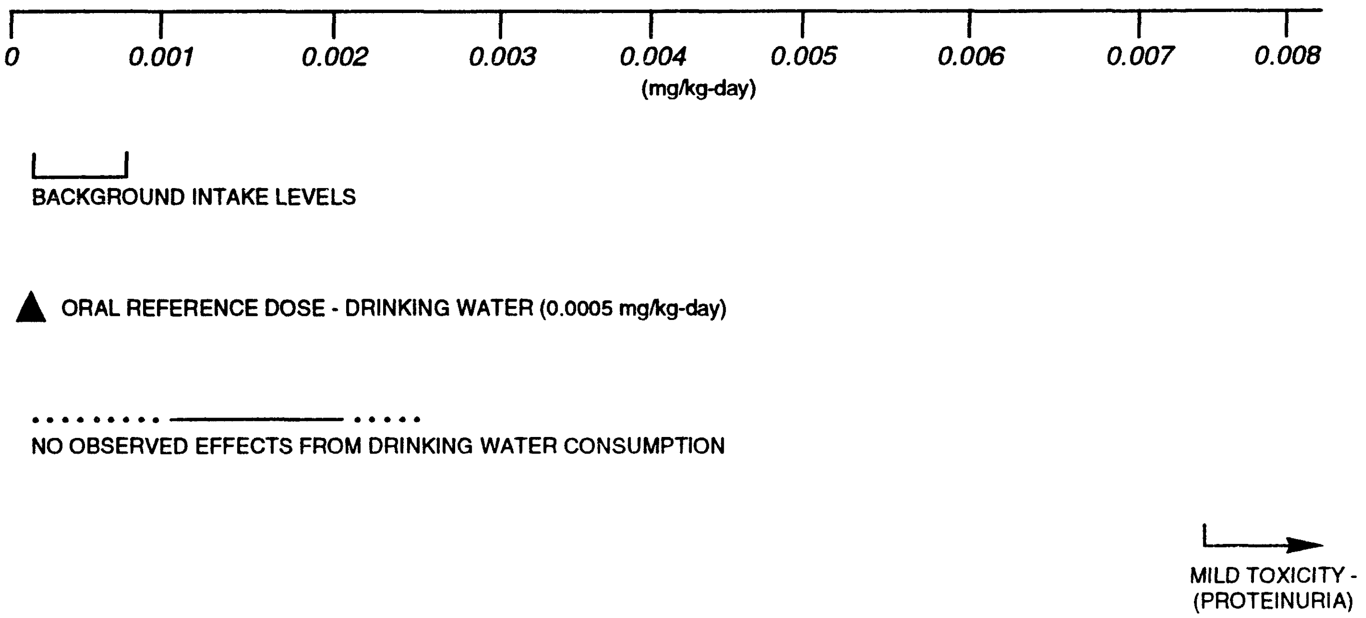


FIGURE 5.2
CADMIUM TOXICITY RANGES

MAC: SITE/GRJ/BRA/CAD-GEN

Apparently, most cobalt is eliminated rapidly (within days) for all exposure routes (inhalation, injection, or ingestion). However, a small proportion is eliminated slowly, with a biological half-time on the order of years (Friberg et al., 1986).

Data are inadequate on the cobalt levels in tissues and fluids of background populations (that is, persons not occupationally exposed) in the United States (DHHS, 1992).

Environmental sources of cobalt

Cobalt occurs naturally in the earth's crust and, as a result, in soil. Cobalt compounds occur naturally in seawater and in some surface, spring, and ground waters. Cobalt also is released into water from industrial and commercial sources. Cobalt is a by-product or coproduct of refining other mined metals (for example, copper and nickel) (DHHS, 1992).

Only limited data are available on the levels of cobalt in United States foodstuffs. Therefore, the cobalt intake from food in the United States cannot be determined (DHHS, 1992).

Toxicity of cobalt

Cobalt is an essential nutrient as an integral component of vitamin B_{12}. No other function for cobalt in human nutrition has been established. Adding cobalt to beer has caused endemic outbreaks of cardiomyopathy (damage to the heart muscle) among heavy beer drinkers, with a 50-percent mortality rate. Similar effects on the heart, including myocardial degeneration and electrocardiographic changes, have been seen in laboratory animals after repeated parenteral or oral exposure to cobalt (Friberg et al., 1986).

The average daily intake of cobalt from food is 5 to 45 micrograms (μg) (about 0.00007 to 0.0006 mg/kg-day). The recommended daily intake of B_{12} for an adult is 3 μg, corresponding to 0.012 μg of cobalt (Friberg et al., 1986)

Cobalt is used in the medical treatment of anemias and has an erythropoietic effect (that is, it stimulates the production of red blood cells). Duckham and Lee (1976) gave 12 anemic patients daily doses of cobalt chloride orally in amounts corresponding to 6.2 and 12.4 mg cobalt per day for 12 to 30 weeks (approximately 0.09 to 0.13 mg/kg-day). This treatment gave rise to an average increase in the hemoglobin concentration of 46 percent. After cobalt treatment ceased, the hemoglobin levels decreased. In addition to cardiomyopathy, polycythemia (increased number of red blood cells) was reported in heavy drinkers of cobalt-contaminated beer. It may be assumed that a very heavy beer drinker consuming up to 10 L/day of beer acquires an additional cobalt intake of approximately 10 mg/day (approximately 0.14 mg/kg-day). Although this figure is excessively high compared with nutritional standards, it is not as large as doses given to treat anemias (Friberg et al., 1986).

High levels of chronic oral cobalt exposure may result in the production of goiter. Epidemiologic studies suggest that the incidence of goiter is higher in regions containing increased levels of cobalt in the water and soil. The goitrogenic effect has been elicited by oral administration of 3 to 4 mg/kg to children in the course of sickle cell anemia therapy (Casarett and Doull, 1991). The toxicity of cobalt is summarized in Figure 5.3.

5.1.4 Fluoride

Absorption

Fluorides in water are absorbed primarily from the gastrointestinal tract; the degree of absorption depends on the solubility of a particular fluoride compound.

The absorption of fluoride from water is estimated to be 100 percent, while protein binding in food sources reduces dietary absorption. In young adults, the absorption of fluoride from milk or baby formula is determined to be 72 and 65 percent, respectively, of that from water (National Research Council, 1989). Poorer absorption, from 37 to 54 percent, has been reported for fluorine in bone meal.

Tissue accumulation and clearance

Fluoride has been detected in all organs and tissues. Following gastrointestinal absorption, fluoride is distributed primarily to bones and is deposited in the skeleton and tooth enamel with lesser deposition in the thyroid, aorta, and kidney (Gilman et al., 1990; National Research Council, 1989). The degree of skeletal storage is related to intake and age. Storage in bone is thought to be function of the turnover rate of skeletal components, with growing bone showing a greater fluoride deposition than in mature organisms. Prolonged periods of time are required for mobilization of fluoride from bone. The half-time for turnover in the young adult skeleton is about 8 to 10 years (Maheshwari et al., 1981).

The major route of fluoride excretion is the kidney; however, fluoride is also excreted in small amounts by the sweat glands, the lactating breast, and the gastrointestinal tract. The fraction of total fluoride excretion contributed by excessive sweating can reach nearly one-half (Gilman et al., 1990). About 70 percent of ingested fluoride is excreted in urine, and about 5 percent of that retained and absorbed is excreted in the feces (Maheshwari et al., 1981). About 90 percent of the fluoride filtered by the glomerulus is reabsorbed by the renal tubules (Gilman et al., 1990).

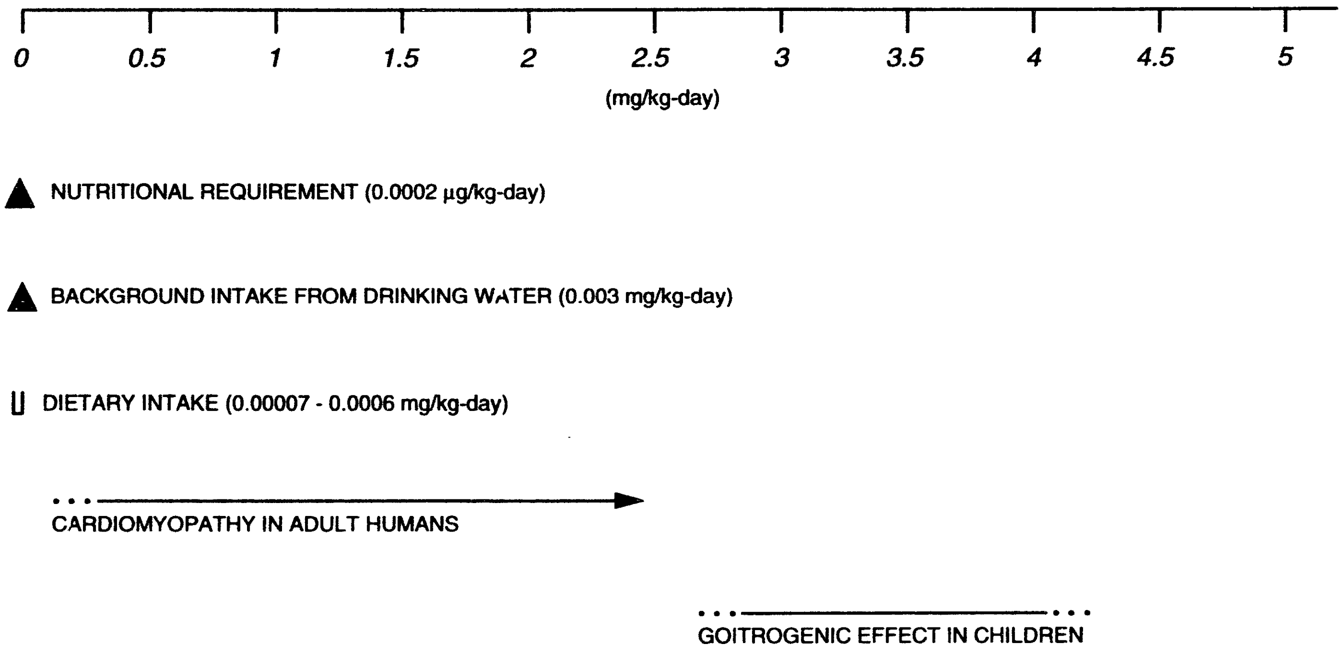


FIGURE 5.3
COBALT TOXICITY RANGES

Environmental sources of fluoride

Drinking water and food are the primary sources of fluoride intake by humans. Drinking water, whether fluoridated or not, can contribute significantly to the total daily fluoride intake. In fluoridated areas, the contribution ranges from about 26 to 54 percent of the total intake (National Research Council, 1980). In unfluoridated areas, it ranges from about 14 to 48 percent.

Most public water supplies contain fluoride, and the majority of them contains less than 1 mg fluoride/L. However, as much as 4.4 mg fluoride/L has been reported (0.18 mg/kg-day for a 25-kg child ingesting 1 L of water per day, or 0.13 mg/kg-day for a 70-kg adult ingesting 2 L of water per day) (National Research Council, 1980). River water contains fluoride concentrations up to 6.5 mg/L; lakes contain up to 1627 mg/L; and sea water has an average concentration of 1.2 mg/L.

The richest dietary sources of fluoride are tea and marine fish consumed with their bones (National Research Council, 1989). In the United Kingdon, tea accounted for 72 percent (1.3 mg) of the total adult daily intake of 1.8 mg (National Research Council, 1989). The fluoride content of cow's milk is approximately 0.02 mg/L. Mean reported values for human milk range from 0.005 to 0.025 mg/L, depending on maternal intake (mothers were drinking water containing 0.2 and 1.7 mg/L, respectively). Dietary fluoride intake up to 3.44 mg/day (0.05 mg/kg-day for a 70-kg adult) has been reported in some areas of the United States (National Research Council, 1980). Average fluoride dietary intake of 0.01 mg/kg-day has been reported for both a 20-kg child and a 70-kg adult (EPA, 1994).

Food processing has a substantial influence on the fluoride content of foods. The fluoride content of various foods can increase severalfold by cooking them in fluoridated water. Cooking in utensils treated with Teflon®, a polymer containing fluoride, can increase the fluoride content, whereas an aluminum surface can reduce it (National Research Council, 1989).

The estimated safe and adequate daily dietary intake of fluoride for adults ranges from 1.5 to 4.0 mg/day (equivalent to 0.02 to 0.06 mg/kg-day for a 70-kg man) (National Research Council, 1989). This accounts for widely varying fluoride concentrations in diets consumed in the United States and includes both food sources and drinking water. For younger age groups, the estimated maximum level of this intake is 2.5 mg/day (equivalent to 0.1 mg/kg-day for a 25-kg child). Ranges of 0.1 to 1 mg/day during the first year of life (equivalent to 0.03 to 0.3 mg/kg-day for a 4-kg infant) and 0.5 to 1.5 mg/day during the subsequent 2 years are suggested as adequate and safe (National Research Council, 1989).

In view of fluoride's beneficial effects on dental health and its suggested safety at the estimated safe and adequate daily dietary levels, the Food and Nutrition

Board recommends that public water supplies be fluoridated if natural fluoride levels are substantially below 0.7 mg/L (National Research Council, 1989).

Toxicity of fluoride

Although fluoride can have beneficial effects on teeth and bone at low doses, higher doses of fluoride can be toxic. Children are particularly sensitive to dental fluorosis, the critical toxic effect of fluoride (EPA, 1994). It has been established that fluoridation of water to a concentration of 1 mg/L (0.04 mg/kg-day for a 25-kg child, assuming ingestion of 1 L of water) is a safe and practical public health measure that substantially reduces the incidence of caries in permanent teeth (Gilman et al., 1990). Fluoride is also used in clinical practice to treat osteoporosis in larger doses than those used to prevent dental caries (Maheshwari et al., 1981). However, the optimal level of fluoride intake for osteoporosis therapy has not been determined.

Fluoride is an inhibitor of several enzyme systems and diminishes tissue respiration and anaerobic glycolysis. It also binds calcium $Ca(+2)$ and inhibits the glycolytic utilization of glucose by erythrocytes (Gilman et al., 1990).

Acute fluoride poisoning usually results from the accidental ingestion of insecticides or rodenticides containing fluoride salts (Gilman et al., 1990). The lethal dose of fluoride for a 70-kg adult is in the range of 32 mg/kg.

In humans, the major manifestations of chronic ingestion of excessive amounts of fluoride are dental fluorosis (mottled enamel) and osteosclerosis (crippling skeletal fluorosis) (Gilman et al., 1990; National Research Council, 1989; Casarett and Doull, 1991). Long-term exposure to excess fluoride causes increased osteoblastic activity.

In very mild tooth mottling, the gross changes consist of small, opaque, paper-white areas scattered irregularly over the tooth surface. In severe cases, discrete or confluent, deep brown- to black-stained pits give the tooth a corroded appearance. Mottled enamel or dental fluorosis is the result of a partial failure of the enamel-forming cells to elaborate properly and lay down enamel. It is a nonspecific response to a number of stimuli, one of which is the ingestion of excessive amounts of fluoride.

Because mottled enamel is a developmental disease, the ingestion of fluoride following the eruption of the tooth has no effect (Gilman et al., 1990). Mottling is one of the first visible signs of an excessive intake of fluoride during childhood. Continuous use of water containing about 0.7 to 1.3 mg fluoride/L (equivalent to 0.03 to 0.05 mg/kg-day for a 25-kg child, assuming ingestion of 1 L of water per day), depending on ambient temperature and diet, produces dental mottling and changes in tooth structure in 10 percent of children (National Research Council, 1980; Gilman et al., 1990). These effects were evaluated as the very mildest form of mottled enamel. At fluoride levels of 4 to

6 mg/L (0.16 to 0.24 mg/kg-day for a 25-kg child, assuming ingestion of 1 L of water), the incidence reaches 100 percent, with marked increase in severity.

In osteosclerosis, as opposed to osteoporosis, bone density and calcification increase. Fluoride intoxication is thought to represent the replacement of hydroxyapatite by the denser fluorapatite. However, the mechanism of fluoride intoxication development is unknown. The degree of skeletal involvement varies from changes that are barely detectable radiologically to marked thickening of the cortex of long bones, numerous exostoses scattered throughout the skeleton, and calcification of ligaments, tendons, and muscle attachments to bone. In its severest form, osteosclerosis is a disabling disease and is designated as crippling fluorosis. It has been estimated that the development of crippling skeletal fluorosis in humans requires daily ingestion of 20 to 80 mg fluoride (0.29 to 1.1 mg/kg-day for a 70-kg adult) over a 10- to 20-year period (National Research Council, 1980, 1989; EPA, 1994). Although the no-observed-effect level for crippling skeletal fluorosis in humans is unknown, a safe total fluoride exposure level (from food and drinking water) for adults is suggested to be 0.12 mg/kg-day. This exposure level would correspond to the consumption of 2 L of water per day containing 4 mg fluoride/L by a 70-kg adult and ingestion of 0.01 mg fluoride per day in the diet.

The EPA oral reference dose (RfD) of 0.06 mg/kg-day was developed based on the no-observed-adverse-effect level (NOAEL) of 1 mg fluoride/L of drinking water, determined in children from 12 to 14 years old, and the assumption that a 20-kg child consumes 0.01 mg fluoride/kg-day in the diet (EPA, 1994).

The toxic effects and doses for fluoride are summarized in Figure 5.4.

5.1.5 Iron

Absorption

The percentage of dietary iron that is absorbed ranges from 2 percent in individuals with diseases of the gastrointestinal tract to 35 percent in rapidly growing, healthy children (Goyer, 1991; Whitney et al., 1990). Normally, 10 to 15 percent of dietary iron is absorbed, but this percentage varies to compensate for the level of iron in the body (Elinder, 1986). For example, patients with iron-deficiency anemia can absorb as much as 60 percent of an oral dose of iron (Josephs, 1958).

Iron absorption also is influenced by factors such as source and chemical form of the ingested iron, other substances in the diet, and the condition of the gastrointestinal tract (Elinder, 1986). Very little is known about the absorption of iron from water and about the chemical species of iron in drinking water from the tap. Although the amount of ferric ion (Fe^{3+}), ferrous ion (Fe^{2+}), and organic complexes of iron in water that are absorbed by humans is unknown, it is clear that a reducing agent such as ascorbic acid increases the absorption of iron in food (National Research Council, 1980). Ferrous ion appears to have

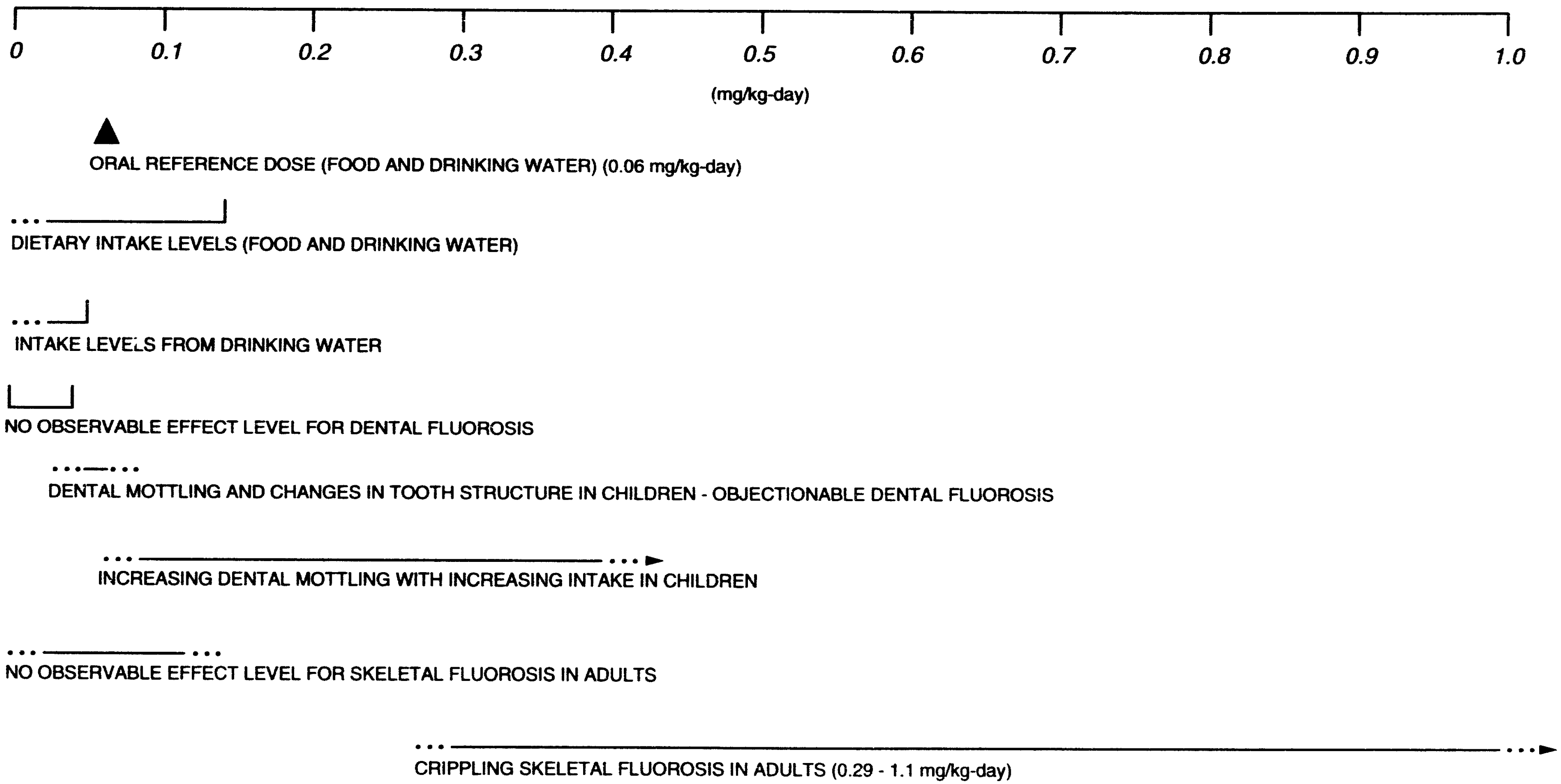


FIGURE 5.4
FLUORIDE TOXICITY RANGES

better availability than does ferric ion. Iron from animal sources is absorbed by humans more effectively than iron from vegetables and grains. Soluble forms of iron such as iron sulfate are taken up more readily than insoluble forms such as iron oxide. The presence of other metals also affects iron absorption. Absorption is decreased in the presence of high levels of phosphate, cobalt, copper, and zinc (Elinder, 1986). Excess manganese can significantly decrease iron absorption by impairing hemoglobin regeneration in the blood (National Research Council, 1980).

Tissue accumulation and clearance

Iron absorption from the gastrointestinal tract occurs in two steps: first, ferrous ions from the intestinal lumen are absorbed into the mucosal cells. Second, they are transferred from the mucosal cells to plasma, where they are bound to transferrin for transfer to storage sites. As ferrous ion is released into plasma, it is oxidized by oxygen in the presence of ferroxidase (Goyer, 1991).

Normally, the adult human body contains about 3 to 5 grams of iron. Two-thirds of this amount is found in the blood, bound to hemoglobin. Less than 10 percent of body iron is found in myoglobin and iron-requiring enzymes. About 20 to 30 percent of the remaining iron in the body pool is bound to iron-storage proteins in the liver, bone marrow, and spleen (Elinder, 1986).

Under normal conditions, the total elimination of iron from the body is limited to 0.6 to 1.0 mg/day, or roughly 0.01 percent of the body stores. Not counting iron not absorbed from the gut, about 0.2 to 0.5 mg of elemental iron per day is eliminated through the feces, about 0.1 to 0.3 mg/day in urine, and the remainder through normal dermal losses in sweat, hair, and nails. Based on these rates of elimination, the biological half-life of iron in the body is 10 to 20 years (Elinder, 1986).

Environmental sources of iron

The iron concentrations of liver, kidney, beef, ham, egg yolk, and soybeans are on the order of 30 to 150 mg/kg fresh weight. Grains and fruits are low in iron, usually ranging from 1 to 20 mg/kg. In both human and cow's milk, iron concentration is about 0.5 mg/L (Elinder, 1986).

Average daily intakes of iron range from 9 to 35 mg/day (0.1 to 0.5 mg/kg-day) (Elinder, 1986). Approximately 35 percent of dietary iron comes from meat, fish, and eggs, while 50 percent is supplied by cereals, root vegetables, and other foods of plant origin (National Research Council, 1980).

Iron concentrations in water vary greatly. In the United States, the iron concentrations of freshwater and public water supplies range from 0.01 to 1.0 mg/L (Elinder, 1986). Assuming a 2 L/day consumption of water by a 70-kg (body weight) adult, this range would result in an intake of 0.0003 to 0.03 mg/kg-day of iron from drinking water.

The recommended daily allowance (RDA) for iron is 10 mg (approximately 0.14 mg/kg-day) for adult males and 18 mg (approximately 0.25 mg/kg-day) for females of reproductive age (National Research Council, 1980).

Toxicity of iron

Iron intoxication is most frequent in children aged 1 to 3 years. This generally occurs when children eat iron supplements formulated for adults in the form of ferrous sulfate tablets with candy-like coatings. Severe poisoning in children may occur following ingestion of more than 0.5 gram (approximately 22 mg/kg) of iron, about 2.5 grams (approximately 110 mg/kg) as ferrous sulfate. Acute iron poisoning has occurred in children who ingested as few as six iron tablets (Whitney et al., 1990). The iron damages the lining of the gastrointestinal tract, producing vomiting as the first symptom. Bleeding of the damaged gastrointestinal tissue frequently results in blood in the vomit and black stools (Goyer, 1991). Shock and metabolic acidosis can develop. If the patient survives the initial crisis, liver damage with hepatitis and coagulation defects often occur within a couple of days. Renal failure and cirrhosis of the liver may occur as delayed effects (Elinder, 1986).

Long-term intake of iron in a form that is readily absorbed and in doses exceeding 50 to 100 mg of iron per day (0.7 to 1.4 mg/kg-day for a 70-kg adult male) (Elinder, 1986) results in an increased body burden of iron because iron is removed from the body at a much slower rate than it is absorbed. As the body burden of iron increases to between 20 and 40 grams (roughly 10 times the normal level), production of the iron-binding protein hemosiderin increases and results in a condition known as hemochromatosis. This condition starts with increased pigmentation of the skin and higher concentrations of iron in the liver, pancreas, endocrine organs, and heart. This increased tissue iron can produce cirrhosis of the liver, disturbances in endocrine and cardiac function, and diabetes mellitus (Goyer, 1991).

Chronic iron toxicity in adults can be caused by genetic factors, excess dietary iron, excessive ingestion of tonics or medicines containing iron, or multiple blood transfusions. The pathologic consequences of iron overload are similar regardless of cause (Goyer, 1991).

The toxic effects and doses for iron are summarized in Figure 5.5.

5.1.6 Manganese

Absorption

Following ingestion, manganese absorption is homeostatically controlled: the rate of absorption depends on both the amount ingested and tissue levels of manganese. For adult humans, approximately 3 to 4 percent of dietary manganese is absorbed (Saric, 1986). Manganese can be absorbed following exposure by inhalation, ingestion, and dermal contact. In humans, available

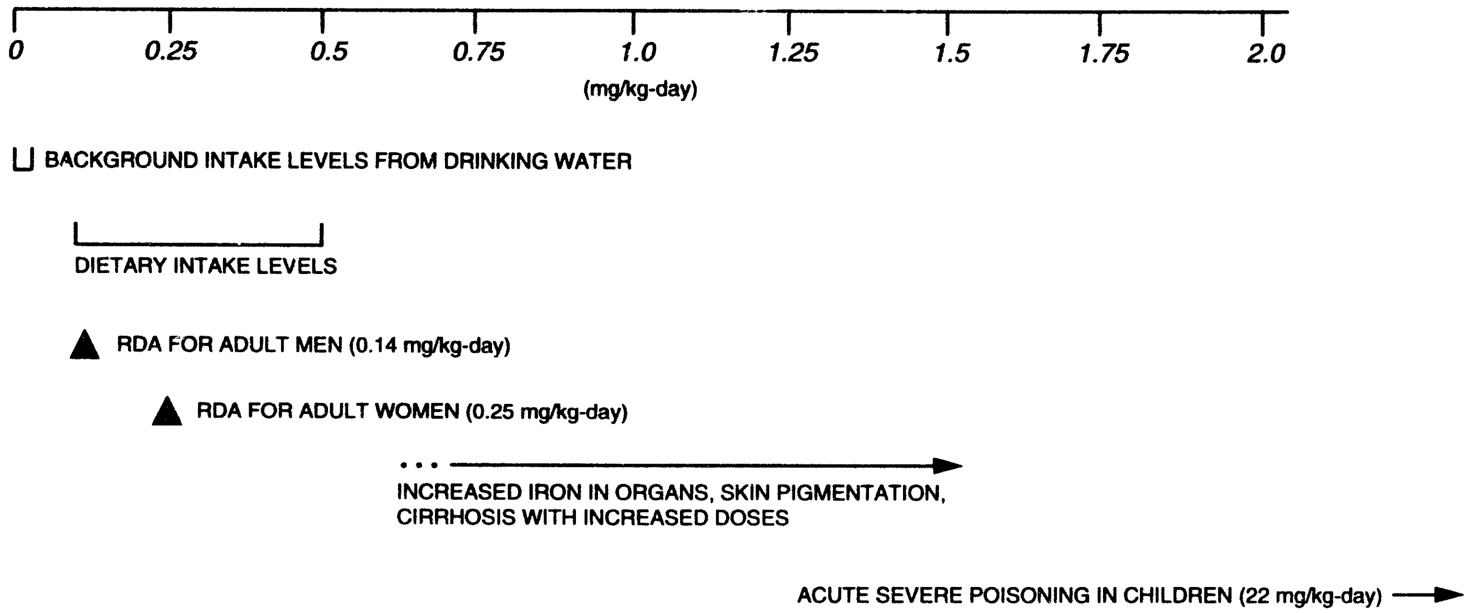


FIGURE 5.5
IRON TOXICITY RANGES

data indicate that only 3 percent of an ingested dose of manganese chloride is absorbed (Mena et al., 1969). The rate of absorption is influenced by iron and other metals. In states of iron deficiency, manganese is actively absorbed from the intestine. Individuals with anemia can absorb more than twice the percentage of an ingested dose. However, in states of excess iron, absorption of manganese is by diffusion only (Saric, 1986). High levels of dietary calcium and phosphorus have been shown to increase the requirements for manganese in several species (Lönnerdal et al., 1987).

Tissue accumulation and clearance

Manganese is widely distributed throughout the body. Highest concentrations are found in the liver and kidney and, to a lesser extent, the hair. The biological half-time in humans is 2 to 5 weeks, depending on body stores. Manganese readily crosses the blood-brain barrier and is more slowly cleared from brain than from other tissues (Goyer, 1991). Normal concentrations in the brain are low, but the half-time in the brain is longer and the metal may accumulate in the brain with excessive absorption (National Research Council, 1973).

Absorbed manganese is rapidly cleared from the blood and concentrates in mitochondria. Initial concentrations are greatest in the liver. Manganese penetrates the placental barrier in all species and is more uniformly distributed throughout the fetus than in adult tissues. It is secreted into milk.

Absorbed manganese is almost totally secreted in bile and reabsorbed from the intestine as necessary to maintain body levels. At excessive exposure levels, other gastrointestinal routes may participate. Excess manganese is eliminated in the feces; urinary excretion is negligible (Goyer, 1991; Saric, 1986).

Environmental sources of manganese

On the whole, food constitutes the major source of manganese intake for humans. The highest manganese concentrations are found in plants, especially wheat and rice. Drinking water generally contains less than 0.1 mg/L. Manganese levels in soil range from 1 to 7000 mg/kg, with an average of 600 to 900 mg/kg. Mining and natural geological background variation can contribute to this variability. Manganese bioaccumulates in marine mollusks up to 12,000-fold, and there is evidence for toxic effects in plants (phytotoxicity) and plant bioaccumulation. The Illinois Institute for Environmental Quality has recommended a criterion of 1 to 2 mg/kg for manganese in soil and 200 mg/kg in plants (Saric, 1986).

Variations in manganese intake can be explained to a large extent by differences in nutritional habits. In populations with cereals and rice as main food sources, the intake will be higher compared to areas where meat and dairy products make up a larger part of the diet. The average daily intake has been estimated to be between 2.0 and 8.8 mg/day (0.03 and 0.13 mg/kg-day) (EPA, 1994), but intakes as high as 12.4 mg (about 0.2 mg/kg-day) have been reported in countries with high cereal intake (Saric, 1986).

Drinking water generally results in an intake of less than 0.2 mg (0.003 mg/kg-day), although some mineral waters can increase this amount by more than threefold (Saric, 1986). One study from Greece reported drinking water concentrations of manganese in excess of 2 mg/L, which would result in daily intakes in the range of 0.06 to 0.07 mg/kg-day (EPA, 1994).

Toxicity of manganese

Manganese is an essential nutrient. Estimated safe and adequate daily dietary intakes for adults range from 0.03 to 0.07 mg/kg-day (Saric, 1986). The EPA NOAEL for drinking water is set at 0.005 mg/kg-day while the lowest-observed-adverse-effect level (LOAEL) for drinking water sources is 0.06 mg/kg-day. The EPA RfD for drinking water is 0.005 mg/kg-day. The RfD for food ingestion is 0.14 mg/kg-day. There is some indication that manganese in drinking water is potentially more bioavailable, i.e., more readily absorbed, than is manganese in dietary food sources. This would result in toxic effects with lower ingested doses of manganese in drinking water than in food (EPA, 1994).

Inhalation of manganese in industrial settings has provided the largest source of data on chronic manganese toxicity. These data indicate that excess manganese can result in a central nervous system disorder consisting of irritability, difficulty in walking, speech disturbances, and compulsive behavior that may include running, fighting, and singing. With continued exposure, this condition can progress to a mask-like face, retropulsion or propulsion, and a Parkinson-like syndrome. The condition reverses slowly with removal of manganese exposure. Metal chelating agents are ineffective in treatment, but L-dopa has been effective in treatment (Goyer, 1991), suggesting that manganese produces functional deficit in the central nervous system.

Limited information is available on the effects of manganese ingestion. Because effects from drinking water seem to differ from those from food sources, only studies on water consumption will be considered here. A Japanese study of 25 people drinking well water with manganese concentrations of 14 mg/L (0.4 mg/kg-day estimated intake) reported symptoms of intoxication, including a mask-like face, muscle rigidity and tremors, and mental disturbances. There were two cases (8 percent) of death among intoxicated people. A Greek study of over 4000 individuals drinking water with manganese concentrations varying from 0.081 to 2.3 mg/L (estimated intake at 2 L/day for a 70-kg individual range from 0.002 to 0.07 mg/kg-day) showed varying degrees neurological effects in individuals drinking from 0.007 to 0.07 mg/kg-day, but no effects in individuals drinking less than 0.005 mg/kg-day (Kondakis et al., 1989).

The chemical form of manganese has complex effects on its toxicity. Although more soluble forms are more readily absorbed from the gastrointestinal tract, they also appear to be more rapidly cleared. Exposure to insoluble forms results in lower manganese absorption, but higher chronic tissue levels and therefore greater toxicity (EPA, 1994). Only limited information is available on the effects of various forms of manganese.

Few data are available on manganese toxicity in infants, but it is likely that infants will be more susceptible to toxicity due to greater absorption and greater penetration into the central nervous system (EPA, 1994; Saric, 1986).

The toxicity of manganese from drinking water exposure is summarized in Figure 5.6.

5.1.7 Molybdenum

Absorption

Absorption of molybdenum in the gastrointestinal tract depends on the species of the metal. Both inorganic and hexavalent forms such as molybdenum trioxide, sodium molybdate, and ammonium molybdate are readily absorbed from both food and water, whereas molybdenite is not. Human absorption rates of 40 percent to 70 percent have been observed for molybdenum (Tipton et al., 1969; Robinson et al., 1973; Alexander et al., 1974).

Tissue accumulation and clearance

In humans, the highest concentrations of molybdenum occur in the liver, kidney, and adrenals (Casarett and Doull, 1991). With normal dietary intake, molybdenum levels in the body slowly increase until approximately age 20, then begin to decline steadily. The principal route of excretion in humans is in the urine. Human studies indicate the biological half-life in humans is considerably longer than in animals and may be as long as 2 weeks (Rosoff and Spencer, 1964).

Environmental sources of molybdenum

The occurrence of natural molybdenum is in combination with other metals, including uranium, lead, iron, cobalt, and calcium. Native soil concentrations can vary by as much as two orders of magnitude, from 0.1 mg/kg to 10 mg/kg, leading to large variations in the molybdenum concentrations in plant materials. Natural concentrations in ground water have been reported from 0.00011 mg/L to 0.0062 mg/L. Human dietary intake of molybdenum has been estimated at 0.05 to 0.24 mg/day (0.0007 to 0.003 mg/kg-day). The contribution of drinking water is estimated to range from 0 percent to 95 percent. The nutritional range of intake for molybdenum is from 0.0015 to 0.0054 mg/kg-day. No symptoms of molybdenum deficiency have been reported in humans. Nonetheless, molybdenum is an essential trace element that functions as a necessary constituent of several enzymes, including xanthine oxidase (which is involved in the metabolism of uric acid) and nitrate reductase (Friberg et al., 1986).

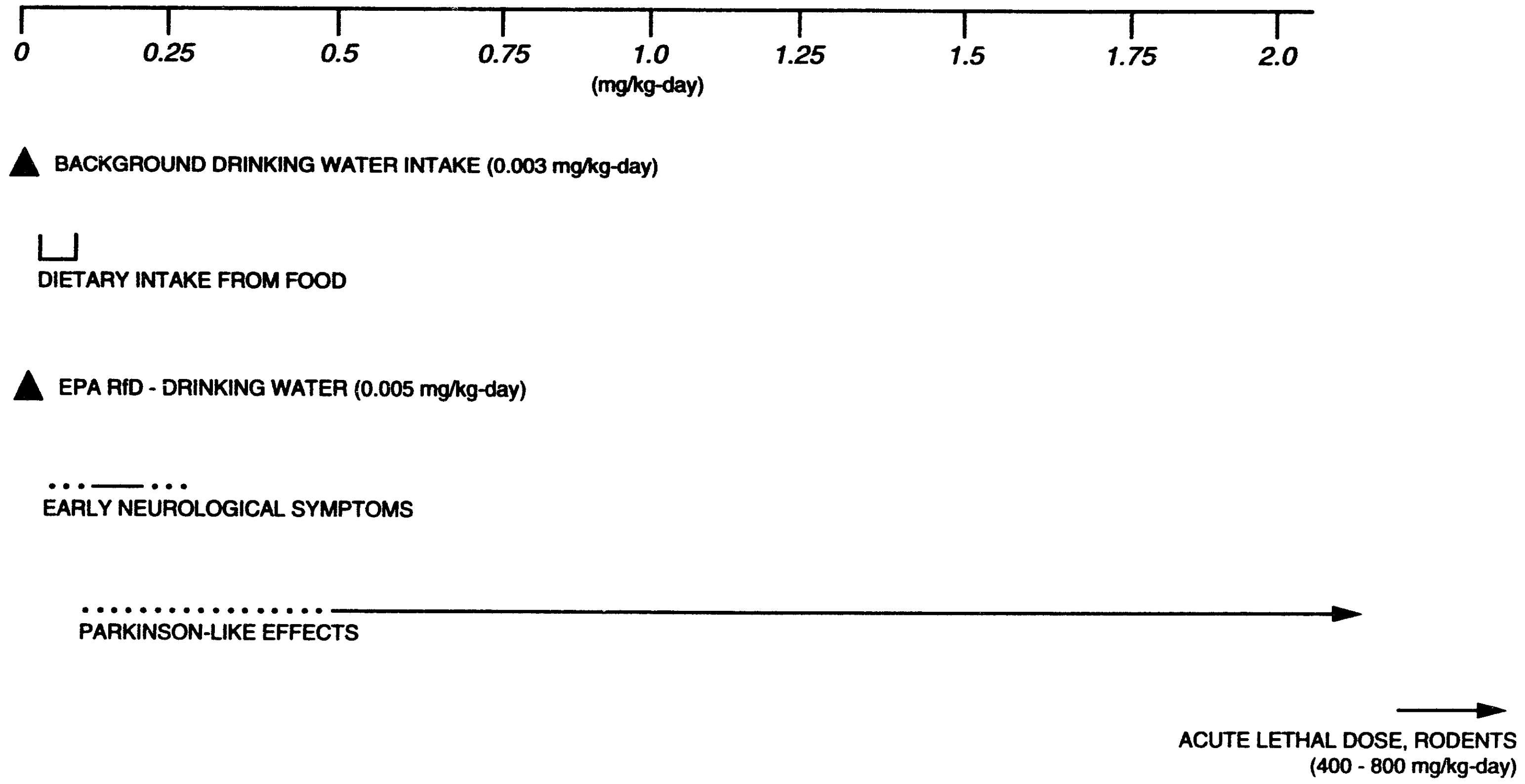


FIGURE 5.6
MANGANESE TOXICITY RANGES

Toxicity of molybdenum

Acute toxic effects of molybdenum have not been reported. No adverse health effects have been reported with chronic intake of less than 0.008 mg/kg-day of molybdenum. The primary toxicity of molybdenum is related to its interactions with copper and sulfur, leading to altered excretion patterns for these elements. Increased levels of molybdenum also increase the levels of xanthine oxidase, which is responsible for the production of uric acid. High levels of uric acid can accumulate in joints and lead to symptoms of gout and other joint disorders.

Intake of 0.008 to 0.022 mg/kg-day of molybdenum can produce mineral imbalance as a result of increased copper excretion. Excretion of copper has been reported to double with molybdenum intakes at the upper end of this range. Copper is an essential nutrient important in many metabolic pathways, including the synthesis and function of hemoglobin. A copper deficiency resulting from excess excretion will impair the oxygen-carrying capacity of the blood, and severe copper deficiencies can lead to hypochromic microcytic anemia. In humans, gout-like symptoms and joint deformities have been reported in regions of Russia where elevated soil concentrations of molybdenum and subsequent increased molybdenum concentrations in food would lead to molybdenum intakes in the range of 0.14 to 0.21 mg/kg-day. These health effects are summarized in Figure 5.7 as a function of dose.

5.1.8 Nickel

Absorption

Absorption studies in humans report that 27 percent of inorganic nickel (administered as nickel sulfate) was absorbed when it was administered in drinking water, whereas only 0.7 percent was absorbed when it was given in food. In a separate study, the bioavailability of nickel, measured by serum nickel levels, increased by 80 μg/L after 3 hours in fasted individuals given nickel sulfate in drinking water; it was not elevated in individuals given nickel in food (DHHS, 1993). Other human studies report that generally less than 10 percent of ingested nickel is absorbed by the gastrointestinal tract. This finding is consistent with studies reporting between 1 and 10 percent oral absorption in several animal species (Friberg et al., 1986). Absorbed nickel is transported in the plasma bound to serum albumin and various organic ligands, amino acids, or polypeptides (Casarett and Doull, 1991).

Tissue accumulation and clearance

In humans, serum nickel levels reportedly peaked 2.5 to 3 hours after ingestion of nickel sulfate. In individuals who accidentally drank water contaminated with nickel sulfate and nickel chloride, the mean serum half-time of nickel was 60 hours. No human data were located regarding levels of nickel in specific tissues or organs following nickel compound ingestion.

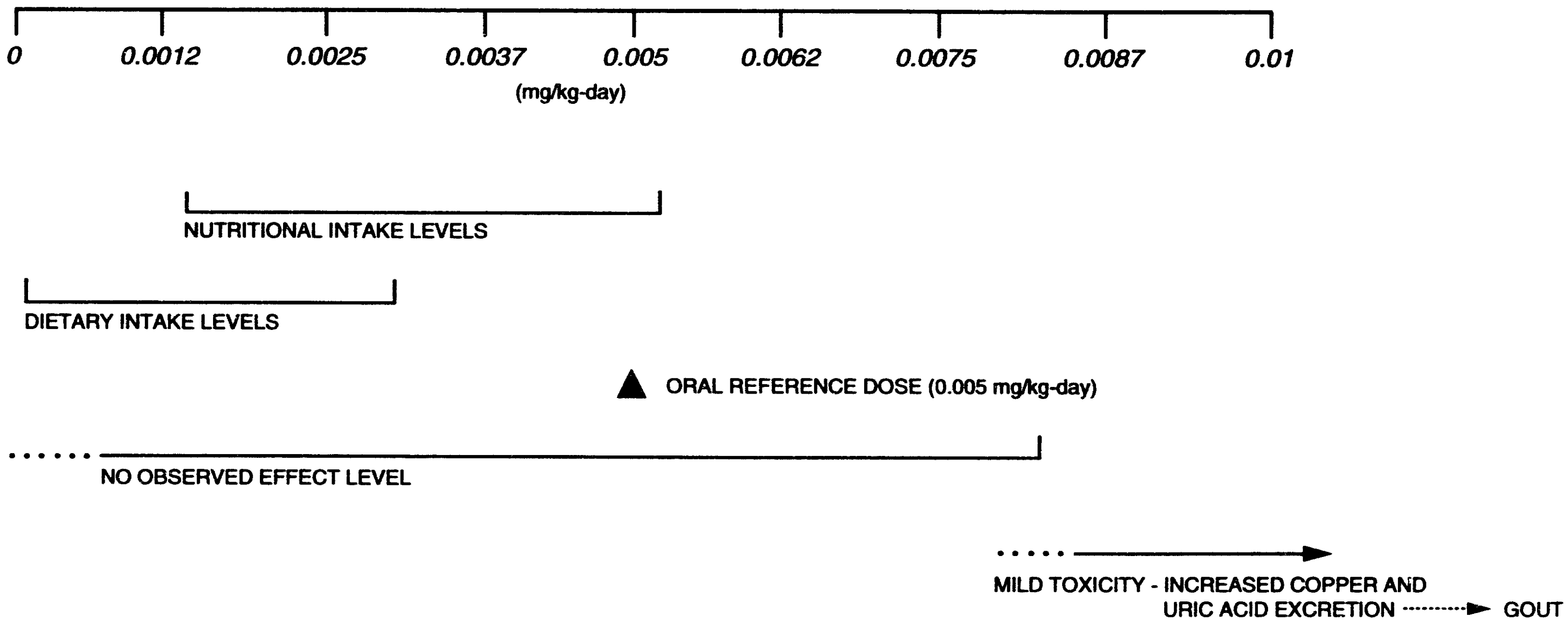


FIGURE 5.7
MOLYBDENUM TOXICITY RANGES

In animals, nickel administered orally (various nickel compounds) distributed primarily to the kidneys. Significant levels were also found in the liver, heart, lung, fat, peripheral nervous tissue, and brain. Nickel also was found at increased levels in the fetuses of animals exposed orally to nickel compounds, suggesting that nickel crosses the placental barrier (DHHS, 1993).

In humans, most ingested nickel is excreted in the feces, due to limited absorption. Nickel absorbed in the gastrointestinal tract is excreted in the urine. Excretion of a given dose of nickel is nearly complete in 4 or 5 days (Casarett and Doull, 1991), with approximately 26 percent of the dose excreted in the urine and the remainder eliminated in the feces (DHHS, 1993).

Environmental sources of nickel

Exposure to nickel can occur through inhalation of ambient air and tobacco smoke and ingestion of water and food. Most intake occurs through the diet (DHHS, 1993). In grains, fresh-weight nickel concentrations reportedly range from 0 to 6.45 micrograms per gram (μg/gram). In vegetables and fruits, levels range from 0 to 2.59 μg/gram, and in seafood from 0.3 to 107 μg/gram. Average daily dietary intake is approximately 165 μg (Friberg et al., 1986).

Nickel is not commonly present at harmful levels in ground water. A survey of ground water in the United States showed that 97 percent of all samples (total of 2053 samples) contained less than 20 μg/L and 80 percent contained less than 10 μg/L. In areas near nickel mining operations, levels as high as 200 μg/L have been reported (Friberg et al., 1986).

Toxicity of nickel

Acute exposure to high levels of nickel in drinking water (1-day duration) has reportedly produced symptoms of gastrointestinal distress, including nausea, abdominal cramps, diarrhea, and vomiting. The estimated exposure dose of between 7.1 and 35.7 mg/kg also produced transient hematological effects, muscular pain, transient increases in urine albumin, and neurological effects (giddiness and weariness). These health effects are summarized in Figure 5.8 as a function of dose.

The effects of chronic ingestion of nickel in humans have not been well documented. In laboratory animals (dogs and rats), the primary effects reported following long-term dietary administration of nickel sulfate were decreases in body weight and changes in organ weight. Low hematocrit and polyuria were reported in dogs (DHHS, 1993).

In rats, a NOAEL of 100-parts-per-million (ppm) diet (5 mg/kg-day) was reported. A human chronic RfD was derived from this NOAEL. Considering the uncertainties with interspecies extrapolation and protection of sensitive populations, an oral RfD of 0.02 mg/kg-day has been developed for nickel

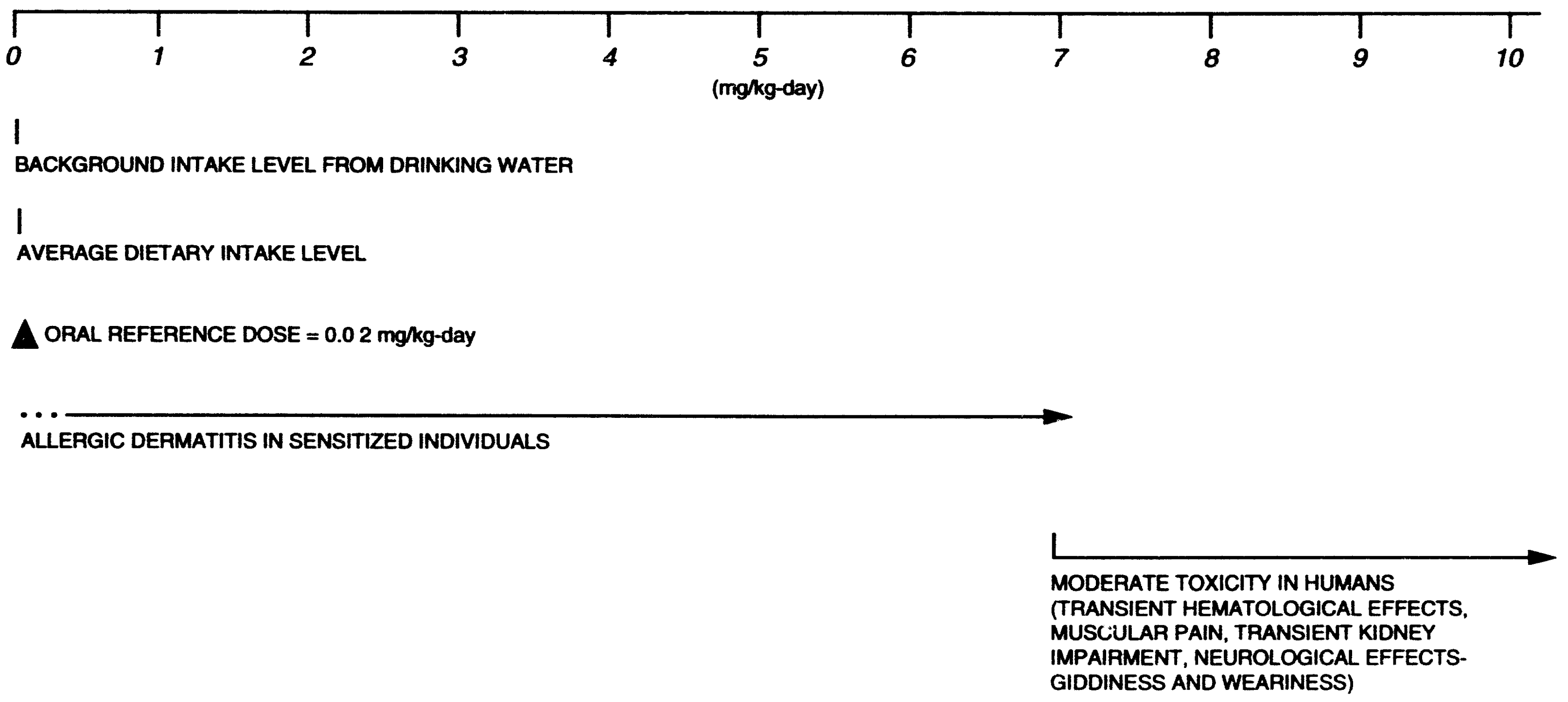


FIGURE 5.8
NICKEL TOXICITY RANGES

(EPA, 1994). This value represents a chronic daily ingestion dose that would not be expected to produce adverse health effects in humans.

5.1.9 Sulfate

Absorption

Sulfate absorption from the gastrointestinal tract is similar between humans and other animals. Generally, greater than 90 percent absorption has been reported for doses of sulfate below 150 mg/kg, decreasing to 50 to 75 percent as the dose increases into the grams per kilogram range.

Tissue accumulation and retention

Ingestion of high levels of sulfate results in transient increases in both blood and urine concentrations. For sulfate doses of approximately 75 mg/kg, approximately 50 percent of the dose is excreted over 72 hours. The urinary excretion mechanism is transport-limited and can therefore become saturated at high doses of sulfate. Excess sulfate is also excreted in feces in its inorganic form. To date, no data are available that indicate sulfate is accumulated, even with chronic ingestion of above-normal levels. However, extremely high chronic doses do not appear to have been examined in humans.

Sulfate is used in the biosynthesis of collagen, cartilage, and dentin and in the formation of sulfate esters of both endogenous compounds (such as lipids and steroids) and exogenous compounds (such as phenols). Sulfation is important in detoxication pathways because it increases the solubility of these compounds, which enhances their excretion in the urine. Exposure to high concentrations of compounds that are conjugated with sulfate and excreted can produce a transient decrease in sulfate concentrations in plasma.

Environmental sources of sulfate

Drinking water in the western United States in 1978 showed a range of sulfate concentrations from 0 to 820 mg/L, with a mean concentration of 99 mg/L of sulfates. The EPA estimates a normal sulfate intake range of 0.00023 to 0.0064 mg/kg-day from air and 0.000 to 2.9 mg/kg-day from drinking water in the concentration range found in supplies in the western United States. No estimates are available on intake of sulfates from food sources.

Toxicity of sulfate

As with nitrate toxicity, the acute and chronic effects of sulfate toxicity differ more in severity than in symptoms or mechanisms. Therefore, this discussion will combine acute and chronic toxicity. As mentioned above, there are no data to indicate a bioaccumulation of sulfate with chronic exposure. Sulfate salts of magnesium and sodium are used medicinally as cathartics. The presence of high concentrations of unabsorbed sulfate salts in the gut can pull large amounts of water into the gut, greatly increasing the normal volume of feces. This is the basis of the toxic effects as well.

Toxicity in humans is primarily manifested in diarrhea; the severity of the diarrhea is dose-dependent. Chronic ingestion of sulfate can result in persistent diarrhea, leading to ionic imbalances and dehydration similar to that seen with extremely high acute doses. In the case of drinking water contaminated with sulfate, the taste of the water may make it unpalatable and reduce consumption. However, this is not necessarily the case. In regions such as Saskatchewan with high sulfate concentrations in the drinking water, residents adapt to the taste and find the water palatable (EPA, 1992b). In cases where consumption is reduced, a lower water intake could compound the dehydration effects of the diarrhea. Extreme dehydration can lead to death. As with nitrate toxicity, infants seem to be the most susceptible population for sulfate-induced diarrhea. Also, there are data to indicate diabetic and elderly populations with compromised kidney function may be more sensitive than healthy adults to the effects of sulfates (EPA, 1992b).

These health effects are summarized in Figure 5.9 as a function of dose.

5.1.10 Uranium

Naturally occurring uranium, present at UMTRA Project sites, consists of three radioactive isotopes: uranium-234, uranium-235, and uranium-238. More than 99 percent of natural uranium occurs in the form of uranium-238 (Cothern and Lappenbusch, 1983). Uranium-238 undergoes radioactive decay by emitting alpha particles to form uranium-234, thorium-230, radium-226, radon 222, polonium-210, and other radioisotopes. The radioactive decay chain of uranium-238 and uranium-234 is summarized in Figure 5.10. As all uranium isotopes in nature are radioactive, the hazards of a high uranium intake are from both its chemical toxicity and potential radiological damage. This section focuses on the chemical toxicity of natural uranium. Carcinogenic potential associated with exposure to radioactive isotopes of natural uranium is discussed in Section 5.3.

Absorption

Absorption of uranium in the gastrointestinal tract depends on the solubility of the uranium compounds. The hexavalent uranium compounds, especially the uranyl salts, are water soluble, while tetravalent compounds generally are not (Weigel, 1983). Even with soluble compounds, only a small fraction is absorbed. Human gastrointestinal absorption rates of 0.76 to 7.8 percent have been determined (Wrenn et al., 1985).

Tissue accumulation and clearance

In humans exposed to background levels of uranium, the highest concentrations of uranium were found in the bones, muscles, lungs, liver, and kidneys (Fisenne et al., 1988). Uranium retention in bone consists of a short retention half-time

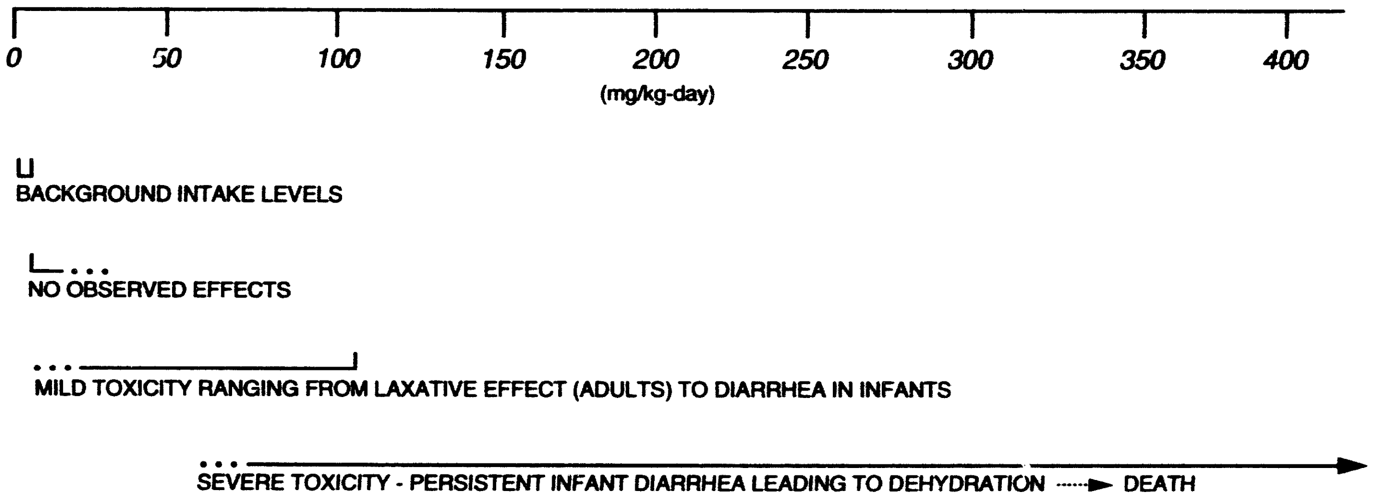


FIGURE 5.9
SULFATE TOXICITY RANGES

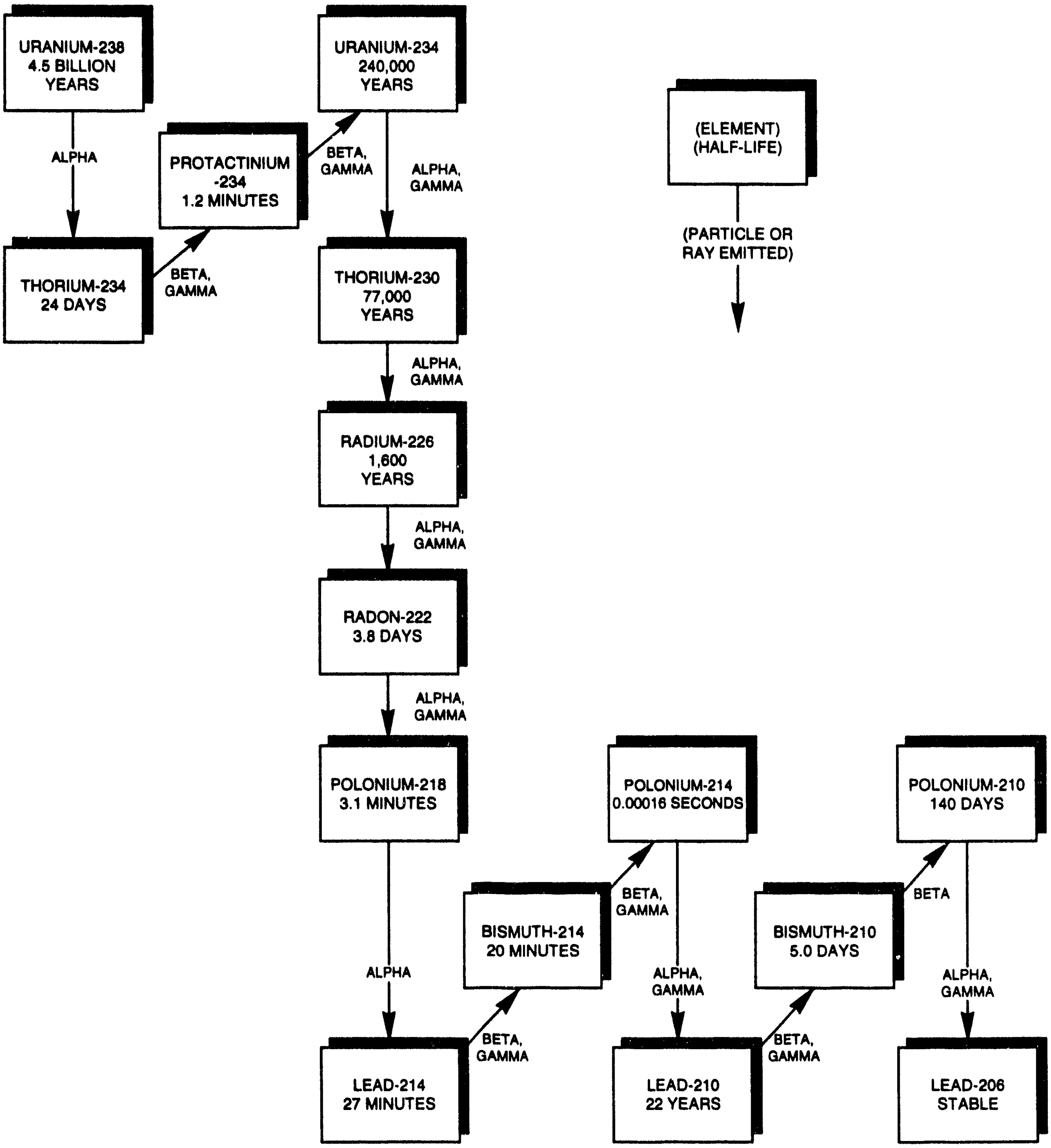


FIGURE 5.10
HALF-LIVES AND EMISSIONS FROM DECAY CHAIN OF URANIUM-238

of 20 days, followed by a long retention half-time of 5000 days for the remainder (Tracy et al., 1992).

In body fluids, uranium tends to be converted into water-soluble hexavalent uranium (Berlin and Rudell, 1986). Approximately 60 percent of the uranium in plasma complexes with low-molecular-weight anions (e.g., bicarbonates, citrates), while the remaining 40 percent binds to the plasma protein transferrin (Stevens et al., 1980). Following oral exposure in humans, more than 90 percent of uranium is excreted in the feces and not absorbed in the gastrointestinal tract. Of the small percent that is absorbed (typically less than 5 percent), approximately 60 percent is excreted in the urine within 24 hours and 98 percent is excreted within 7 days, based on animal studies (Ballou et al., 1986; Leach et al., 1984; Sullivan et al., 1986). A small portion of the absorbed uranium is retained for a longer period.

Environmental sources of uranium

Uranium is a ubiquitous element, present in the earth's crust at approximately 4 ppm. Uranium concentrations in ground water and surface water averaged 1 pCi/L and 3 pCi/L, respectively (NCRP, 1984). It is absorbed from the soil into plant tissues to an extent that depends on the plant species and the depth of its root system (Berlin and Rudell, 1986). Plant concentrations of uranium averaged 0.075 μg/kg of fresh plant material (Tracy et al., 1983).

The main dietary source of natural uranium for the general population is food products such as potatoes, bakery products, meat, and fresh fish, which may contain uranium concentrations between 10 and 100 μg/kg (Prister, 1969). The total dietary intake of uranium from the consumption of average foods is approximately 1 μg/day, additionally approximately 20 to 50 percent of that total can come from drinking water. Cereals and vegetables, particularly root crops, are likely to contribute most to the daily intake of uranium (Berlin and Rudell, 1986).

Toxicity of uranium

Exposure of the general public to natural uranium is unlikely to pose an immediate lethal threat to humans. No human deaths have been reported that are definitely attributable to uranium ingestion, therefore, no lethal dose has been determined for humans. In laboratory animals, lethal doses of uranium ($LD_{50,23}$) have been reported to be as low as 14 mg/kg-day following 23 day oral exposures, depending on the solubility of the uranium compound tested (higher solubility compounds have greater toxicity), route of exposure, and animal species. High doses of uranium cause complete kidney and respiratory failure.

No chronic toxic effects have been reported in humans following oral exposure to uranium. Data available from populations occupationally exposed to high concentrations of uranium compounds through inhalation and information from studies in experimental animals indicate that the critical organ for chronic uranium toxicity is the proximal tubule of the kidney (Friberg et al., 1986). In humans, chemical injury reveals itself by increased catalase excretion in urine and proteinuria. Dose-response data for the toxic effect of uranium on the human kidney are limited.

The lowest dose of uranyl nitrate that caused moderate renal damage was given to rabbits in diet at 2.8 mg/kg-day (Maynard and Hodge, 1949).

The health effects for uranium are summarized in Figure 5.11 as a function of dose.

5.1.11 Vanadium

Absorption

Absorption of vanadium from the gastrointestinal tract is low. In a healthy young man, orally administered sodium metavanadate (12.5 mg/day for 12 days) was completely recovered: The majority (87.6 percent) of the dose was unabsorbed in feces, and the remainder (12.4 percent) was excreted in urine (Proescher et al., 1917). Essentially the same result was obtained by Tipton et al. (1969) in a dietary balance study (50 weeks, two subjects). The urine-to-diet ratio for vanadium was approximately 0.13; the same value was obtained for the urine-to-excreta ratio.

Less than 0.1 percent of an intragastric dose was detectable in the blood of rats at 15 minutes postexposure, and less than 1 percent at 1 hour postexposure (Roshchin, 1968). Uptake of radioactive V_2O_5 given orally to rats was 2.6 percent of the administered dose. The ICRP (1960) estimate for the absorption of soluble vanadium compounds is 2 percent. Soluble vanadium compounds that are inhaled and deposited are readily absorbed. Because vanadium is a metal of low solubility, absorption through the skin is probably minimal (EPA, 1977).

Tissue accumulation and clearance

Vanadium is found in all body tissues in concentrations ranging from 0.08 μg/g wet weight in spleen tissue to 0.14 μg/g in brain and heart tissue and 0.33 μg/g in aorta tissue (Yakawa and Suzuki-Yasumoto, 1980). Concentrations of vanadium in human blood serum are reported to be 0.016 to 0.939 nanogram (ng)/mL. In hair, concentrations of vanadium ranging from 20 to 60 ng/gram have been reported by different authors, with higher values found in manic-depressive patients than in normal control groups (57 versus 29 ng/gram).

The distribution of vanadium in humans following oral exposure may be extrapolated from animal studies. In acute-duration exposures, vanadium is rapidly distributed, primarily in the bones. After intermediate-duration exposure, vanadium concentrations reaching the tissues are low, with the kidneys, bones, liver, and lungs initially showing the highest levels.

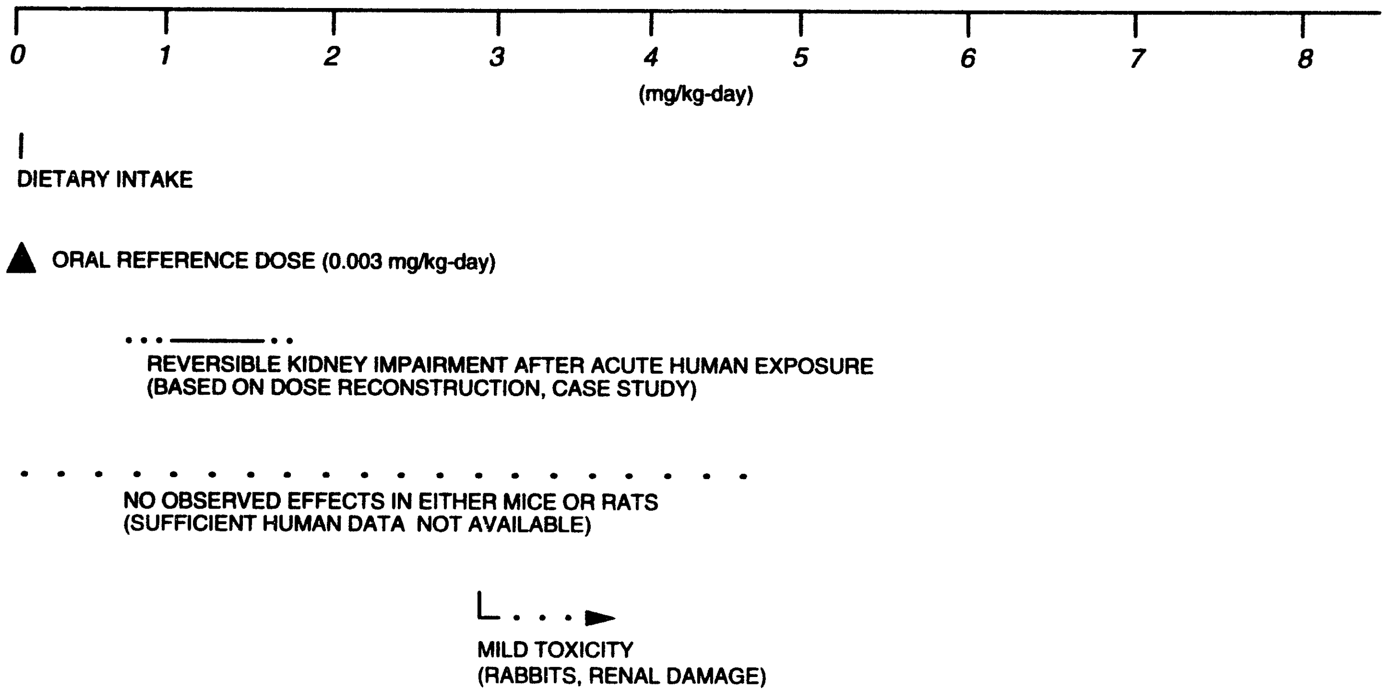


FIGURE 5.11
URANIUM TOXICITY RANGES

Vanadium is an element and is not metabolized. However, in the body, there is an interconversion of two oxidation states of vanadium: vanadyl and vanadate. Vanadium can reversibly bind to the protein transferrin in the blood and then be taken up into erythrocytes. There is a slower uptake of vanadyl into erythrocytes compared to the vanadate form, possibly due to the time required for the vanadyl form to be oxidized to vanadate. Initially, vanadyl leaves the blood more rapidly than vanadate, possibly because of the slower vanadyl uptake into cells (Harris et al., 1984). Five hours after administration, blood clearance is essentially identical for the two forms.

Because vanadium is poorly absorbed in the gastrointestinal tract, a large percentage of vanadium in rats is excreted unabsorbed in the feces following oral exposure. In rats, the principal route of excretion of the small absorbed portion of vanadium is through the kidneys. The mean urinary output per 24 hours is reported to be 10 μg.

Environmental sources of vanadium

Elemental vanadium does not occur in nature, but its compounds exist in more than 50 different mineral ores and in association with fossil fuels. The single largest release of vanadium to the atmosphere occurs through the combustion of fossil fuels, particularly residual fuel oils. The largest amount of vanadium released to soil and water occurs through the natural weathering of geological formations (Byerrum et al., 1974; Van Zinderen Bakker and Jaworski, 1980).

Food constitutes the major source of exposure to vanadium for the general population (Lagerkvist et al., 1986). As a whole, dietary intake is estimated to be 6 to 18 μg/day (Pennington and Jones, 1987), although other estimates from older studies using different and possibly less sensitive analytical methods have been as high as 2 mg/day (Schroeder et al., 1963).

Drinking water is not considered an important source of vanadium exposure for the general population. Water samples taken from across the United States show 92 percent with values below 10 μg/L. Typical values appear to be around 1 μg/L (Lagerkvist et al., 1986). The estimated daily intake of vanadium by inhalation is 1 μg (Byrne and Kosta, 1978).

Although vanadium is considered an essential element for chickens and rats, human dietary requirements remain uncertain. For animals, the daily requirement is about 10 to 25 μg/day (Pennington and Jones, 1987).

Toxicity of vanadium

The major adverse health effect to humans from vanadium is seen in workers exposed to large amounts of vanadium pentoxide dusts. The probable oral lethal dose of vanadium pentoxide for humans is between 5 and 50 mg/kg (Gosselin et al., 1976).

Systemic effects of vanadium exposure have been observed in the liver, kidneys, nervous and cardiovascular systems, and blood-forming organs. Metabolic effects include interference with the biosynthesis of cystine and cholesterol, depression and stimulation of phospholipid synthesis and, at higher concentrations, inhibition of serotonin oxidation. Other effects of vanadium on mammalian metabolism include depression of phospholipid synthesis (Snyder and Cornatzer, 1958), reduction of coenzyme Q levels in mitochondria (Aiyar and Sreenivasan, 1961), and stimulation of monoamine oxidase, which oxidizes serotonin (Perry et al., 1955).

Vanadium salts were given to patients in several studies to reduce cholesterol (Curran et al., 1959; Somerville and Davies, 1962; Dimond et al., 1963; Schroeder et al., 1963). The doses of vanadium in these studies varied from 7 mg/day to 30 mg/day. Transient decreases in serum cholesterol levels were observed in some patients, as were loosened stool and cramps. Green tongue, a hallmark of vanadium exposure, was observed in all patients.

A relationship between the concentration of vanadium in drinking water and the incidence of dental caries in children is reported by Tank and Storvick (1960). Dental caries incidence in children aged 7 to 11 years was reduced three times (compared to controls) by applying ammonium vanadate in glycerol to the teeth (Belehova, 1969). This relationship was not found in other studies (Hadjimarkos, 1966; 1968).

It has been suggested that raised tissue levels of vanadium are important in the etiology of manic-depressive illness. Improvement after treatment with ascorbic acid or reduced vanadium intake was seen both in manic and depressed patients.

The toxicity of vanadium is summarized in Figure 5.12.

5.1.12 Zinc

Absorption

The absorption of zinc from the gastrointestinal tract is highly variable. Reported absorption ranges from less than 10 percent to over 90 percent, depending on body weight, zinc status of the body, interferences from other components of the diet, and metallothionein levels (Elinder, 1986). Absorption is increased when body levels are low or when body weight is low, but dietary calcium can reduce the absorption of zinc. Because there is homeostatic control over zinc absorption, the percent absorbed from a given dose will be lower with very high doses.

Tissue accumulation and clearance

The majority of zinc in the body is in muscle (60 percent) and bone (30 percent), but the highest concentrations per gram of tissue are found in

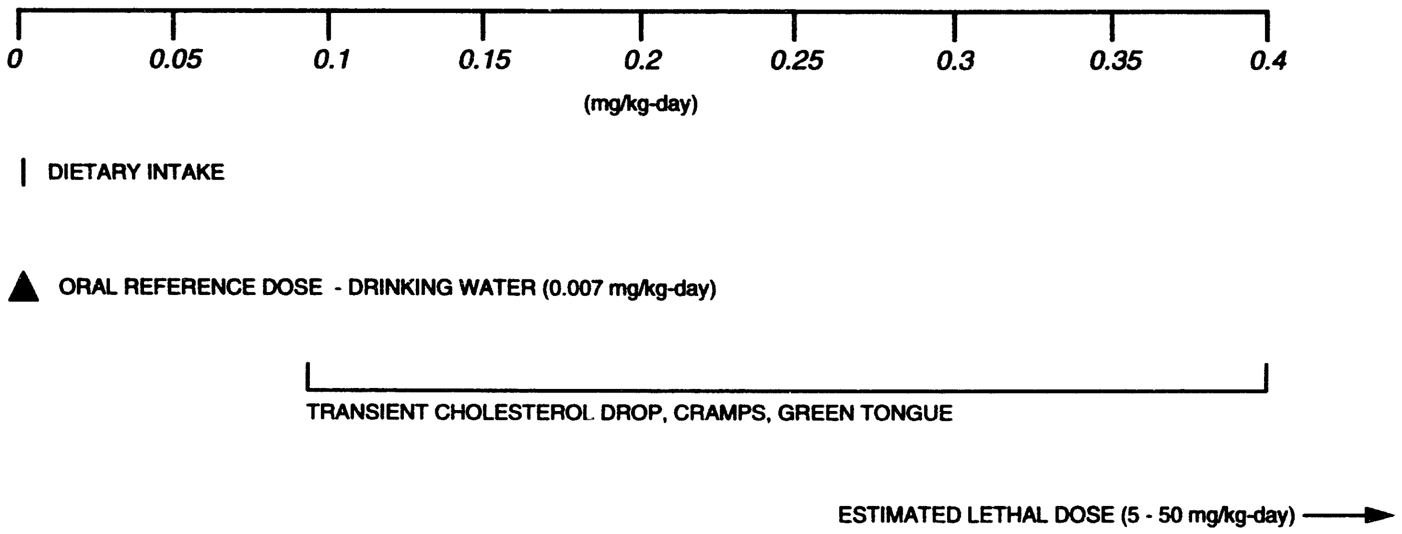


FIGURE 5.12
VANADIUM TOXICITY RANGES

MAC: SITE/GRJ/BRA/VAN-GEN

prostate, then bone, then muscle (Elinder, 1986). Zinc is an essential regulatory element for many enzymes and therefore can be found in all tissues as well as serum and plasma.

The primary route of excretion of zinc is in the feces (75 percent), with the remainder in urine. About 1 percent of an absorbed oral dose is eliminated per day. Because zinc levels in the body appear to be under homeostatic control, the rate of elimination increases if dietary intake is increased.

Because both absorption and excretion are highly variable, the biological half-time for zinc can range from 100 to 500 days in humans (Elinder, 1986).

Environmental sources of zinc

The concentration of zinc in drinking water generally ranges from 1 to 10 μg/L, but levels as high as 2 mg/L can result if galvanized supply pipes are used (Elinder, 1986). The low concentration normally found in drinking water provides only minimal contribution to the average daily intake.

Zinc is also found in protein-rich food such as meat and fish. Milk contains about 3 mg/L of zinc (Elinder, 1986). Dietary intake ranges from 5 to 15 mg/day (0.07 to 0.26 mg/kg-day) (EPA, 1994).

Concentrations of zinc in soil and water can be significantly elevated in areas near smelter operations.

Toxicity

Zinc is a necessary cofactor in the proper function of at least 20 enzymes responsible for basic metabolic functions, including maintenance of the body's acid-base balance, production of deoxyribonucleic acid and ribonucleic acid and subsequent protein synthesis, and lipid and carbohydrate metabolism. It also plays a role in endocrine function and in the metabolism of foreign substances such as alcohol. Zinc deficiency, therefore, can interfere with proper function of many systems. Strong homeostatic control of absorption and excretion helps keep zinc at adequate levels in the body.

Although toxic effects of excessive zinc intake can occur in both men and women, the effects are gender-specific. In women, excess zinc intake primarily decreases copper absorption. This effect can be observed with zinc intake as low as 0.26 mg/kg-day, but copper and iron deficiency can be observed in women with intakes of zinc in the range of 1 mg/kg-day. Intakes of more than 2.5 mg/kg-day can result in copper deficiency anemia. For men, the primary effect of increased zinc intake is a transitory increase in high-density lipoproteins (HDL) followed after a couple of weeks by a decrease in HDL. With zinc intakes in the range of 4.4 mg/kg-day, the decrease in HDL is accompanied by an increase in low-density lipoproteins. This effect is observed in men with intakes of more than 0.7 mg/kg-day. The decrease in HDL seen with zinc intakes of

2.5 mg/kg-day can be severe enough to be associated with cardiovascular risk. Women are reported to show only transitory decreases in HDL, and not until zinc intake exceeds 1.8 mg/kg-day. Decreases in immune function have been observed with zinc intakes of more than 4 mg/kg-day (EPA, 1994).

The toxic effects and doses for zinc are summarized in Figure 5.13.

5.2 CONTAMINANT INTERACTIONS

Some information is available on potential interactions between contaminants found at UMTRA Project sites. However, discussions of potential interactions can generally be presented only qualitatively. In addition to physiological variables among individuals that can affect toxicity, uncertainties in interactions also result from 1) differences in the relative exposure concentrations of the different contaminants compared to the concentrations tested experimentally, and 2) the presence of additional ground water constituents that may occur in sufficient quantities to modify predicted toxicities, even though they themselves are not considered contaminants of concern for human health. Therefore, the interactions described below should be recognized as factors that can influence the predicted toxicity, although the precise nature and magnitude of that influence cannot be determined.

A primary concern at the Grand Junction site is the potential for interactions between metals. Interactions between several similar metals can alter the predicted absorption, distribution in the body, metabolism, toxicity, or clearance of a metal of interest.

For example, the absorption of cadmium, manganese, and zinc can be considerably altered under conditions of high calcium and iron or low protein (Elinder, 1986; Nordberg et al., 1985). Absorption of cadmium, manganese, and zinc from the intestine may significantly decrease in the presence of high dietary iron, leading to decreased toxicity of cadmium, manganese, and zinc (DHHS, 1989; 1992; Flanagan et al., 1978). High levels of cadmium may inhibit manganese uptake. Conversely, high levels of manganese lead to decreased iron absorption. Cadmium and zinc would decrease the bioavailability of calcium. Calcium salts decrease the absorption of fluoride from the gastrointestinal tract (National Research Council, 1980). Additionally, cadmium, manganese, and zinc can induce synthesis of the metal-binding protein metallothionein (DHHS, 1989, 1992; Casarett and Doull, 1991). This protein seems to have a paradoxical effect on the systemic toxicity of cadmium. Metallothionein appears to bind cadmium and in this way protect certain organs, such as the testes, from cadmium toxicity. But, at the same time, metallothionein may enhance cadmium nephrotoxicity, possibly because the cadmium-metallothionein complex is taken up by the kidney more readily than is the free ion. However, because both cadmium and zinc bind strongly to metallothionein, in the continued presence of both zinc and cadmium, there may be competition for metallothionein-binding sites.

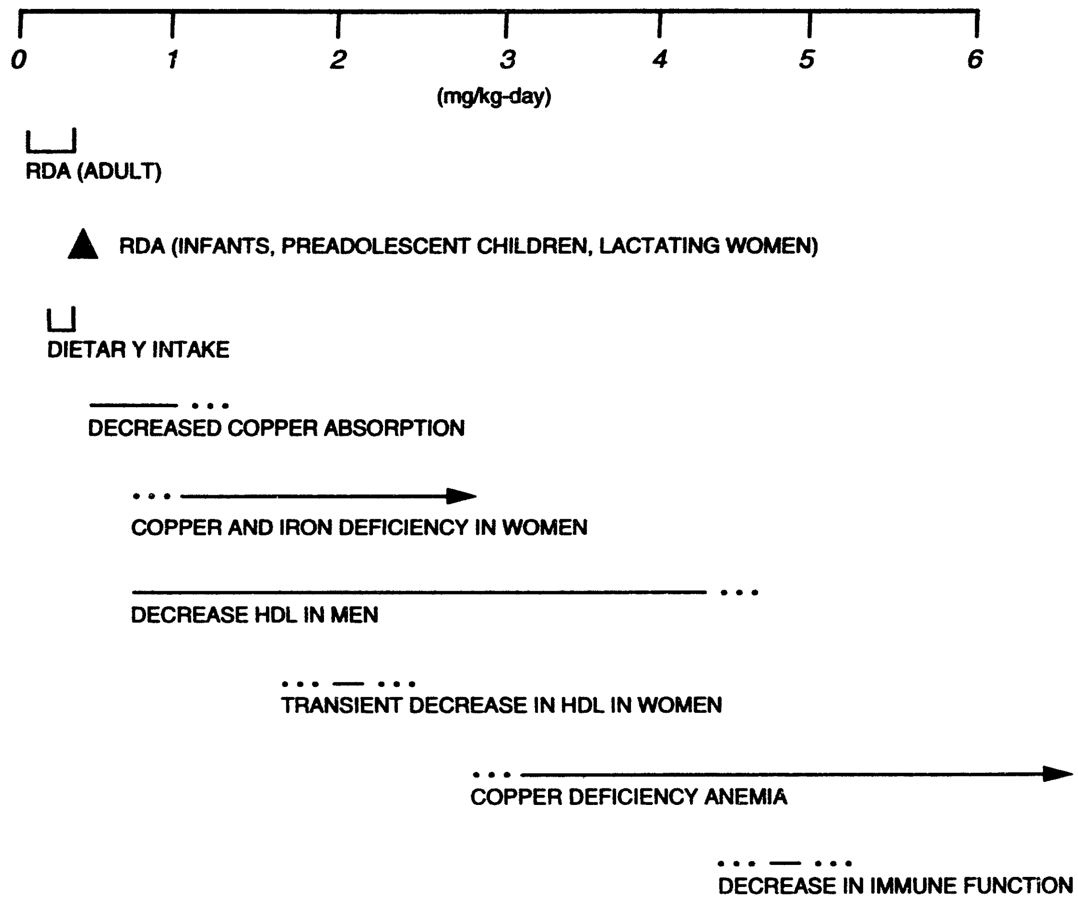


FIGURE 5.13
ZINC TOXICITY RANGES

Physiological interactions of cadmium and zinc may occur (DHHS, 1989). Exposure to cadmium may change the distribution of zinc, with accumulation of zinc in the liver and kidney. This accumulation may result in a deficiency in other organs. On the other hand, zinc can increase absorption of cadmium from the intestine (DHHS, 1993a).

The combinations of vanadium with manganese and vanadium with nickel administered to pregnant mice caused some alterations in behavioral development of the pups as compared to either element administered alone (DHHS, 1992). Oral administration of vanadium may interfere with copper metabolism by inhibiting the intestinal absorption of copper.

Nickel may interact with other heavy metals such as iron, manganese, zinc, and cadmium (DHHS, 1993b). The toxicity of nickel can be mitigated by high levels of zinc. Conversely, high levels of cadmium can enhance the nephrotoxicity and hepatotoxicity of nickel. The cadmium-damaged renal cells may be more susceptible to nickel, or cells not damaged by cadmium may be damaged by nickel; however, the mechanism of this interaction could not be determined. Under conditions of high iron levels, nickel may inhibit the passive diffusion of iron, decreasing its gastrointestinal absorption, but only when the iron is present in the form of ferric ion (DHHS, 1993b). However, based on geochemical models for the Grand Junction site, iron exists in the divalent form in ground water. Therefore, nickel should not inhibit absorption of iron at this site. An interrelationship exists between nickel and cobalt sensitization in individuals exposed to the two metals (DHHS, 1992; 1993c). The combination of nickel sensitivity and irritant eczema may result in a high risk for developing an allergy to cobalt.

No information on interactions of uranium with other metals has been found. However, the common target organ suggests interaction with arsenic, cadmium, and nickel in the production of kidney toxicity.

It should also be kept in mind that diarrhea-caused dehydration may lead to excessive concentration of the contaminants (those excreted in urine) in the kidney; thus, it may enhance the predicted toxicity resulting from exposure to these contaminants.

5.3 CONTAMINANT RISK FACTORS

The EPA Office of Research and Development has calculated acceptable intake values, or RfDs, for long-term (chronic) exposure to noncarcinogens. These values are estimates of route-specific exposure levels that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifetime. The RfDs include safety factors to account for uncertainties associated with limitations of the toxicological data base, including extrapolating animal studies to humans and accounting for variability in response from sensitive individuals. These values are updated quarterly and published in the Health Effects Assessment Summary Tables (HEAST). They are also provided through the EPA's IRIS data base. The most recent oral RfDs for the noncarcinogenic contaminants of potential concern are summarized in Table 5.2. RfDs for cobalt, iron, and sulfate have not been determined.

With respect to carcinogenic effects, the EPA currently classifies all radionuclides as Group A, or known human carcinogens, based on their property of emitting ionizing radiation and on the evidence provided by epidemiological studies of radiation-induced cancer in humans. At sufficiently high doses, ionizing radiation acts as a complete carcinogen (as both initiator and promoter), capable of increasing the probability of cancer development. However, the actual risk is difficult to estimate, particularly for the low doses and dose rates encountered in the environment. Most of the reliable data were obtained under conditions of high doses delivered acutely. It is not clear whether cancer risks at lower doses are dose proportional (that is, the linear dose-response hypothesis) or whether the risk is greatly reduced at low doses and rates (the threshold hypothesis). A conservative action is that no threshold dose exists below which there is no additional risk of cancer. Risk factors are published in HEAST and IRIS for correlating intake of carcinogens over a lifetime with the increased excess cancer risk from that exposure. The most recent cancer SFs for arsenic and for the uranium-234/-238 radioactive decay series, including radium-226, are given in Table 5.1.

Table 5.2 Toxicity values: potential noncarcinogenic effects

Chemical	Chronic oral RfD (mg/kg-day)	Confidence level	Critical effect/organ	RfD basis/RfD source	Uncertainty factor
Arsenic (inorganic)	3.0E-04	High to medium	Keratosis, hyperpigmentation	Water/HEAST	3
Cadmium (water)	5.0E-04	High	Kidney	Water/HEAST	10
Cobalt	NA[a]	NA	NA	NA	NA
Iron	NA	NA	NA	NA	NA
Fluoride	6.0E-02	High	Fluorosis/tooth	Water/IRIS	1
Manganese (water)	5.0E-03	Medium to low	Central nervous system	Water/IRIS	1
Molybdenum	5.0E-03	High to medium	Bone	Water/HEAST	2
Nickel	2.0E-02	Medium	Decreased weight	Diet/IRIS	1
Sulfate	NA	High	Diarrhea	Water/NA	1
Uranium (soluble salts)	3.0E-03	Medium	Kidney, body weight	Water/IRIS	100-1000
Vanadium	7.0E-03	Low	Hair	Water/HEAST	100
Zinc	3.0E-01	Medium	Anemia	Water/IRIS	3

[a] Not available.

6.0 HUMAN RISK EVALUATION

To evaluate human health risks to an individual or population, the results of the exposure assessment are combined with the results of the toxicity assessment. As discussed in Section 5.0, potential adverse health effects are a function of how much of the contaminant an individual takes into his or her body. Indeed, at lower levels many of the contaminants associated with the mill tailings are beneficial to health, since they are essential nutrients. At higher levels, these same elements can cause adverse health effects or, at very high levels, death. In this section, the expected intake, if ground water within the plume were used as drinking water, is correlated to potential health effects from these levels of exposure.

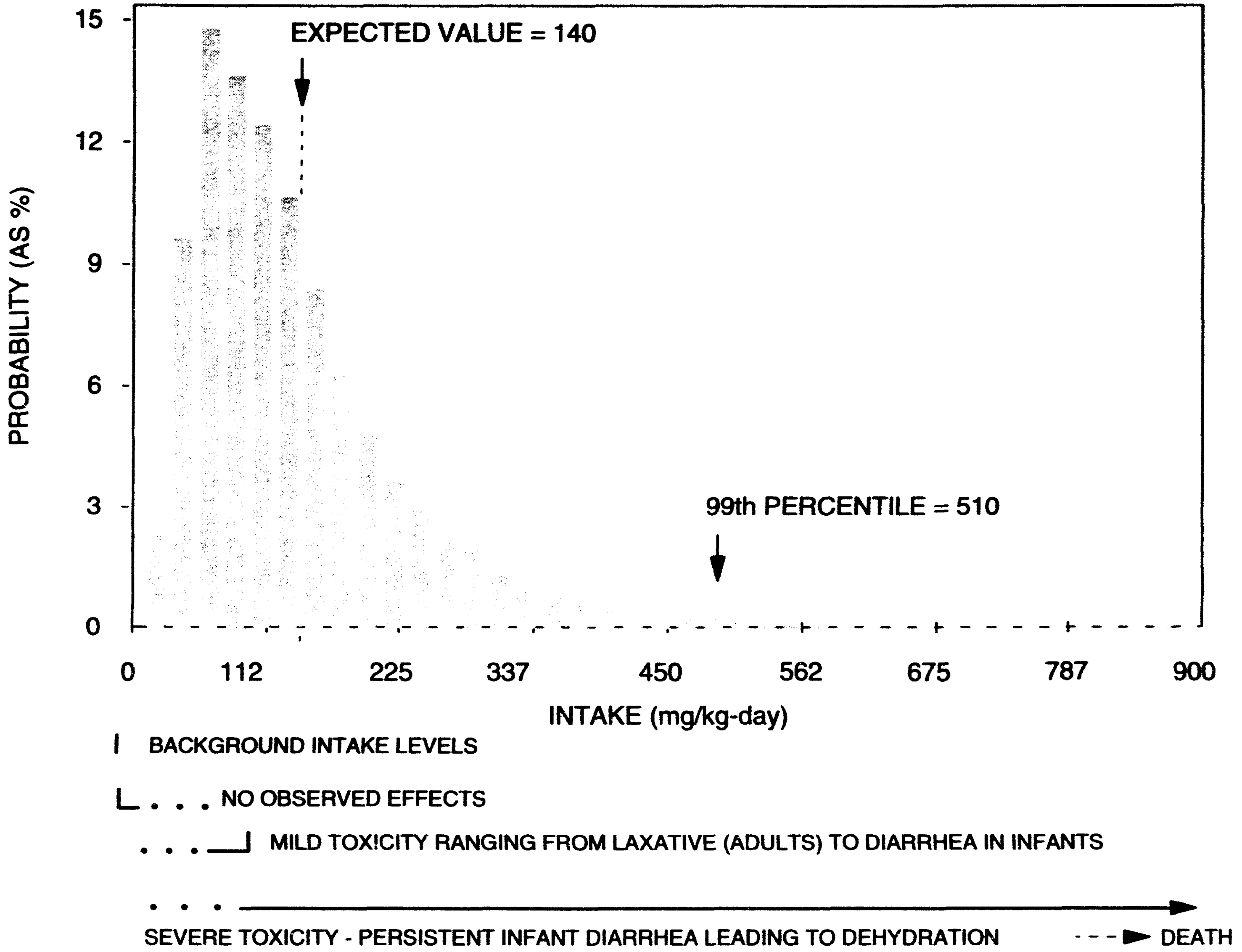
6.1 POTENTIAL NONCARCINOGENIC HEALTH EFFECTS

The results from the exposure assessment for either the age group with the highest intake-to-body-weight ratios (or highest doses) or the toxicologically most sensitive group are used to evaluate potential health effects for noncarcinogens. For the contaminants of potential concern at the Grand Junction site, the highest intake-per-body-weight group is children 1 to 10 years old. Because infants are the most susceptible age group for sulfate toxicity, infants are used to model potential health risks from sulfate ingestion.

The primary concerns for human health from ingestion of contaminated ground water at the Grand Junction site are from exposure to sulfate, manganese, and, to a lesser extent, fluoride, vanadium, cadmium, iron, and arsenic. Diarrhea could be expected to result from nearly all predicted infant exposure to sulfate. The highest 75 percent of predicted exposure would produce in infants severe diarrhea that could result in death from dehydration in the most severe cases (Figure 6.1). As discussed in Section 5.1.9, the toxic effects of sulfate for adults drinking this water would be less, because adults would be exposed to lower concentrations (per kilogram of body weight) and would be less sensitive to the effects.

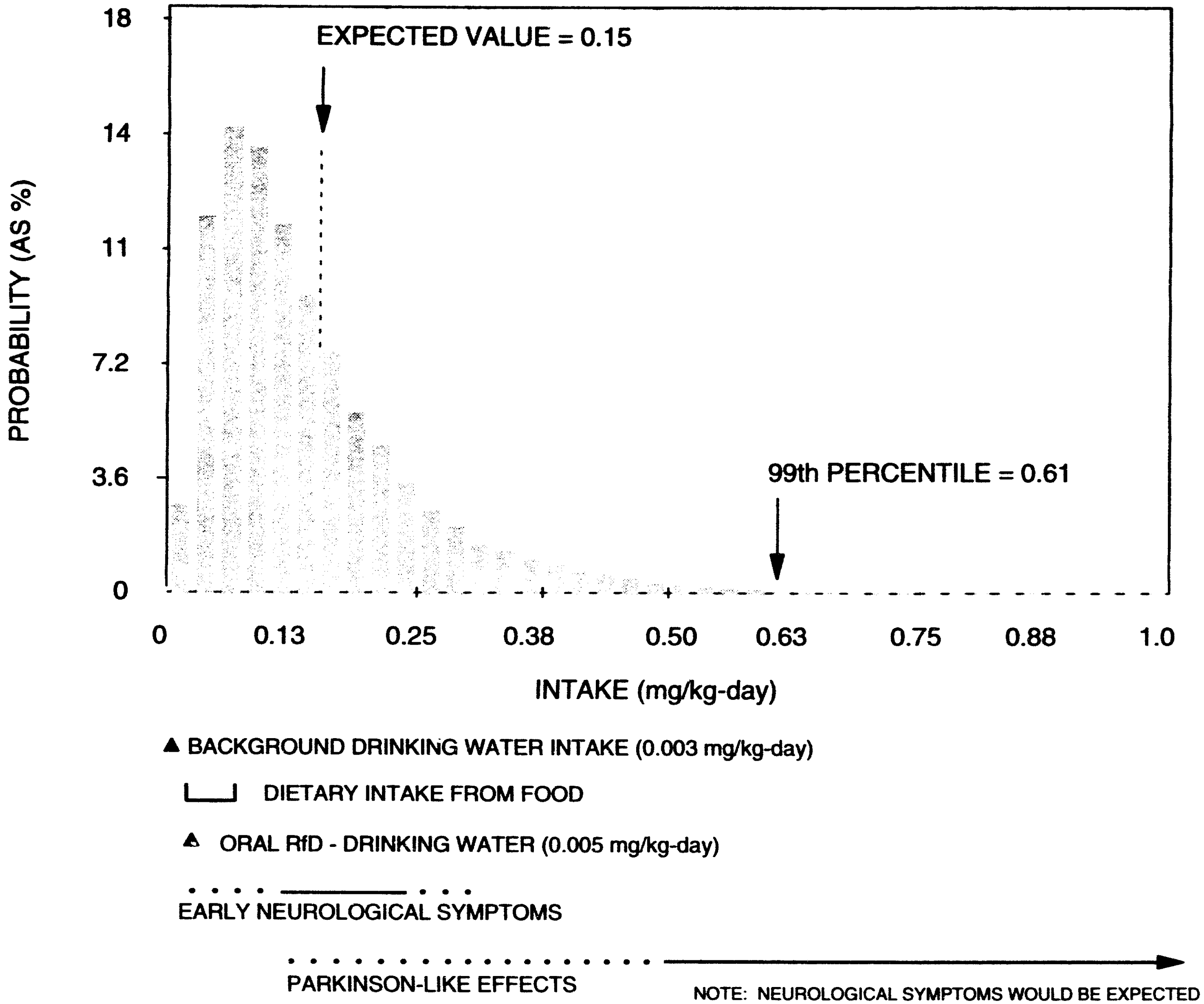
For manganese, nearly all predicted exposures are greater than the oral RfD derived by the EPA. Approximately 60 percent of the predicted exposure range is greater than the levels reported to produce mild neurological symptoms often similar to early symptoms of Parkinson's disease. Less than 3 percent of the distribution falls above levels that have been reported to produce more fully developed Parkinson's-like effects following chronic exposure through drinking water (Figure 6.2).

Approximately 75 percent of the potential range of fluoride exposure from ground water ingestion is above the level reported to result in tooth mottling in children. Although the exposure range simulated for fluoride is only for children, toxicity information is presented on Figure 6.3 for adults as a reference. A small portion (less than 10 percent) of the potential exposure range falls above the reported dose that could result in crippling skeletal fluorosis in adults



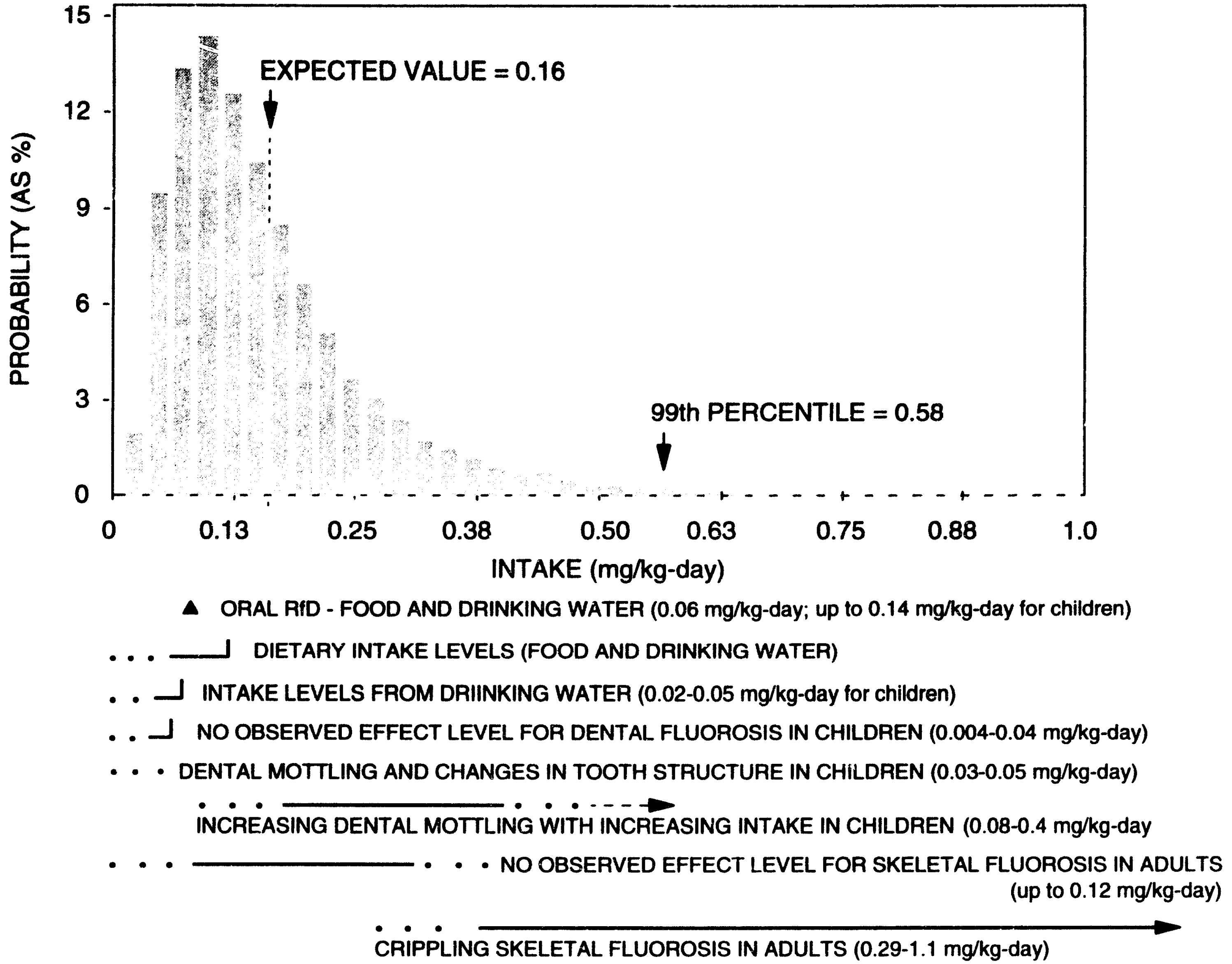
NOTE: SEVERE PERSISTENT DIARRHEA LEADING TO DEHYDRATION WOULD RESULT FROM 75% OF THE PREDICTED INTAKES IN INFANTS.

FIGURE 6.1
HEALTH EFFECTS OF POTENTIAL SULFATE INTAKES FOR INFANTS
GRAND JUNCTION, COLORADO, SITE



NOTE: NEUROLOGICAL SYMPTOMS WOULD BE EXPECTED TO RESULT FROM THE MAJORITY OF MANGANESE INTAKES. THE UPPER 3% OF THE INTAKES ARE IN A RANGE REPORTED TO PRODUCE PARKINSON-LIKE EFFECTS.

FIGURE 6.2
HEALTH EFFECTS OF POTENTIAL MANGANESE INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE



NOTE: PREDICTED INTAKES OF FLUORIDE WOULD PRODUCE MOTTLING OF TOOTH ENAMEL IN CHILDREN. ADULT INTAKES (NOT SHOWN) COULD PRODUCE SKELETAL FLUOROSIS AT THE HIGHER RANGES (LESS THAN 10% OF THE POTENTIAL INTAKES).

FIGURE 6.3
HEALTH EFFECTS OF POTENTIAL FLUORIDE INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE

following long-term ingestion (10 to 20 years) of contaminated water (Figure 6.3).

Mild toxic effects, including transient drops in cholesterol, cramps, and green discoloration of the tongue, have been observed with vanadium intakes in the range of the upper 90 percent of exposures predicted if children drank ground water at this site (Figure 6.4).

The majority (approximately 60 percent) of the potential range of cadmium exposure is within the range at which no adverse effects have been reported. Mild toxicity (proteinuria) has been associated with drinking water exposure encompassing the upper 25 percent of the predicted exposure range (Figure 6.5). The absorption of cadmium, and therefore its toxicity, can increase as much as fourfold in conditions of low iron stress in the body (Section 5.1.2). Because of the high levels of iron in site ground water, this condition is unlikely. However, individuals with congenital impairment of iron absorption may still be more sensitive to cadmium toxicity.

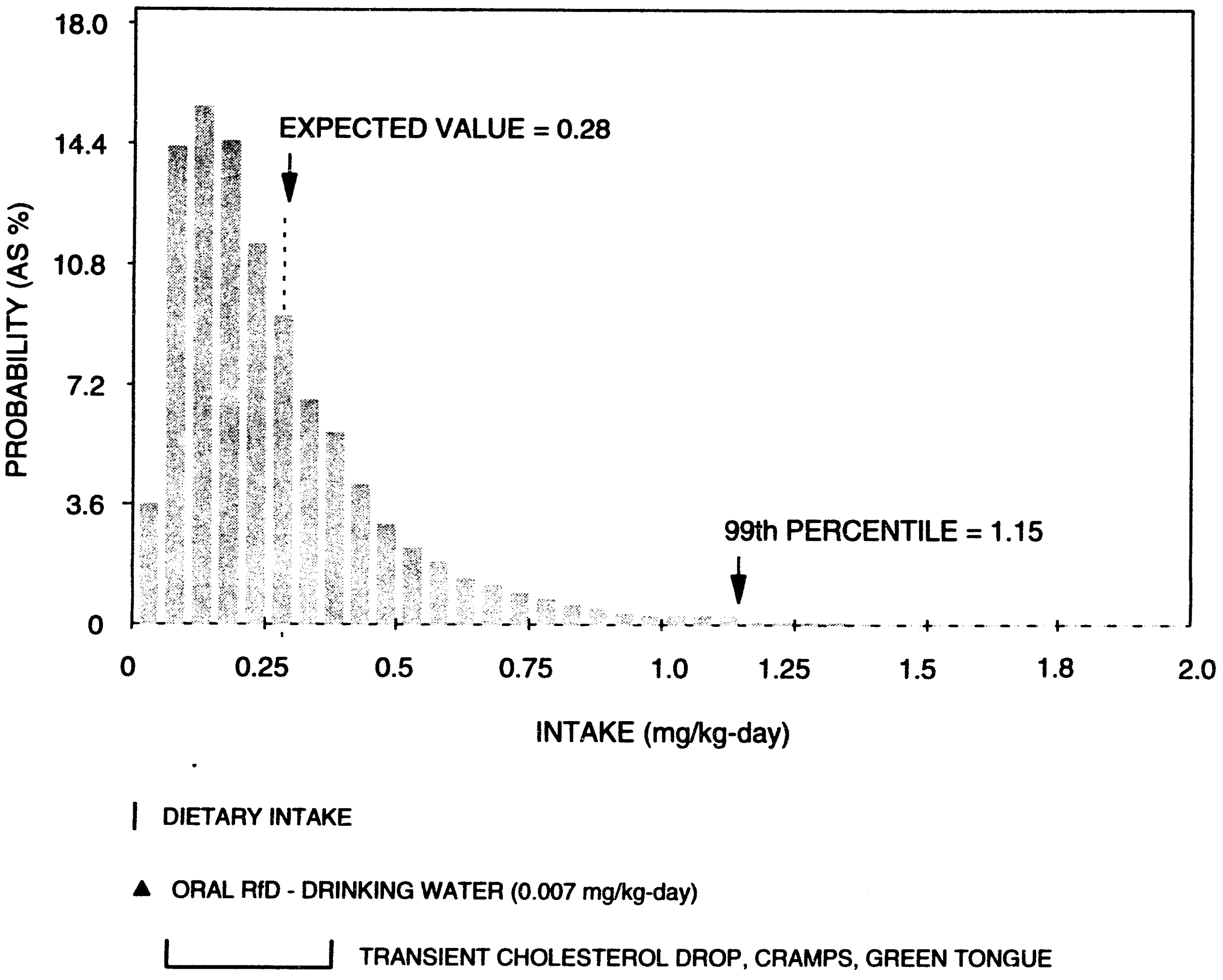
For iron, a large portion (approximately 70 percent) of the potential exposure range is within normal dietary intake levels (Figure 6.6). Less than 15 percent of the potential exposure range is above the dose that produces chronic iron toxicity, including pigmentation of the skin, potential disruption of liver and endocrine function, and, after long-term exposure, cirrhosis of the liver and/or development of diabetes. Based on the geochemical modeling for the Grand Junction site, all of the iron in ground water is expected to be in the divalent form, which is more readily absorbed, and therefore more toxic, than the trivalent form.

For the noncarcinogenic effects of arsenic, nearly all of the potential exposure range for ingestion of contaminated ground water is within the range associated with reported background intake levels (Figure 6.7). However, toxic effects have been observed in the upper range of background levels, and less than 5 percent of the exposure range predicted here has been associated with skin pathology and arterial thickening.

Approximately 50 percent of the potential molybdenum exposure range falls above the daily intake range that would be associated with mild toxicity manifested primarily by mineral imbalances (increased copper excretion) (Figure 6.8).

For zinc, approximately 90 percent of the potential exposure range falls below levels associated with adverse effects. A small portion (less than 5 percent) of the simulated daily intake is above the level associated with decreased copper absorption (Figure 6.9).

Approximately 90 percent of the potential exposure range for nickel is below the oral RfD and would not be expected to result in adverse effects in most people. For individuals sensitized to nickel, roughly 65 percent of the potential exposure

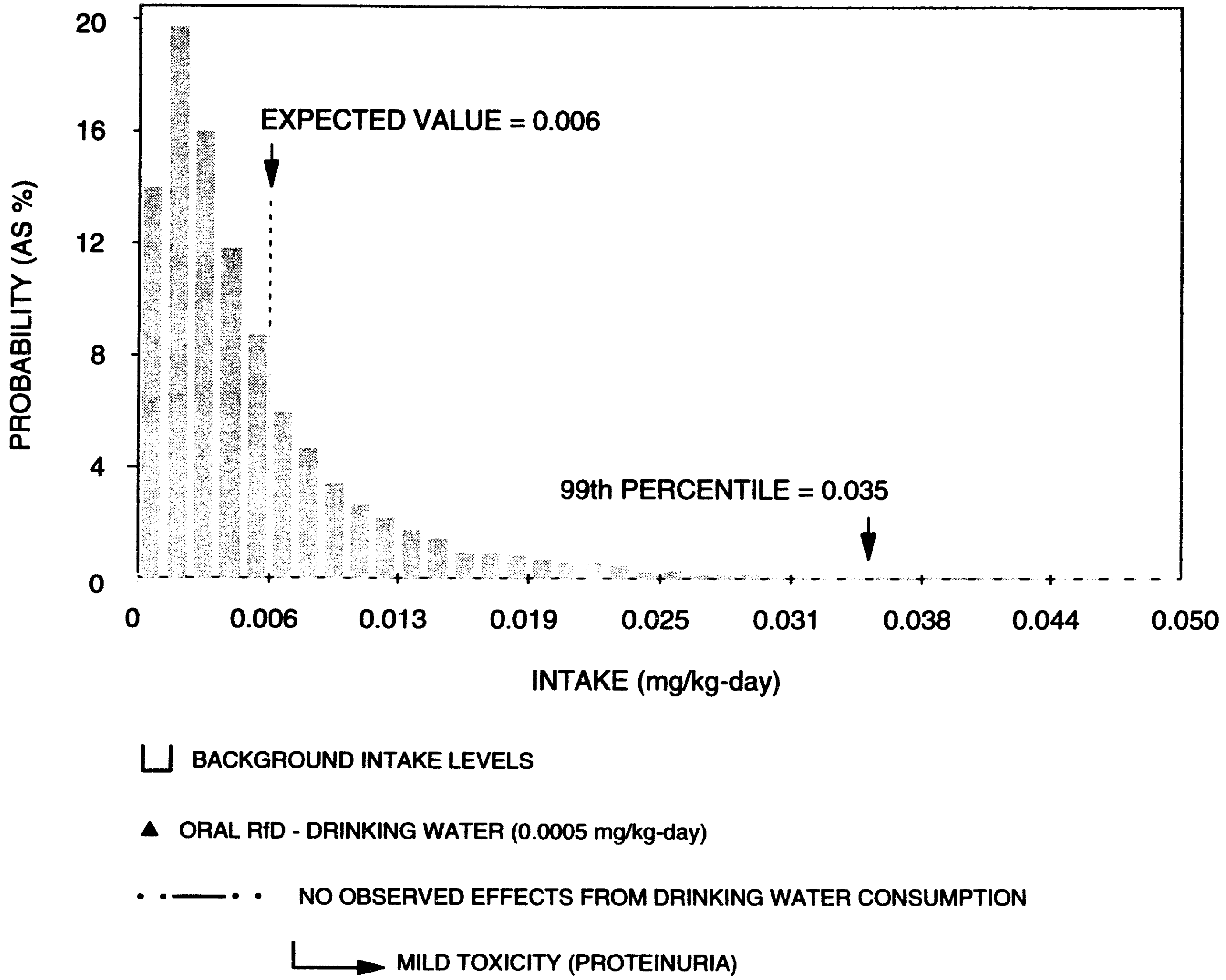


NOTE: THE MAJORITY OF THE POTENTIAL INTAKE IN CHILDREN FALLS ABOVE THE THRESHOLD LEVEL WHERE TOXIC EFFECTS ARE MANIFESTED BY TRANSIENT CHOLESTEROL DROP, CRAMPS, AND GREEN TONGUE.

FIGURE 6.4
HEALTH EFFECTS OF POTENTIAL VANADIUM INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE

PATH I:\FL3.01\GRJBRA

HLTHVANA.DRW

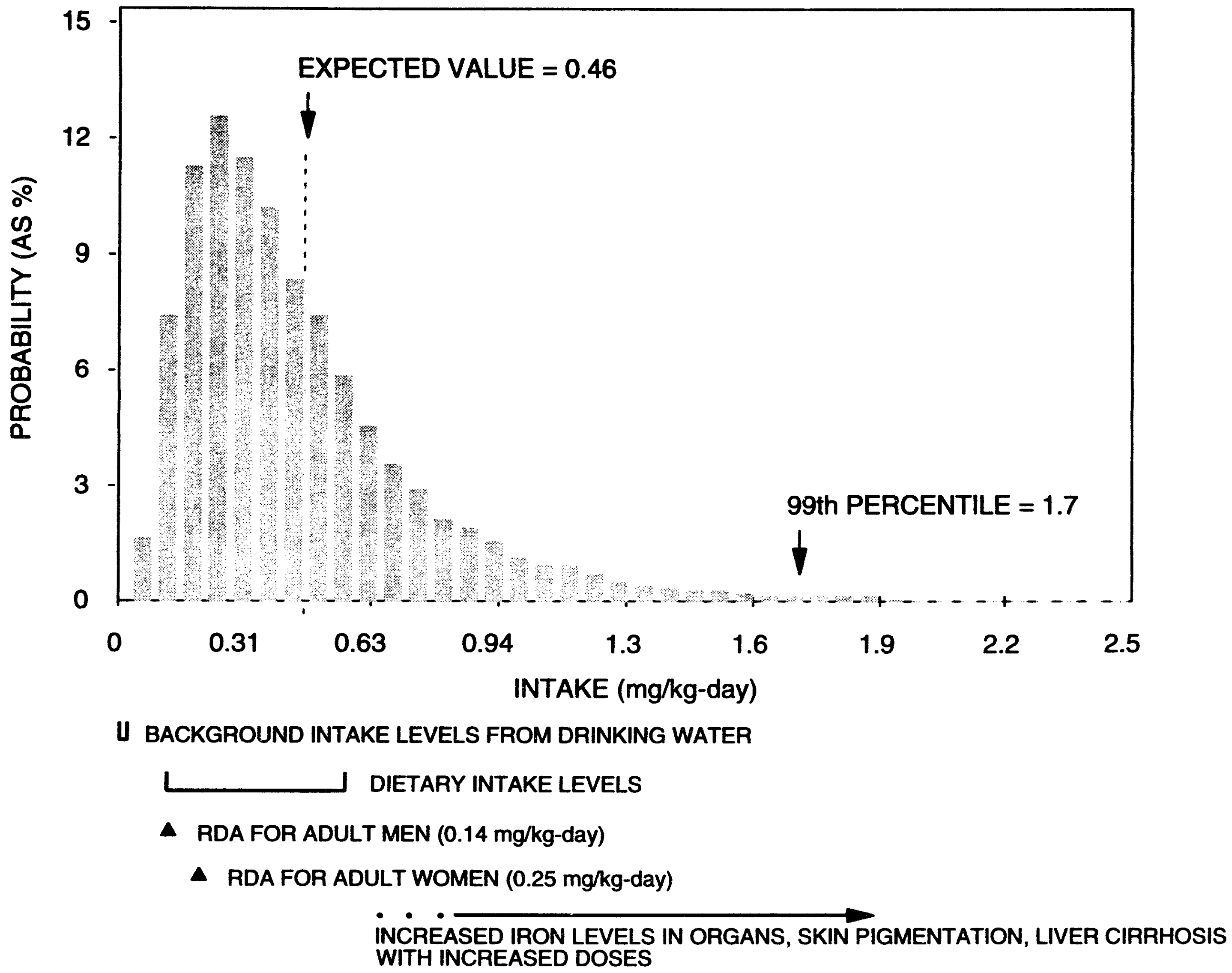


NOTE: THE MAJORITY (APPROXIMATELY 75%) OF THE POTENTIAL INTAKE IS BELOW THE LEVEL AT WHICH MILD TOXICITY HAS BEEN REPORTED.

FIGURE 6.5
HEALTH EFFECTS OF POTENTIAL CADMIUM INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE

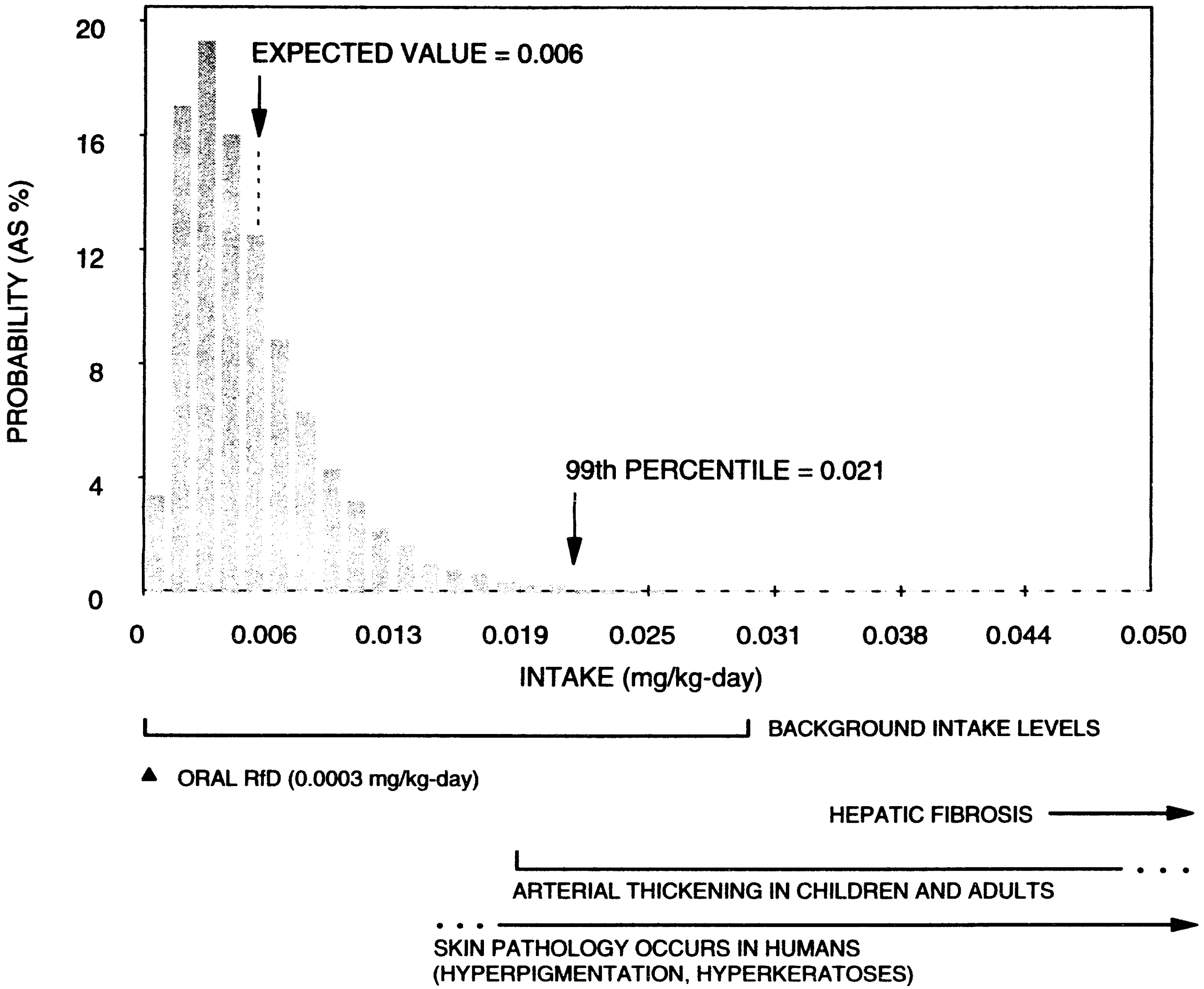
PATH I:\FL3.01\GRJBRA

HLTHCAD.DRW



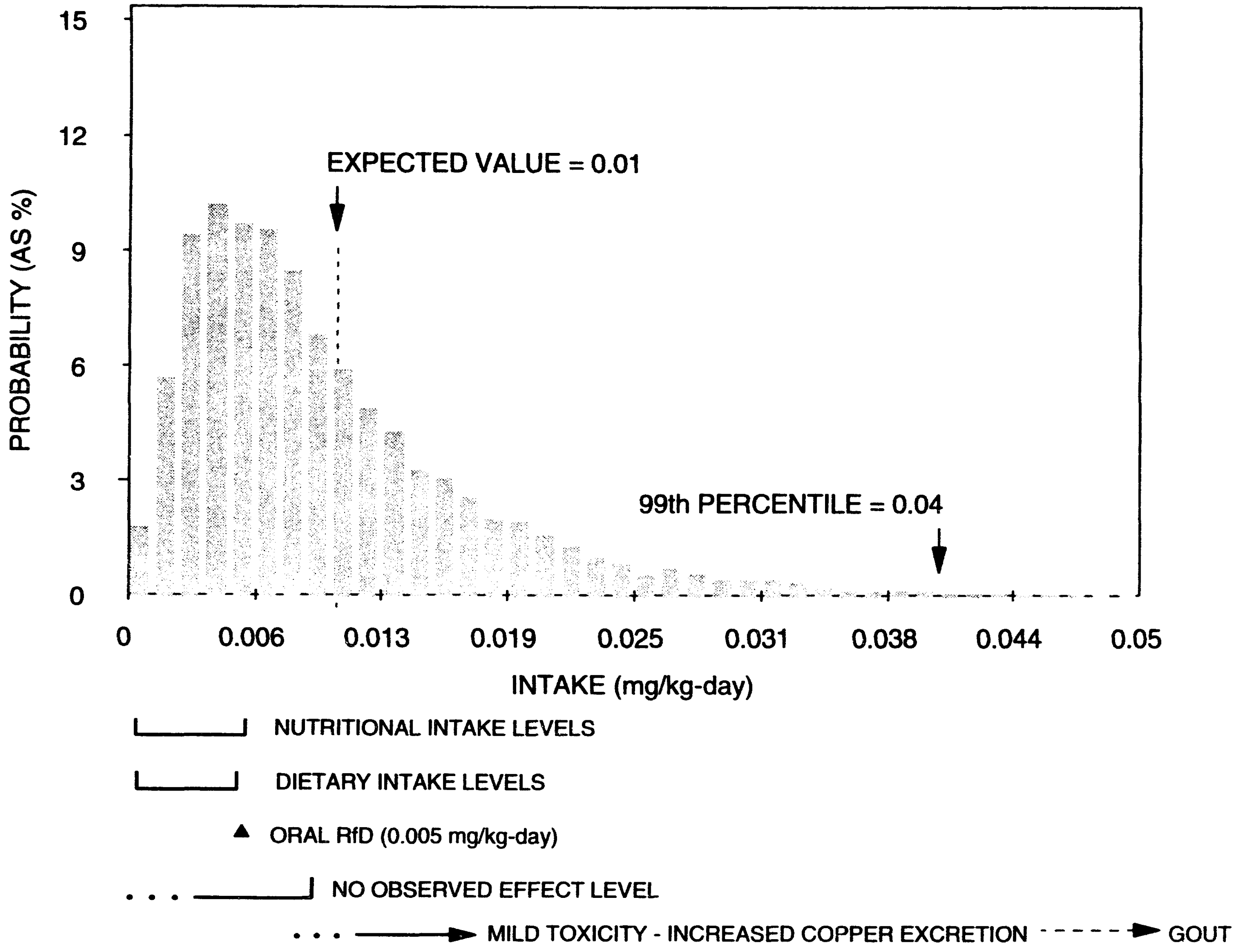
NOTE: A LARGE PORTION OF THE POTENTIAL INTAKE IS WITHIN DIETARY INTAKE LEVELS. LESS THAN 15% OF THE POTENTIAL INTAKE FALLS ABOVE THE LEVEL WHERE TOXIC EFFECTS HAVE BEEN REPORTED IN THE LITERATURE.

FIGURE 6.6
HEALTH EFFECTS OF POTENTIAL IRON INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE



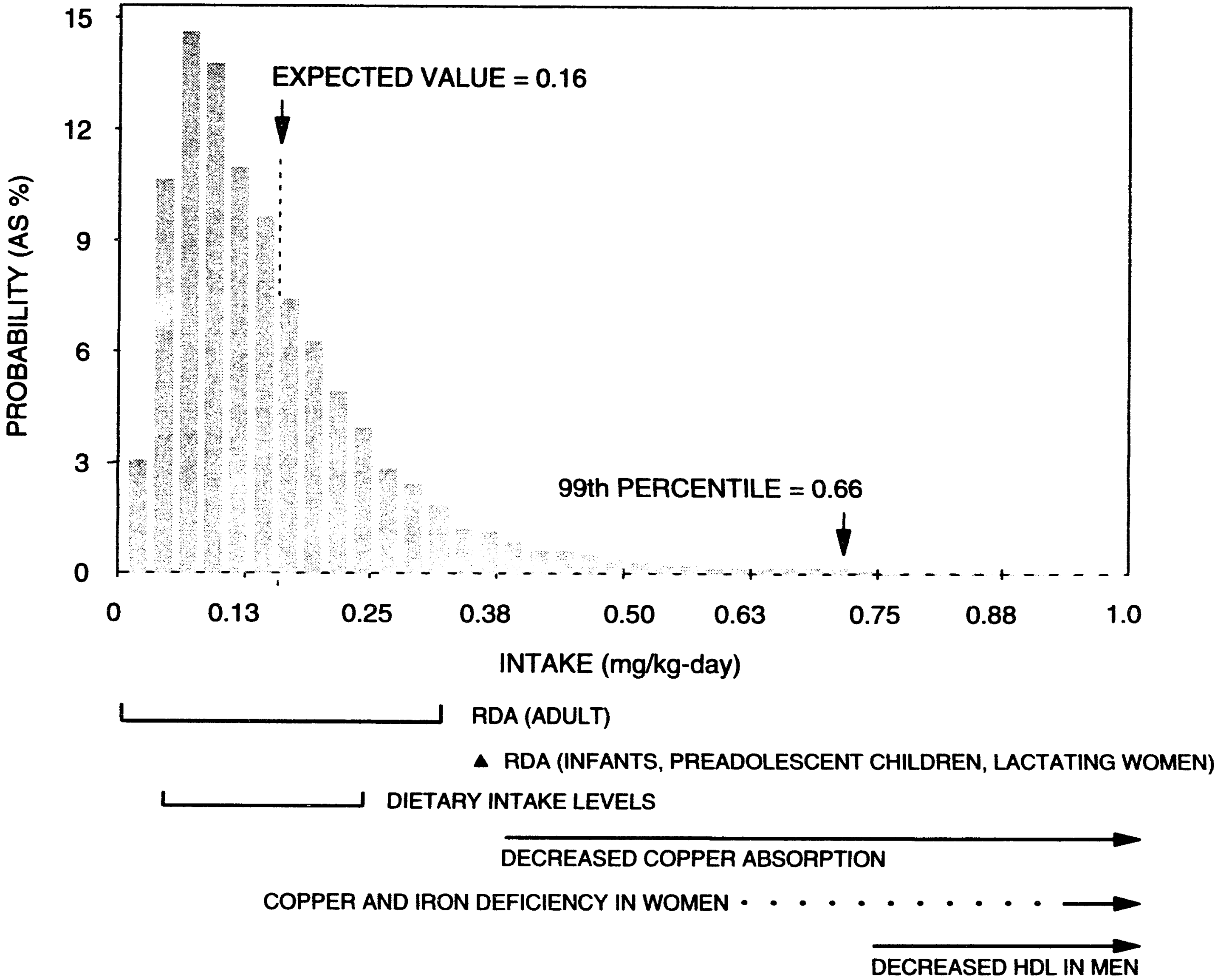
NOTE: ONLY A SMALL AMOUNT (LESS THAN 5%) OF THE POTENTIAL INTAKE IN CHILDREN WOULD BE ASSOCIATED WITH SKIN PATHOLOGY AND CARDIOVASCULAR SYMPTOMS.

FIGURE 6.7
HEALTH EFFECTS OF POTENTIAL ARSENIC INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE



NOTE: MILD TOXICITY SUCH AS INCREASED COPPER EXCRETION IS ASSOCIATED WITH THE UPPER 50% OF THE PREDICTED INTAKES.

FIGURE 6.8
HEALTH EFFECTS OF POTENTIAL MOLYBDENUM INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE



NOTE: THE MAJORITY OF THE POTENTIAL INTAKE IN CHILDREN WOULD NOT BE ASSOCIATED WITH ADVERSE HEALTH EFFECTS. A SMALL AMOUNT (LESS THAN 5%) WOULD RESULT IN SYMPTOMS OF COPPER DEFICIENCY.

FIGURE 6.9
HEALTH EFFECTS OF POTENTIAL ZINC INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE

range has been reported to result in allergic dermatitis, even with oral exposure (Figure 6.10).

For the noncarcinogenic effects of uranium, approximately 90 percent of the exposure distribution falls above the oral RfD. However, the potential exposure to uranium would not be expected to be associated with toxicity in animals (Figure 6.11). Although animal data are not always predictive of human toxicity, the lack of available human data to evaluate oral toxicity of uranium at these concentrations should not be seen as an indication that no toxic effects will occur. RfDs incorporate safety and uncertainty factors and therefore are generally conservative values designed to protect human health. However, they are based on careful evaluation of existing data bases; therefore, exposures that significantly exceed the RfD should be considered as potential problems. Reversible kidney damage has been reported in humans following acute exposures approximately six times greater than the highest values in this simulated potential exposure distribution.

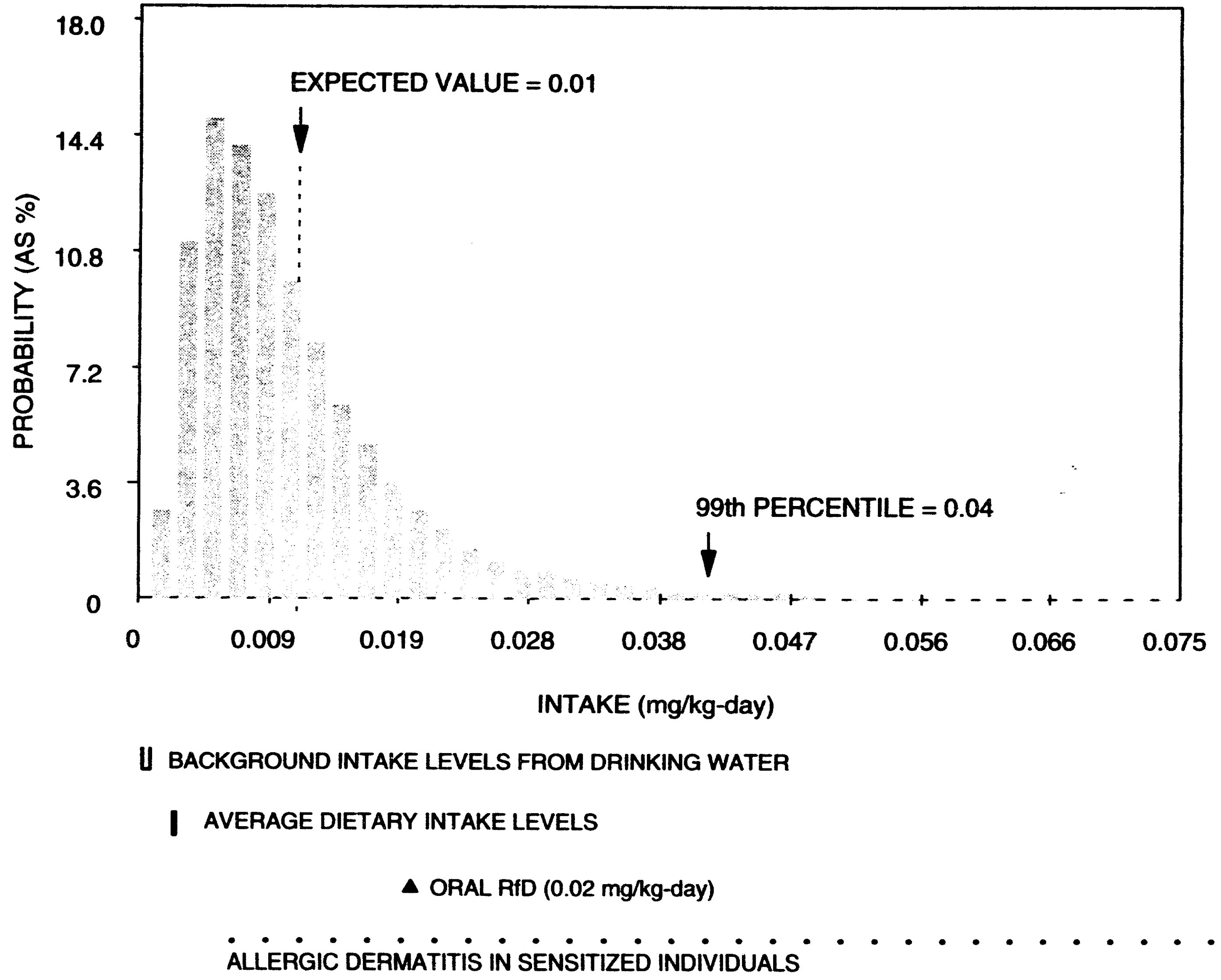
The range of potential exposure to cobalt (Figure 6.12) is below any potential toxic effects. However, concurrent exposure to both cobalt and nickel could produce allergic responses to cobalt following sensitization to nickel.

For each contaminant of potential concern except zinc, the contribution of exposure from other pathways (Section 4.2) would be approximately 1 percent or less. For zinc, the contribution of exposure from other pathways would be approximately 5 percent. These increases in potential exposure would not significantly increase the risk over that predicted from the drinking water pathway alone.

As discussed in Section 5.2, physiological interactions among the numerous metal contaminants at this site are likely. These interactions are likely to alter the absorption of some metals in the presence of others, compete for binding sites once absorbed, alter the concentrations of binding proteins that often transport metals in the bloodstream and through cell membranes, and alter the tissue distribution of individual metals. Research on interactions of two metals is relatively rare, and research on more than two is virtually nonexistent. The complexity of these interactions and the number of metal contaminants at Grand Junction make it impossible to predict whether the toxicity of a given metal will be increased or decreased by concurrent exposure to multiple other metals.

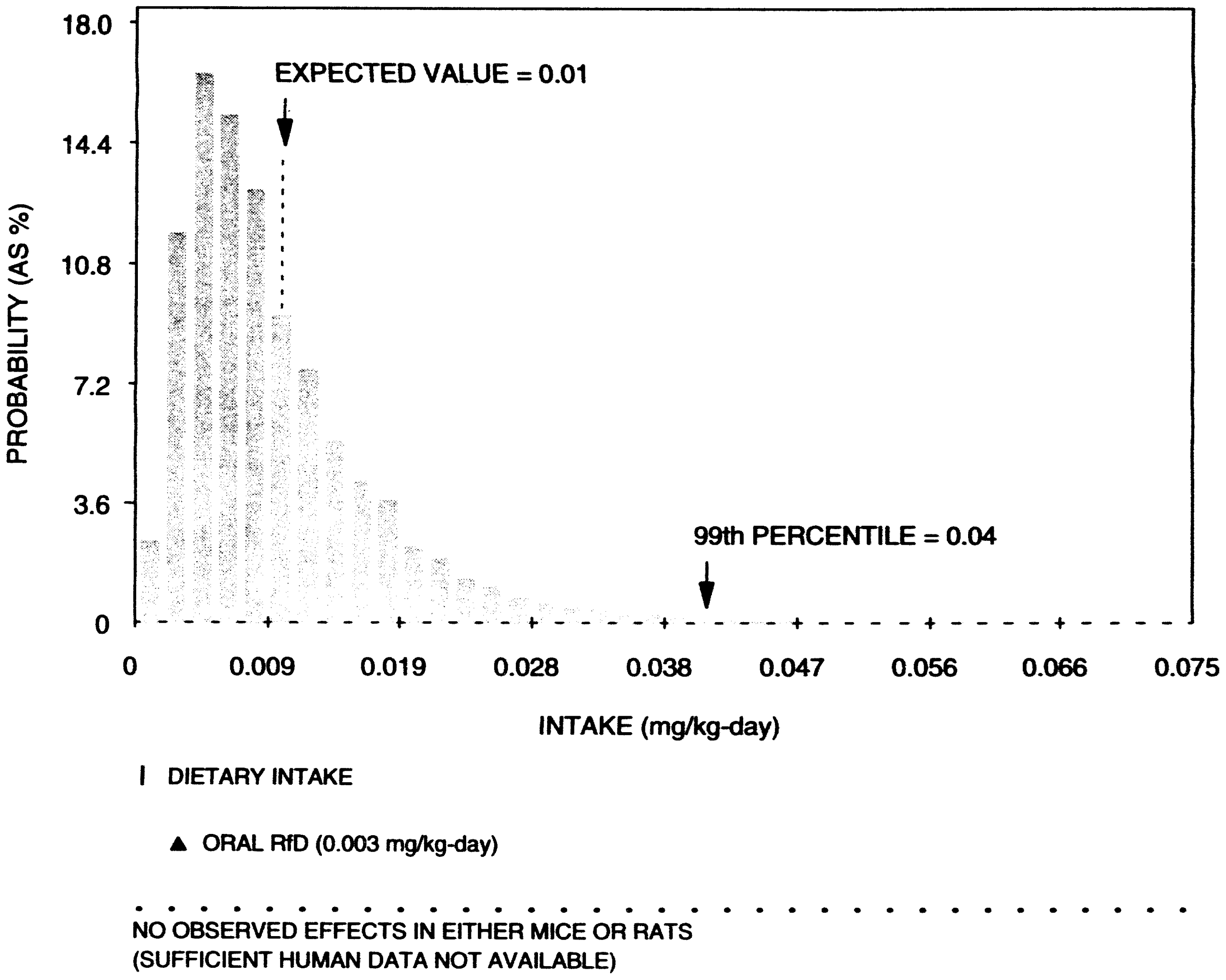
6.2 POTENTIAL CARCINOGENIC HEALTH EFFECTS

All uranium isotopes are radioactive and, as such, are considered potential carcinogens. Arsenic is not radioactive but is carcinogenic due to its chemical properties. Although the carcinogenic potential of arsenic is currently under evaluation by the EPA, the cancer SF previously assigned to arsenic is used in this risk assessment. Table 6.1 presents estimates of the excess lifetime cancer risk predicted to result from exposure to arsenic, uranium, and radium-226 through ingestion of contaminated ground water at the Grand Junction site.



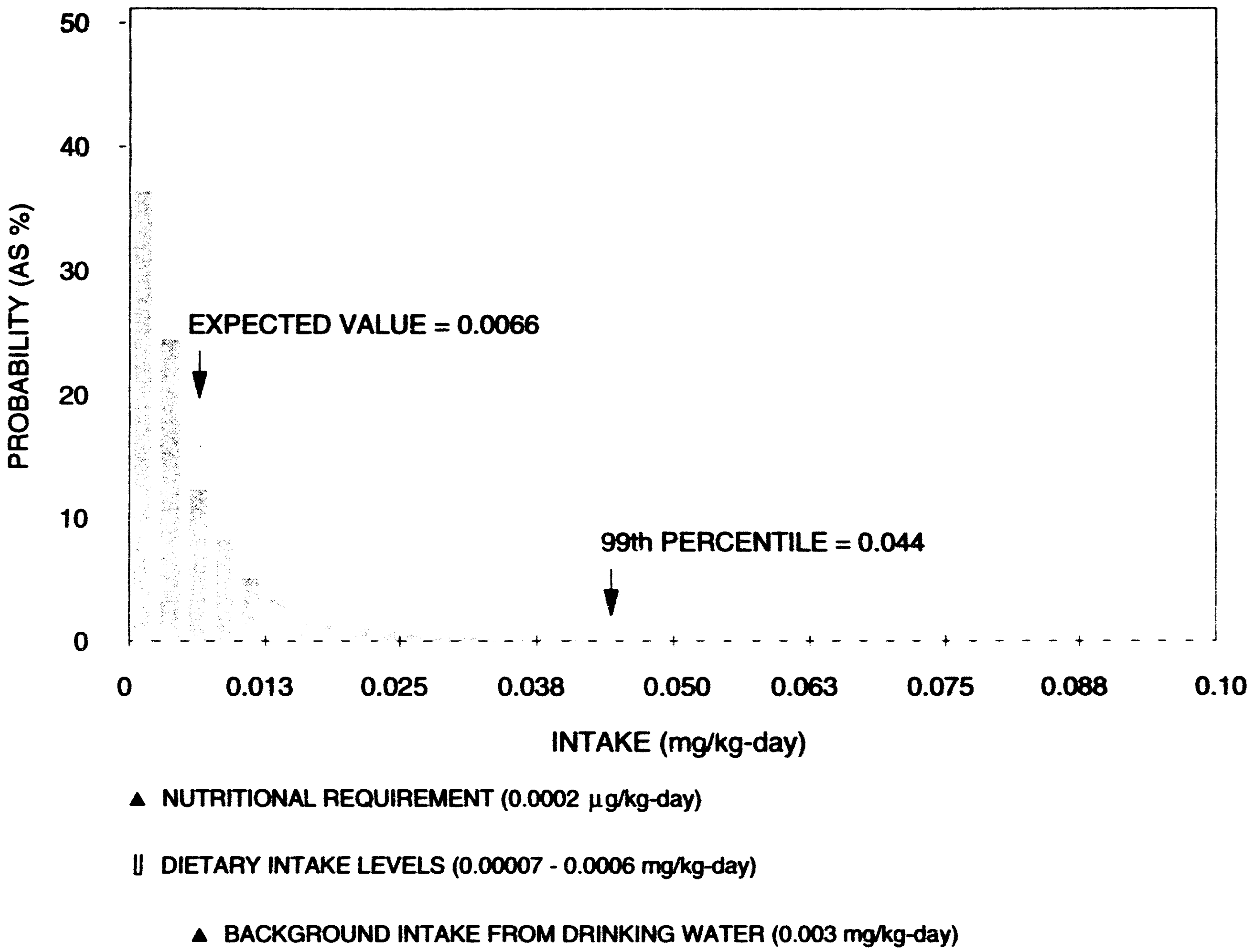
NOTE: THE MAJORITY OF THE POTENTIAL INTAKE IN CHILDREN WOULD NOT BE ASSOCIATED WITH ADVERSE HEALTH EFFECTS IN NONSENSITIZED INDIVIDUALS. HOWEVER, THE UPPER 65% OF THE POTENTIAL INTAKE WOULD RESULT IN ALLERGIC DERMATITIS IN SENSITIZED INDIVIDUALS.

FIGURE 6.10
HEALTH EFFECTS OF POTENTIAL NICKEL INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE



NOTE: NO OBSERVED ADVERSE HEALTH EFFECTS WOULD BE ASSOCIATED WITH THE POTENTIAL INTAKES IN CHILDREN.

FIGURE 6.11
HEALTH EFFECTS OF POTENTIAL URANIUM INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE



NOTE: NO ADVERSE HEALTH EFFECTS HAVE BEEN ASSOCIATED
WITH THE POTENTIAL EXPOSURES IN CHILDREN

FIGURE 6.12
HEALTH EFFECTS OF POTENTIAL COBALT INTAKES FOR CHILDREN
GRAND JUNCTION, COLORADO, SITE

Table 6.1 Excess lifetime cancer risk calculations for ground water ingestion by a hypothetical future adult resident, Grand Junction UMTRA Project site, Grand Junction, Colorado

Contaminant of potential concern	Concentration in ground water (mg/L; pCi/L)		Ground water exposure doses (pCi per lifetime)		Oral SF (mg/kg-day; pCi)$^{-1}$	Excess lifetime cancer risk[a]	
	Median	Maximum	Median	Maximum		Median	Maximum
Arsenic	0.016	0.18	1.9E-04	2.1E-03	1.8E+0	3×10^{-4}	4×10^{-3}
Radium-226	2.1	29	4.4E+04	6.1E+05	1.2E-10	5E-06	7E-05
Uranium[b]	114	234	2.4E+06	4.9E+06	1.6E-11	4E-05	8E-05
					Total:	3E-04	4E-03

[a] Excess lifetime cancer risk is calculated by multiplying the exposure dose by the SF.

[b] Uranium-234 and uranium-238 combined. The oral SF is the same for both isotopes.

These estimates are based on the cancer SFs developed by the EPA; however, natural uranium has not been demonstrated to cause cancer in humans or animals following ingestion exposures. Neither the median nor the maximum estimates for excess cancer risk from radium-226 or uranium exposure exceed the National Contingency Plan (NCP) guidance for maximum increased lifetime cancer risk of 1×10^{-4}. However, both of the excess lifetime cancer risk estimates for arsenic exceed the NCP guidance. Summing the risks calculated in Table 6.1 to one significant figure, median and maximum excess cancer risk estimates from ground water ingestion of arsenic and the two radionuclides combined would be 3×10^{-4} and 4×10^{-3}, respectively.

6.3 LIMITATIONS OF RISK EVALUATION

The following potential limitations apply to interpretations of this risk evaluation:

- This risk assessment evaluates only risks related to inorganic ground water contamination. Potential contamination with any of the few organic constituents used in uranium processing has not been addressed.
- With the exception of individuals sensitized to nickel, subpopulations that might have increased sensitivity are not specifically addressed on the graphs.
- Some individuals may be more sensitive to the toxic effects of certain constituents for reasons that have not been determined.
- Data available to interpret potential adverse health effects are not always sufficient to allow accurate determination of all health effects (i.e., lack of testing in humans or testing of dose ranges other than those expected at this site).
- Although plume movement is evaluated hydrologically and geochemically, the monitoring locations sampled may not be in the most contaminated portion of the plume.
- Only the drinking water exposure pathway has been considered in depth, although other pathways were screened to determine their contribution.

The evaluation presented here has considered these limitations and compensated wherever possible by presenting toxicity ranges rather than point estimates to incorporate as much variability as could be reasonably defined. The impact of these potential limitations is discussed more fully in Section 8.2.

7.0 LIVESTOCK AND ENVIRONMENTAL EVALUATION

The objective of the environmental portion of the risk assessment is to determine whether contaminants detected at the site have the potential to adversely affect the existing biological community at or surrounding the site. Currently, the EPA has no guidance for quantifying potential impacts to ecological receptors but has developed a qualitative approach generally used for ecological evaluation (EPA, 1989b). With the qualitative approach, the EPA recommends that ambient environmental media concentrations be compared to water quality, sediment quality, or other relevant criteria to determine whether any of the concentrations that the ecological receptors are expected to encounter exceed these criteria.

The effects of contaminants on ecological receptors are a concern; however, it is difficult to predict whether observed effects on individual populations will result in any damage to the ecosystem. Populations are dynamic; therefore, information concerning the normal range of variability within the population needs to be known. Sublethal effects, which may be very important to overall ecosystem health, are difficult to detect, and contaminants present at low concentrations may not kill organisms directly but may diminish their ability to survive and reproduce.

7.1 EXPOSURE CHARACTERIZATION

This section identifies the potential exposure pathways associated with the Grand Junction processing site. For risk to exist, a receptor must be exposed to contaminants. Exposure can occur only if there is both a source of contamination and a mechanism of transport to a receptor population or individual.

Currently impacted media at the site include contaminated ground water and potentially contaminated surface water and sediments. Since the tailings pile and contaminated soil have been removed from the processing site, some direct exposure pathways (such as incidental ingestion of soil, dermal contact with soil, and inhalation of air containing particulates) do not represent an ecological concern and will not be evaluated in this baseline risk assessment. Other direct exposure pathways (such as ingestion of surface water and sediments potentially affected by contaminated ground water or particulate transport and bioconcentration of contaminants in surface water by aquatic organisms) and indirect exposure pathways (such as consumption of previously exposed organisms) are possible at the site.

The net accumulation by organisms of a constituent directly from the surrounding environment is known as bioconcentration. Net accumulation by organisms as a result of all routes of exposure, including the diet, is known as bioaccumulation. Generally, bioconcentration is measured for uptake of chemicals from water by aquatic organisms. BCFs for ingestion of, and dermal contact with, soils are too variable and dependent on site conditions to make identification of generic soil BCFs possible.

The main surface water body in the area of the Grand Junction processing site is the Colorado River. The river forms the southern boundary of the site. The Colorado River is braided by several islands from the upstream side of the processing site to a point about 0.5 mi (0.8 km) downstream of the site. Approximately 47 ac (19 ha) of the Colorado River floodplain near the Grand Junction site have been identified as wetlands by the U.S. Army Corps of Engineers (Figure 7.1) (DOE, 1986). About 0.75 mi (1.2 km) downstream of the processing site is the Colorado River confluence with the Gunnison River. Several manmade drainage ditches exist in the vicinity of the processing site.

Surface water bodies are potential exposure points for resident aquatic life and for terrestrial wildlife (including domestic animals) to come in contact with surface water and/or sediments. These exposure pathways were evaluated in this risk assessment.

Another potential current pathway could involve plant uptake of contaminants in ground water. Due to the shallow depth to ground water (approximately 5 ft [1.5 m] or less below land surface), plants can reach contaminated ground water. To evaluate plant uptake in this risk assessment, plant roots were assumed to reach soil saturated with ground water containing the mean concentrations for the most contaminated wells for the contaminants of potential concern. Plant BCFs from the literature were used in the evaluation.

For the purposes of this baseline risk assessment, it was assumed as a conservative measure that a domestic well could, at some point in the future, be placed on the site at a location that intercepts the most contaminated ground water in the plume. The water from this hypothetical well could be used for a livestock watering pond (which could also be stocked with fish) or for irrigation of agricultural crops.

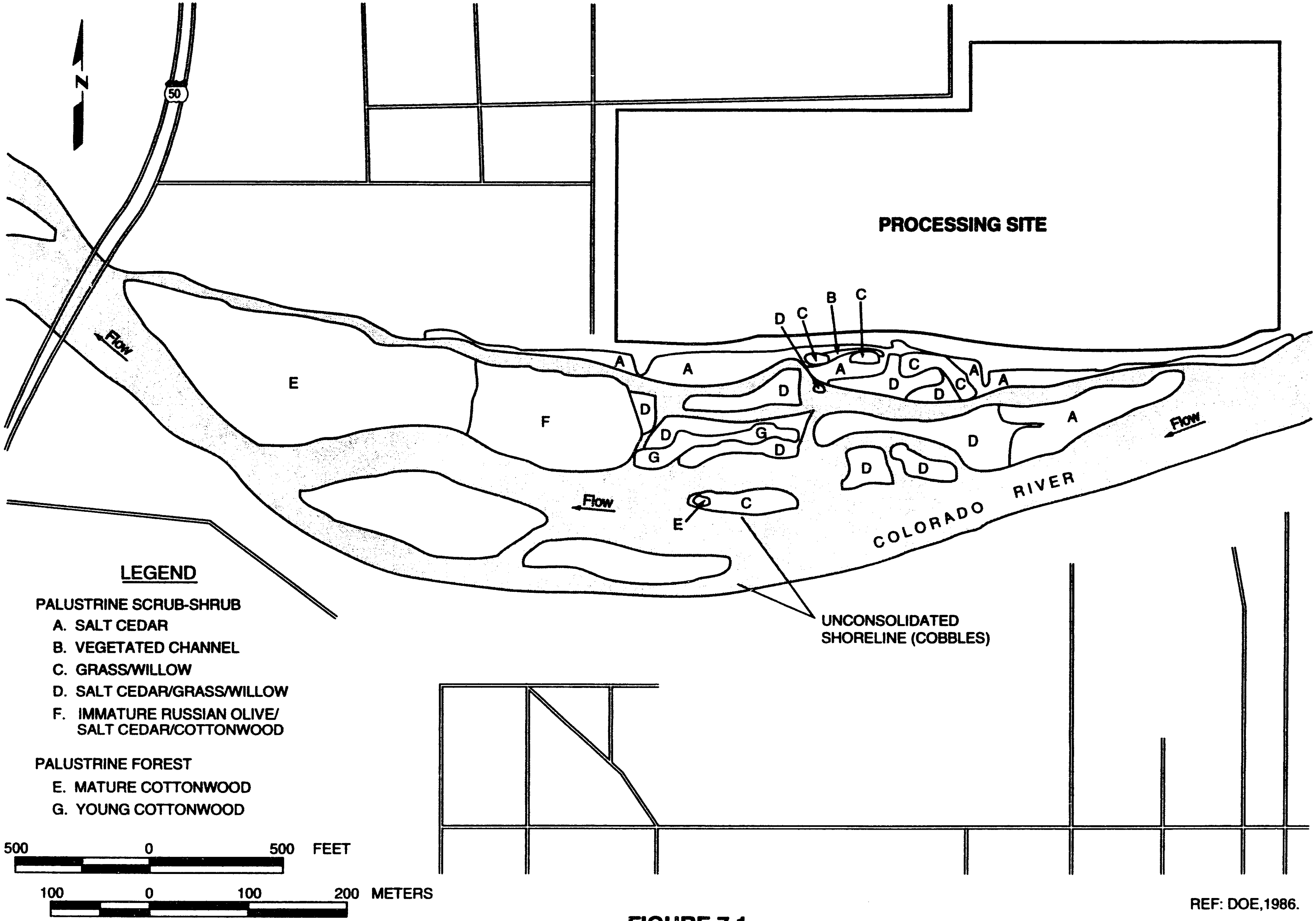
7.2 ECOLOGICAL RECEPTORS

This section identifies the ecological resources present at the site and its vicinity that are potentially exposed to site-related contaminants.

The following information on ecological receptors is based primarily on surveys done before the tailings removal process was initiated and is provided as a historical perspective. Limited observations of aquatic organisms and terrestrial plant and animal communities were conducted at the surface water and sediment sampling locations during a September 22, 1993, qualitative field survey (TAC, 1993). It is recommended that additional ecological characterization be conducted after remediation of the tailings pile is completed.

7.2.1 Terrestrial plant and animal communities

The Grand Junction site is in an urban setting, and the only terrestrial plant communities that have not been highly altered by man are the riparian communities along the river on the south side of the site. This riparian area



REF: DOE,1986.

FIGURE 7.1
WETLAND PLANT COMMUNITY TYPES ADJACENT TO THE GRAND JUNCTION PROCESSING SITE

MAC: SITE/GRJ/BRA/WETCOMTYPES

consists of a number of small islands with Colorado River side channels and backwaters creating a high level of interspersion of aquatic and terrestrial habitats (Figure 7.1). The dominant woody vegetation on most of the islands is salt cedar, which forms very dense thickets in some areas. Russian olive and willow are common subdominant species on these islands. Ground cover dominated by reed canary grass is very dense, particularly in the more open areas; other common species in the open area are spotted snapweed and giant reed. Species such as *Juncus* sp., *Carex* sp., *Eleocharis* sp., softstem bullrush, and arrowhead were common in the muddy shores of the side channels and backwaters and in small wetland areas on the islands (TAC, 1985; 1993).

Wildlife use of the riparian habitat is extensive. Species such as the yellow warbler, mourning dove, song sparrow, and black-billed magpie are common nesting species within the salt cedar and willow plant communities. Water birds commonly observed were the mallard and great blue heron. Beaver activity was evident throughout the area, and muskrat, raccoon, and skunk tracks were common. Amphibians observed included the bull frog and leopard frog (TAC, 1985; 1993).

An analysis of threatened and endangered species for the Grand Junction site is in the Grand Junction environmental impact statement (DOE, 1986). An occasional wintering bald eagle would be the only terrestrial endangered species that may occur in the area of the Grand Junction site.

7.2.2 Aquatic organisms

The Colorado River in the area of the Grand Junction processing site meanders through residential and industrial areas and is braided around gravel and vegetated islands. The river has large annual fluctuations in flow, temperature, and turbidity. No quantitative surveys of aquatic organisms occurring in the Colorado River near the site have been conducted to date as part of a Technical Assistance Contractor investigation. A brief qualitative survey of the aquatic organisms in the Colorado River was conducted in the vicinity of historic surface water sampling locations 423, 424, 425, 427, and the new location, 428, which is in a channel that bisects an island adjacent to the site. The following organisms were observed using a fine-mesh dip-net at all of the locations: mayfly nymphs (*Ephemeroptera*); dragonfly and damselfly nymphs (*Odonata*); water striders (*Gerridae*); and backswimmers (*Notonectidae*). Additionally, midge larvae (*Chironomidae*) and several 1- to 3-inch (2.5- to 5-cm) minnows (*Cyprinidae*) were observed at location 428. Aquatic life known to occur in the area are the bluehead sucker, flannelmouth sucker, common carp, roundtail chub, red shiner, sand shiner, and fathead minnow. Game species include the green sunfish, bluegill, largemouth bass, black crappie, black bullhead, and channel catfish (FWS, 1982a; DOE, 1986).

Threatened or endangered fish species potentially in the Grand Junction area include the humpback chub, bonytail chub, Colorado squawfish, and razorback sucker (FWS, 1982b).

7.3 CONTAMINANTS OF ECOLOGICAL CONCERN

The complete list of ground water contaminant levels that exceed background levels (Table 3.4) was considered in assessing contaminants of potential concern for ecological receptors potentially exposed to ground water (for example, plant uptake, livestock watering, and fish stocked in a pond). This list of contaminants was then compared with the surface water data. Table 7.1 presents the surface water data for those contaminants detected in the surface water samples and above background levels in ground water.

If a contaminant was never detected in surface water (for example, cadmium) or the concentration detected downstream of the site was less than or equal to the concentration upstream of the site (the background level), it was excluded as a contaminant of potential concern for ecological receptors. Additionally, boron, fluoride, and zinc are excluded as contaminants of potential concern because the difference between the adjacent and downstream location concentrations versus the upstream concentration was minimal (less than 10 percent).

The concentrations used in these comparisons to background levels were from unfiltered samples for most of the constituents. It would be preferable to use data from filtered samples, because most of the state of Colorado's water quality standards are stated as dissolved (filtered) metal concentrations; however, the majority of available data are from unfiltered samples. The only filtered samples that have been collected from the river were in 1991. Several rounds of unfiltered samples have been collected between 1991 and 1993. The unfiltered data are used in this preliminary assessment because they are considered more representative of conditions in the river.

After these comparisons were made, the following contaminants of potential ecological concern were selected for the Colorado River: ammonium, copper, iron, radium-226, uranium, and vanadium.

No sediment samples were collected from the Colorado River before the September 1993 sampling. Only five constituents were analyzed at that time. Thus, the constituents analyzed in the sediments (molybdenum, selenium, strontium, sulfate, and uranium) were evaluated in this assessment. This list of analytes was selected prior to development of the risk assessment. The sediment data, by location, are presented in Table 7.2.

Table 7.1 Occurrence of constituents detected in the Colorado River near the Grand Junction UMTRA Project site, Grand Junction, Colorado

	Upstream of the site	Adjacent to the site	Downstream of the site
Constituent	Location ID 423	Location IDs 424, 425, 428	Location ID 426
Ammonium	0.13	0.25	0.25
Arsenic	0.0085	0.0045	0.0045
Boron	0.042	0.046	0.046
Cadmium	ND	ND	ND
Calcium	93	89	90
Chloride	170	150	170
Cobalt	0.011	0.0099	ND
Copper	0.0098	0.013	0.0062
Fluoride	0.33	0.34	0.30
Iron	4.6	6.2	3.1
Magnesium	29	26	26
Manganese	0.11	0.11	0.081
Molybdenum	0.016	0.016	0.011
Nickel	0.018	0.014	ND
Potassium	4.7	4.7	4.1
Radium-226 (pCi/L)	0.93	1.4	1.6
Sodium	130	120	130
Sulfate	240	190	220
Uranium	0.0047	0.0049	0.0062
Vanadium	0.019	0.027	0.015
Zinc	0.034	0.036	0.020

Note: All concentrations are mean concentrations, reported in milligrams per liter from unfiltered samples. Samples collected from 1991 through 1993.

ND—not detected.

Table 7.2 Occurrence of constituents analyzed for in Colorado River sediments, Grand Junction UMTRA Project site, Grand Junction, Colorado

	Upstream	Adjacent			Downstream
	Location ID	Location IDs			Location ID
Constituent	423	424	425	428	427
Molybdenum	4	<1	<1	<1	<1
Selenium	<0.5	<0.5	<0.5	<0.5	<0.5
Strontium	160	127	108	108	107
Sulfate	105	273	500	158	56
Uranium	1.5	1.2	2.0	1.2	4.9

Note: All results reported in milligrams per kilograms dry weight (mg/kg DW). The samples were collected September 22, 1993.

7.4 POTENTIAL IMPACTS TO WILDLIFE AND PLANTS

7.4.1 Ground water

Concentrations of the contaminants of potential concern in plant tissue were estimated using soil-to-plant BCFs. Soil concentrations in the saturated zone were estimated by multiplying the ground water concentration by the soil-water distribution coefficient, Kd. The methodology and parameters used to estimate root uptake and plant tissue concentrations at harvestable maturity for the contaminants of potential concern are presented in Table 7.3. This methodology is described in detail elsewhere in the literature (Baes et al., 1984) and therefore will not be presented here.

The estimated tissue concentrations for the contaminants of potential concern in the vegetative portions (for example, stems and leaves) and in the nonvegetative portions (for example, fruits and tubers) of plants at harvestable maturity were compared to approximate concentrations (in mature leaf tissue) that have been reported to be toxic to plants (phytotoxic) (Table 7.3). As illustrated in Table 7.3, few available data relate tissue concentrations to phytotoxicity. The reported phytotoxic concentrations are not representative of very sensitive or highly tolerant plant species. The estimated tissue concentrations for the contaminants of potential concern in plants that may reach soil saturated with contaminated ground water do not exceed the available phytotoxicity data. No comparison data were available for calcium, chloride, iron, magnesium, potassium, radium-226, sodium, sulfate, and uranium. Thus, it is not possible with available information to evaluate whether the estimated tissue concentrations could result in adverse effects to plants.

Table 7.3 Comparison of estimated plant concentrations to phytotoxic concentrations, Grand Junction UMTRA Project site, Grand Junction, Colorado

Contaminant of potential concern	Mean concentration in ground water (mg/L)	Kd (L/kg)	Estimated soil concentration (mg/kg DW)	Soil-to-plant concentration factors		Estimated concentration in vegetative growth[a] (mg/kg DW)	Estimated concentration in fruits/tubers[b] (mg/kg DW)	Approximate concentration in mature leaf tissue that is toxic[c] (mg/kg DW)
				Bv	Br			
Arsenic	0.034	5.9	0.20	0.04	0.006	0.008	0.0012	5-20
Boron	0.57	0.19	0.11	4	2	0.43	0.22	50-200
Calcium	540	0	NC	3.5	0.35	NC	NC	NA
Chloride	770	0	NC	70	70	NC	NC	NA
Cobalt	0.084	1.9	0.16	0.02	0.007	0.003	0.0011	15-50
Copper	0.047	42	2.0	0.4	0.25	0.80	0.50	20-100
Fluoride	3.8	0	NC	0.06	0.006	NC	NC	50-500
Iron	5.8	15	87	0.004	0.001	0.35	0.087	NA
Magnesium	285	70	19,950	1	0.55	19,950	10,970	NA
Manganese	14	17	240	0.25	0.05	60	12	200[d]-1000
Molybdenum	0.26	40	10	0.25	0.06	2.5	0.6	10-50
Nickel	0.16	12	1.9	0.06	0.06	0.11	0.11	10-100
Potassium	88	0	NC	1.0	0.55	NC	NC	NA
Sodium	930	0	NC	0.075	0.055	NC	NC	NA
Sulfate[e]	3800	0	NC	0.5	0.5	NC	NC	NA
Uranium	0.21	0	NC	0.0085	0.004	NC	NC	NA
Vanadium	2.1	50	105	0.0055	0.003	0.58	0.32	5-10
Zinc	2.9	13	38	1.5	0.9	57	34	100-400
Radionuclide								
Radium-226[f]	6.2E-09	24	1.4E-07	0.015	0.0015	2.2E-09	2.2E-10	NA

Table 7.3 Comparison of estimated plant concentrations to phytotoxic concentrations, Grand Junction UMTRA Project site, Grand Junction, Colorado (Concluded)

[a] Estimated concentration in vegetative portions, calculated as estimated soil concentration multiplied by Bv.
[b] Estimated concentration in nonvegetative portions, calculated as estimated soil concentration multiplied by Br.
[c] Concentrations are not presented for very sensitive or for highly tolerant plant species (Kabata-Pendias and Pendias, 1992).
[d] The value of 200 mg/kg is a recommended criterion of the Illinois Institute of Environmental Quality (Saric, 1986).
[e] Bv and Br factors available for elemental sulfur only; thus, these factors were reduced by a factor of 3 for sulfate.
[f] The ground water concentration in pCi/L was converted to mg/L.

Kd—soil-water distribution coefficient; from PNL, 1989.
Bv—soil-to-plant elemental transfer factor for vegetative portions of food crops and feed plants (Baes et al., 1984).
Br—soil-to-plant elemental transfer factor for nonvegetative portions (e.g., fruits, tubers), of food crops and feed plants (Baes et al., 1984).
DW—dry weight.
mg/kg—milligrams per kilogram.
mg/L—milligrams per liter.
L/kg—liters per kilogram.
NA— not available.
NC—value cannot be calculated because Kd is zero.

No Kd, Bv, or Br values available for ammonium.

Bioaccumulation in terrestrial organisms as a function of contaminants of concern in ingested plants or animals (for example, birds eating fish) is a potential exposure pathway at the site. Birds and other vertebrates consuming these plants and animals can bioaccumulate some of the contaminants of concern from this diet if the amount ingested exceeds the amount eliminated. This is often a function of the areal extent of contamination versus the areal extent of the animals' feeding range. In the case of small contaminated areas, the amount of food in the diet usually exceeds the impacted food, and bioaccumulation is not a concern. Therefore, exposure via the diet for all food chain species is possible in certain areas (for example, wetland areas), but the potential for bioaccumulation is not always a concern.

Biomagnification is a more severe situation in which the concentration of a constituent increases in higher levels of the food chain because the contaminant concentrations are accumulated through each successive trophic level. Of particular concern for biomagnification effects are the top predators, especially the carnivorous birds and mammals. Only a limited number of constituents have the potential for magnifying in the food chain. Most constituents are metabolized in organisms and eliminated at each level of the food chain. Thus, the constituent concentration does not increase up the food chain. Based on available information, the potential for the detected contaminants of potential concern to represent a concern via food chain transfer is probably low.

To evaluate the potential impact that use of contaminated ground water in a livestock pond might have on wildlife (that is, animals drinking from the pond or fish stocked in the pond), the median ground water concentrations for the contaminants of potential concern were compared to available comparison water quality criteria (Table 7.4). There are no available federal or state criteria or standards established for the protection of terrestrial wildlife via water exposure. Therefore, it is difficult to evaluate the potential hazards to terrestrial receptors without additional information. However, available surface water quality values for the protection of freshwater aquatic life do exist and include the state of Colorado standards (CDH, 1991).

Mean ground water concentrations for chloride, iron, manganese, radium-226, and zinc exceeded the comparison aquatic life water quality values (Table 7.4), indicating that this water would be unacceptable for aquatic organisms. Ground water concentrations for arsenic, boron, copper, molybdenum, nickel, and uranium were below the comparison values. No comparison water quality values are available for ammonium, calcium, cobalt, fluoride, magnesium, potassium, sodium, sulfate, and vanadium.

Another future hypothetical use of the ground water in the area is for irrigating agricultural crops. Table 7.4 compares the approximate concentrations in water used for irrigation purposes that should be protective of plants with the ground water concentrations (EPA, 1972). The EPA developed these approximate irrigation water concentrations to protect agricultural crops from toxicity associated with buildup of a particular constituent in the soil. Eleven of the

Table 7.4 Comparison of contaminants of potential concern in ground water with available water quality values, Grand Junction UMTRA Project site, Grand Junction, Colorado

Contaminant of potential concern	Mean concentration in ground water	Aquatic life water quality value[a]	Water concentration protective of livestock[b]	Concentration in irrigation water protective of plants[b]
Ammonium	340	NA	NA	NA
Arsenic	0.034	0.15	0.20	0.10
Boron	0.57	1.0[c]	5.0	0.75
Calcium	540	NA	NA	NA
Chloride	770	230[d]	NA	NA
Cobalt	0.084	NA	1.0	0.05
Copper	0.047	0.19[e]	0.5	0.20
Fluoride	3.8	NA	2.0	1.0
Iron	5.8	1.0	NA	5.0
Magnesium	285	NA	NA	NA
Manganese	14	1.0	NA	0.20
Molybdenum	0.26	50 (0.79[f])	NA	0.010
Nickel	0.16	1.1[e]	NA	0.20
Potassium	88	NA	NA	NA
Radium-226	6.1 pCi/L	5 pCi/L	NA	NA
Sodium	930	NA	NA	NA
Sulfate	3800	NA	1000[h]	NA
Uranium	0.21	52[e]	NA	NA
Vanadium	2.1	NA	0.10	0.10
Zinc	2.9	1.6[e]	25	2.0

[a] Value obtained from the Basic Standards and Methodologies for Surface Water, 3.1.0 (5 CCR 1002-8), Colorado Department of Health, Water Quality Control Commission (CDH, 1991), unless specified otherwise. These values are standards protective of aquatic life via chronic exposure.

[b] From EPA (1972), unless specified otherwise. Irrigation water values shown are for water used continuously on all soils.

[c] No state or federal water quality value available. Value presented is the current boron criterion recommended by the FWS for the protection of aquatic life (Eisler, 1990).

[d] No state water quality value available. Value presented is the Federal Water Quality Criterion (FWQC) for the protection of freshwater aquatic life via chronic exposure (EPA, 1992).

[e] Water hardness-related state standard (CDH, 1991). Criterion presented was calculated using the mean hardness (2500 mg/L) determined from concentrations of calcium and magnesium in the plume wells.

[f] No state or federal water quality value available. Value presented is the current molybdenum criterion recommended by the FWS for the protection of aquatic organisms, with one exception. The exception is for newly fertilized eggs of rainbow trout, which are sensitive to molybdenum concentrations above 0.79 mg/L (Eisler, 1989).

[g] From National Research Council (1971).

Concentrations reported in milligrams per liter unless otherwise noted.
NA—not available.

contaminants of potential concern—arsenic, boron, cobalt, copper, fluoride, iron, manganese, molybdenum, nickel, vanadium, and zinc—have comparison values. The mean ground water concentrations for cobalt, fluoride, iron, manganese, molybdenum, vanadium, and zinc exceed the comparison values, while the concentrations of arsenic, boron, cobalt, copper, iron, nickel, and zinc are below the comparison values. No comparison values are available for the remaining contaminants of potential concern. Thus, it is not possible to evaluate the potential for these compounds to adversely affect plants when applied in irrigation water.

Based on the available information, use of the alluvial ground water near the site (containing the mean concentrations) as a continuous source of irrigation water may result in deleterious effects to crops, primarily because of the elevated concentrations of cobalt, fluoride, iron, manganese, molybdenum, vanadium, and zinc.

7.4.2 Colorado River water

Aquatic life in the Colorado River may be exposed to contaminants associated with the site. The plume of contaminated ground water is believed to be discharging to the river. It is also possible that contaminants may have washed into the river through surface water runoff before the tailings were removed.

Terrestrial wildlife can be directly exposed to contaminants in the Colorado River by ingesting surface water, sediments, aquatic organisms, and plants that may have bioconcentrated contaminants. However, good information on generic BCFs for terrestrial wildlife is currently not available in the scientific literature.

Risk to aquatic life

Surface water data collected from the river at the upstream location (location 423) were compared with data from locations adjacent to the site (locations 424, 425, and 428) and the downstream location (location 426) as part of the selection process for the contaminants of ecological concern (Section 7.3). The results of the comparison indicated that most of the constituents did not exceed background concentrations in the river. The mean concentrations for copper, iron, and vanadium at the locations adjacent to the site were slightly higher than those detected at the background location, but then returned to background levels at the downstream location (Table 7.5). The mean concentrations for ammonium, radium-226, and uranium at the locations adjacent to and downstream of the site were higher than at the background location (Table 7.5). However, the mean concentrations for each of these contaminants were within the same order of magnitude, and the differences between the upstream concentrations and the adjacent or downstream concentrations ranged from approximately 3 to 48 percent. For purposes of this risk assessment, the mean concentrations for the contaminants of potential concern were compared to available surface water quality criteria. These comparison criteria are state of

Table 7.5 Comparison of contaminants of potential concern in the Colorado River with available water quality values, Grand Junction UMTRA Project site, Grand Junction, Colorado

Contaminant of potential concern	Upstream of the site	Adjacent to the site	Downstream of the site	Aquatic life water quality value[a]	Water concentration protective of livestock[b]
	Location ID 423	Location IDs 424, 425, 428	Location ID 426		
Ammonium	0.13	0.25	0.25	NA	NA
Copper	0.0098	0.013	0.0062	0.033[c]	0.5
Iron	4.6	6.2	3.1	1.0	NA
Radium-226 (pCi/L)	0.93	1.4	1.6	5.0	NA
Uranium	0.0047	0.0049	0.0062	5.6[c]	NA
Vanadium	0.019	0.027	0.015	NA	0.1

[a] Value obtained from the Basic Standards and Methodologies for Surface Water, 3.1.0 (5 CCR 1002-8), Colorado Department of Health, Water Quality Control Commission (CDH, 1991), unless specified otherwise. These values are standards protective of aquatic life via chronic exposure.

[b] From EPA (1972), unless specified otherwise.

[c] Water hardness-related state standard (CDH, 1991). Criterion presented was calculated using the mean hardness (332 mg/L) determined from concentrations of calcium and magnesium in the Colorado River.

All concentrations reported in milligrams per liter from unfiltered samples.
NA—not available.

Colorado water quality standards for the protection of freshwater aquatic life via chronic exposure (CDH, 1991).

A comparison of the historical surface water quality data with water quality standards indicates that for those contaminants where standards are available, none of the concentrations exceed their respective standards upstream, adjacent to, or downstream of the site (Table 7.5). Water quality standards or criteria for aquatic life are not available for ammonium and vanadium. When it is considered that there is no statistically significant difference between the upstream and downstream concentrations for these metals, it is unlikely that these concentrations represent an ecological concern or that they can be attributed to impacted ground water from the alluvium. However, continued monitoring of the river is recommended.

Colorado River sediments

Surficial sediment samples (0 to 4 inches [0 to 10 cm]) were collected on September 22, 1993, from the same locations at which surface water samples were collected (Figure 3.2). Prior to this sampling event, no sediment samples had been collected near the site. The sediment samples were analyzed for molybdenum, selenium, strontium, sulfate, and uranium.

There are no established state or federal sediment quality criteria (SQC) for the protection of aquatic life for the constituents at this site (EPA, 1988).

The EPA is evaluating a methodology based on a three-phase sorption model for free metal ion activity and is assessing its applicability for determining the bioavailable fraction within sediments (EPA, 1989c). Currently, a number of other predictive models and methods are being investigated for metals, but no single approach has been accepted to adequately develop sediment-based metals criteria (Shea, 1988; Chapman, 1989; EPA, 1989c; NOAA, 1990; DiToro et al., 1991; Burton, 1991). Therefore, only a qualitative hazard assessment of the metals detected in sediments will be presented in this risk assessment.

National Oceanic and Atmospheric Administration (NOAA) effects-based sediment quality values are available for evaluating the potential for constituents in sediment to cause adverse biological effects. However, there are no NOAA values for the constituents that have been analyzed for in the Colorado River sediments.

The concentrations detected at the adjacent and downstream locations were compared with the upstream concentrations. Selenium was not detected (less than 0.5 mg/kg) at any location (Table 7.2). Molybdenum was detected only at the upstream location. The highest concentration of strontium detected was at the upstream location. Sulfate was detected at the adjacent locations at concentrations higher than the upstream and downstream concentrations. The downstream sulfate concentration was less than that detected at the upstream location. The concentrations of uranium found in sediments at the locations adjacent to the site were similar to the upstream concentration. The concentration detected at the downstream location (4.9 mg/kg) was approximately 3 times the upstream concentration (1.5 mg/kg) (Table 7.2).

Although there are no sediment quality criteria or guidelines for these constituents, it is unlikely the detected concentrations represent a hazard to aquatic life in the Colorado River because the levels found at locations adjacent to and/or downstream of the site are less than, or similar to, levels found at the background (upstream) location. Because of the limited sediment quality data base (one sampling round), it is not possible to determine whether the constituent concentrations detected at the downstream locations represent an increase (above background levels) related to contaminant releases from the site or whether they are due to analytical variability. In addition, the detected concentrations for each constituent are within ranges reported in naturally occurring soils and other surficial materials in the United States (USGS, 1984). For example, uranium is reported to range from 0.68 to 7.9 mg/kg in the western United States (west of the 96th meridian).

Many of the contaminants identified as exceeding background ground water quality have not been measured in sediments from the Colorado River. Thus, the potential for these other contaminants to represent a hazard to aquatic life cannot be evaluated.

7.5 POTENTIAL IMPACTS TO LIVESTOCK

Contaminated alluvial ground water is believed to be discharging to the Colorado River near the former processing site. The potential exists for livestock to drink water from the Colorado River.

Ingestion by livestock of vegetation that may have bioconcentrated contaminants from alluvial ground water is a potential pathway. However, without additional data (for example, actual plant tissue concentrations or exposure and toxicity information for livestock), it is difficult to evaluate this exposure pathway.

To evaluate the potential impact to livestock that might drink out of the Colorado River near the site, the detected concentrations were compared to approximate drinking water concentrations considered to be protective of livestock (refer to Table 7.5). Guidelines are available for three of the seven contaminants of potential concern, and a comparison of these guidelines to the surface water concentrations suggests that livestock could use the Colorado River as a source of drinking water.

While the likely source of water for any stock watering near the site would be the Colorado River because of its proximity, it is possible that ground water could be used in the future to provide water for a livestock watering pond. To evaluate the potential impact to livestock in this future hypothetical scenario, the mean ground water concentrations for the contaminants of potential concern were compared to approximate drinking water concentrations considered to be protective of livestock (EPA, 1972) (refer to Table 7.4). The comparison water quality guidelines for fluoride, sulfate, and vanadium are exceeded by the mean ground water concentrations, while the mean concentrations for arsenic, boron, cobalt, copper, and zinc are below the guidelines. The effects reported for fluoride and vanadium at the concentrations detected in ground water at the site range from tooth mottling in cattle (for fluoride) to accumulation in certain organs of chickens (for vanadium) (EPA, 1972; NAS, 1974). The concentrations of TDS and sulfate detected in ground water at the site are elevated to the point that adverse effects to cattle would occur. The TDS concentrations in ground water at the site range from approximately 5300 to 8100 mg/L and average approximately 7000 mg/L. The National Academy of Sciences (NAS) has developed a guide for TDS in drinking water for livestock and poultry (NAS, 1974). Levels above 7000 mg/L are not considered acceptable because of potential toxic effects for young cattle or cattle that are pregnant or lactating.

Sulfate was detected in ground water at a mean concentration of 3800 mg/L and a maximum of 4900 mg/L. These levels are above the value (1000 mg/L) reported to cause diarrhea in cattle (National Research Council, 1971; Church, 1984) and are within the range reported in the literature to cause more serious effects. Although the levels reported in the literature are somewhat conflicting, adverse effects including weight loss, sulfhemoglobinemia, incoordination, convulsions, and death have been reported in cattle chronically exposed to sulfate in drinking water at concentrations as low as 2200 mg/L (McKee and Wolf, 1963; EPA, 1972; NAS, 1974).

No livestock drinking water guidelines have been reported for the remaining contaminants of potential concern. However, the available information suggests that the use of ground water as a source of drinking water for livestock would be hazardous due primarily to sulfate and TDS.

7.6 LIMITATIONS OF THE ECOLOGICAL RISK ASSESSMENT

The qualitative evaluation of potential ecological risks presented here is a screening level assessment of the risks associated with potential exposure of plants and animals to contaminated ground water, surface water, and sediment at the Grand Junction site. Sources of uncertainty in any ecological assessment arise from the monitoring data, exposure assessments, toxicological information, and the inherent complexities of the ecosystem. In addition, methods of predicting nonchemical stresses (for example, drought), biotic interactions, behavior patterns, biological variability (differences in physical conditions, nutrient availability, etc.), and resiliency and recovery capacities are often unavailable. In general, limitations for the Grand Junction ecological risk assessment include the following:

- Only a small amount of ecological data were collected during this screening.

- Little is known about site-specific intake rates for wildlife or amounts of contaminants taken up by plants. General literature values were used in many cases.

- Only limited ecotoxicological reference data are available.

- Considerable uncertainty is associated with the toxicity of mixtures of contaminants.

7.7 SUMMARY

Surface water data from the Colorado River near the site indicate the presence of slightly higher concentrations for six constituents (ammonium, copper, iron, radium-226, uranium, and vanadium) at the adjacent and/or downstream locations relative to the background location. However, the differences are not statistically significant and none of the constituent concentrations exceeded available state water quality standards. This suggests that site-related contamination has not adversely affected the water quality of the Colorado River.

A limited data set currently exists of the sediment quality in the surface water bodies near the site. The data from the Colorado River suggest that the site is not acting as a source of sediment-bound metals.

Potential exposure to livestock drinking from the Colorado River near the site was evaluated. A comparison of available livestock drinking water quality values with concentrations detected in the river suggests that livestock could use this surface water as their sole drinking water source without adverse health effects.

Based on available data and criteria, no ecological threat exists to plants at harvestable maturity that may have roots in contact with soil saturated with the most contaminated ground water in the alluvial aquifer. This ground water would not be suitable for continuous long-term use as irrigation water for crops due to cobalt, fluoride, iron, manganese, molybdenum, vanadium, and zinc. Water from the most contaminated wells in this aquifer would not be suitable as a source of water for fish to live in. This ground water would also not be suitable as a long-term source of drinking water for livestock due to the adverse effects, including death, associated with elevated levels of sulfates and TDS.

The potential for the contaminants of concern detected in media at the site to represent a food chain hazard to terrestrial and aquatic wildlife (via bioaccumulation and biomagnification) is considered low, based on available surface water, sediment, and ground water data. However, no tissue analysis from biota (for example, invertebrates and plants) has been conducted.

Insufficient water quality and sediment quality values were available to allow a comprehensive evaluation of the impact of surface water, sediments, and contaminated ground water on ecological receptors. However, based on available data, there is no evidence that the surface water and sediments of the Colorado River near the site have been affected by the former milling activities.

8.0 INTERPRETATION AND RECOMMENDATIONS

8.1 RISK SUMMARY

The UMTRA Project is required by the UMTRCA to protect public health and the environment from radiological and nonradiological hazards associated with the uranium mill sites. This baseline risk assessment was conducted on the Grand Junction site to evaluate the presence of these hazards.

There is no current use of ground water at the site or in the vicinity. It is unlikely that ground water will be used in the future because of the existing public water supply system; however, the most contaminated wells at the processing site were used in this risk assessment, as a conservative measure, to evaluate potential future use of ground water. This assessment is conservative because the contaminant concentrations are expected to decrease over time due to removal of the source of contamination (that is, the tailings), and because the contaminant concentrations decrease with distance from the site due to dilution and dispersion. Health risks would be associated with potential exposures from drinking contaminated ground water at the site.

If ground water at the processing site were ingested, serious adverse health effects would result from potential exposures to sulfate, manganese, and fluoride. Chronic ingestion of ground water containing the concentrations of cadmium, iron, and arsenic detected at the site would be associated with mild toxicity. Excess lifetime cancer risks associated with drinking water exposure to both median and maximum concentrations of arsenic in contaminated ground water are at levels that exceed the NCP guideline of 1 in 10,000 (the median concentration is 3 in 10,000 and the maximum concentration is 4 in 1000). The excess lifetime cancer risks associated with drinking water exposure to the median and maximum concentrations of either uranium or radium-226 are at levels below the NCP guideline of 1 in 10,000.

Use of contaminated ground water from a potential future well at the site for irrigating crops or gardens is not anticipated to result in human health risks. Adverse human health effects would not be expected following ingestion of garden produce watered with the contaminated ground water. However, the water would not be a suitable source for long-term irrigation due to toxic effects on plants following buildup in the soil.

Based on available data, ingestion of fish from the Colorado River near the site is not expected to result in adverse human health effects.

The contaminated ground water at the site would not be acceptable as a source of water for fish to live in or as a source of continuous irrigation water for agricultural crops. In addition, the ground water would not be suitable as a long-term source of drinking water for livestock due to the adverse effects, including possibly death, associated with sulfate and TDS.

The available surface water and sediment data from the Colorado River near the site suggest that contaminated ground water from the site has not adversely affected the surface water and sediment quality. None of the contaminant concentrations detected in the surface water exceeded the available water quality standards or were statistically elevated above background concentrations. No sediment quality values are available to evaluate the potential for adverse effects. However, the concentrations detected in sediments adjacent to and downstream of the site were either less than or similar to background concentrations.

8.2 LIMITATIONS OF THIS RISK ASSESSMENT

The following limitations to this evaluation of health risks should be noted:

- This document evaluates risks associated with exposures only to inorganic contaminants of ground water at the UMTRA site near Grand Junction. Potential organic contaminants (those few related to uranium processing) have not been considered.

- In general, the results presented in this document are based on filtered (0.45-micrometer) water samples. The effect of filtration differs for different elements. Although the difference on UMTRA Project sites is usually not large, filtered samples can have somewhat lower or equal concentrations than unfiltered samples for some constituents. Constituents in suspension may be lost with filtration but can still produce toxic effects if ingested and broken down in the acidic environment of the stomach.

- The toxicity of any contaminant varies from person to person. For example, normal variability in biochemical factors among individuals, differences in medical history, previous exposure to toxicants, and dietary and exercise habits can all affect susceptibility to chemical toxicity. In presenting ranges of exposures that can produce toxic effects, this assessment tries to emphasize that variability. However, it is not possible to account for all sources of variability and still present useful and meaningful analyses. Cases in which specific subpopulations of individuals are known to be more sensitive to toxic effects of given constituents have been noted. Using ranges for expected toxic effects provides the reader with a better understanding of the likelihood that toxicity will occur.

- To assess toxicity, standardized reference values developed by agencies such as the EPA are used to determine plant uptake and toxic effects in humans. These reference values themselves have limitations, including the following:

    - Toxicity, uptake, and bioconcentration data are not available for all constituents elevated above background levels at the site.

  - In some cases, data obtained from laboratory animal testing at exposure doses different from those expected at the site were used to determine toxicity. The relationship between dose and response is not always linear, and humans do not always exhibit the same responses as animals.

  - Data used to determine toxicity are generally based on exposure to only the constituent of concern. In reality, exposures generally occur simultaneously to several chemicals. The interactive effects of multiple constituents and the impact of these interactions on expected toxicity generally cannot be accurately assessed from existing data.

- Considerable effort has been directed at determining plume movement and placing monitor wells in locations that capture maximal contamination. Nevertheless, physical systems and models used to determine contaminant plume migration vary widely and may result in well placements that do not measure the highest contaminant concentrations or determine the fullest extent of plume impact.

- Variability can be introduced through sampling and analytical processes. However, the data at UMTRA Project sites have been collected over many years and subjected to rigorous quality assurance procedures. The use of multiple samples introduces high confidence in the reliability and validity of the collected data.

- The drinking water pathway is considered the major determinant of exposure in this assessment. Although other pathways were screened and determined not to contribute significantly to the total exposure, the additivity of exposure from these pathways should be kept in mind. When a measurable contribution from other pathways could increase expected exposure significantly enough to alter the predicted toxicity, the alternate source contribution is noted in Section 6.0.

By presenting ranges of toxic effects, summaries of available data on health effects and interactions, and outlines of potential limitations, this document provides a reasonable interpretation of potential health risks associated with ground water contamination at this site. This assessment presents both contamination and risk as accurately as possible, based on available data, and conveys areas of uncertainty.

8.3 GROUND WATER CRITERIA

In 1983, the EPA established health and environmental protection standards for the UMTRA Project (40 CFR Part 192 (1993)); in 1987, the EPA proposed revised ground water standards (52 FR 36000 (1987)). The UMTRA Project is required to adhere to the 1987 proposed ground water standards until final standards are published. The ground water standards consist of 1) ground water protection standards to evaluate disposal cell performance, and 2) ground water cleanup standards for existing contamination at processing sites. The contaminants that have a proposed MCL are summarized in Table 8.1. Because an MCL is not established for every contaminant, the proposed standard requires meeting background levels for those contaminants that do not have an MCL.

While the proposed standards apply only to the UMTRA Project, the EPA has also published drinking water health advisory levels for both long- and short-term exposures. These advisories are shown in Table 8.1.

8.4 RISK MITIGATION MEASURES

Because there is a potential for serious health effects following even short-term use of sulfate-contaminated ground water at the former processing site, this section presents possible ways to restrict access to ground water so as to mitigate risks.

Institutional controls are defined in the proposed ground water standards for the UMTRA Project as mechanisms that can be effectively used to protect human health and the environment by controlling access to contaminated ground water.

Although the proposed standards refer to institutional controls for long periods of time (up to 100 years during natural flushing), this concept can also be applied to short-term or interim restriction of access to ground water. Since not all 24 UMTRA Project sites can be evaluated simultaneously, interim institutional controls may be needed before remedial action decisions are made for individual sites.

Well permits

All of the Colorado UMTRA Project sites are located on the Colorado West Slope and are outside the designated ground water basins. Construction of a well in Colorado outside the designated basins requires a written application to the state engineer for a permit to construct a new well. Designated basins are isolated hydrogeologic areas where ground water use is stringently evaluated based on the demands for water rights. The state engineer is required to act on applications for new well permits within 45 days after their receipt. If a well would affect existing water rights or if an applicant wants to establish a legal right, adjudication would likely be required prior to the permit being granted.

Ground water quality

The Colorado Department of Health (CDH) is the state agency responsible for setting water quality standards. Within the CDH, the Water Quality Control Commission is responsible for adopting water quality standards and classifications for waters in Colorado.

The state of Colorado's proposed ground water quality standards require ground water to be free of substances in concentrations shown to be "carcinogenic,

Table 8.1 Concentration limits of constituents

Constituent	MCL (mg/L)	Health advisories 10-kg child, 10-day (mg/L)	Health advisories 70-kg adult, lifetime (mg/L)
Chemicals (inorganic)			
Antimony	-	0.015	0.003[a,b]
Arsenic	0.05[a]	-	-
Barium	1.0	-	2
Boron	-	0.9	0.6[a,b]
Cadmium	0.01[a]	0.04	0.005[a,b]
Chromium	0.05[b]	1.0	0.1[b]
Cobalt	-	-	-
Copper	-	-	-
Fluoride	-	-	-
Iron	-	-	-
Lead	0.05	-	0.015
Manganese	-	-	-
Mercury	0.002	-	-
Molybdenum	0.1[a,b]	0.08	0.04
Nickel	-	1.0	0.1[a,b]
Nitrate	44[a,c]	44[d]	-
Selenium	0.01[a,b]	-	-
Silver	0.05	0.2	0.1
Strontium	-	25	17
Sulfate	-	-	-
Thallium	-	0.007	0.0004
Vanadium	-	0.08[a,b]	0.02
Zinc	-	6.0[a]	2
Radionuclides			
Lead-210	-	-	-
Polonium-210	-	-	-
Radium-226/-228	5 pCi/L[a]	-	-
Thorium-230	-	-	-
Uranium (U-234/-238)	30 pCi/L[a,b] (0.044 mg/L)	0.03 mg/L[e]	0.1 mg/L[e]

[a] Exceeded in plume wells.
[b] Exceeded in background wells.
[c] Equal 10 mg/L nitrate as nitrogen.
[d] Under review.
[e] Proposed values, under review; expected revision 1995.

mutagenic, teratogenic or toxic to human beings and/or a danger to public health, safety, or welfare." (CDH, 1990).

The state engineer is authorized to enforce the state water quality standards. However, the state engineer does not have jurisdiction to deny a permit for drilling a new well based on water quality, as private domestic well water quality is not regulated by the state. The state engineer's office can issue a warning to well users if the well is placed in a known contaminated aquifer. Well water to be consumed by 25 or more people does have to meet state water quality standards, and use can be restricted by the CDH, Water Quality Control Division, Drinking Water Section.

Land and water use restrictions

Development within the Grand Junction city limits consisting of a conditional use permit, rezoning, or a subdivision is subject to review and approval by the Grand Junction Planning Commission and/or the Grand Junction City Council. All other development is reviewed and approved by the City Community Development Department. With the support of other city agencies, the City Community Development Department reviews development plans for compliance with the city of Grand Junction Zoning and Development Code (Grand Junction City Council, 1989).

The Grand Junction Zoning and Development Code requires that all potable water supply systems, whether individual or public, comply with state and county health departments as well as all city or other applicable regulations. The code also requires that all development be served by the city water treatment and distribution system and empowers the city utilities director to grant exceptions if the requirement is deemed unreasonable or impracticable.

Access to river water

Swimming in the Colorado River is not prohibited; however, it is not encouraged. The state requires a fishing license that is valid for 1 year, and tubing and canoeing are allowed on the river. A new riverfront park with a boating concession is planned near the processing site. The concession would be under contract to the state. River water will be used to irrigate the park landscaping, and municipal water will be used for drinking water. A yearly state parks pass will be required for vehicular access to the park, and an entrance station is planned to control vehicular traffic. There will be no control of pedestrian or bicycle users.

8.5 FUTURE SITE ACTIVITIES

For the UMTRA Ground Water Project, ground water and potential surface expression points should continue to be monitored until detailed characterization of the site ground water is complete.

Although recent water use surveys have indicated that shallow ground water is not used for domestic purposes or any other purpose, the Grand Junction processing site and its vicinity should continue to be periodically monitored to identify any new potential uses.

Monitoring of the Colorado River, for the contaminants of potential concern identified in this risk assessment, should continue to further evaluate the potential impact of contaminated alluvial ground water on surface water and sediment quality in the river.

8.6 RECOMMENDATIONS

In general, the proposed ground water standards consisting of MCLs or background concentrations are sufficient to protect human health and the environment. However, in some cases, a risk assessment may identify site-specific factors that suggest these standards may be either too restrictive or not restrictive enough. When standards are too restrictive, there may be no potential for exposure, and a less restrictive alternate concentration limit (ACL) may be sought. In other cases, the standards may not be sufficiently protective (for example, if there are many contaminants near the MCL with additive or synergistic adverse health effects).

At Grand Junction, no permanent physical barrier prevents access to contaminated ground water at the former processing site. Therefore, ACLs are not likely to be justified for those constituents with MCLs. However, for constituents that exceed background levels and do not have MCLs, this assessment suggests that background levels are more restrictive than necessary. This includes contaminants that were demonstrated to be at concentrations well below adverse health effect levels, such as copper. ACLs should be sought for these contaminants. Because there are no permanent physical barriers, measures should be implemented to restrict access to contaminated ground water to protect human health and the environment.

To develop a better understanding of the geochemistry at the Grand Junction site, the redox state of the alluvial aquifer needs to be better characterized. Also, the constituents contributing to the total organic carbon fraction of the ground water should be further evaluated.

It is recommended that additional sampling be conducted for bromide due to the limited data set, to confirm whether it is present in the plume at elevated levels.

It is recommended that additional characterization be conducted to further evaluate surface water and sediment conditions of the Colorado River and potential ecological receptors near the site.

9.0 LIST OF CONTRIBUTORS

The following individuals contributed to the preparation of this report.

Name	Contribution
C. Day	Overall document responsibility; authorship; ecological assessment
J. Lewis	Technical review
K. Smith	Statistical evaluation
T. Monks	Hydrogeology
J. Blount	Geochemistry
R. Neri Zegal	Institutional controls
J. Lewis, B. Toth	Toxicology
R. Meyers	Surface water
C. Slosberg, L. Keith	Text processing
E. Bond, T. Gagliano	Graphic design
D. Thalley	Technical editing

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