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Dugway Proving Ground Closure Plan Module 2

SWMUs 20, 164, 166, and 170

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This Closure Plan module outlines the activities required to close the four Consent Order solid waste management units (SWMUs 20, 164, 166, and 170) at Dugway Proving Ground in western Tooele County, Utah. The plan for each SWMU includes a description of the unit and its physical environment along with an assessment of the contamination found at each SWMU and the risk it poses to human health and the environment. Based on the requirements found in state of Utah Administrative Rule R315-101, no corrective action is required at any of the four SWMUs.

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LIST OF ACRONYMS AND ABBREVIATIONS

µg/L	micrograms per liter
µg/g	micrograms per gram
ACET	Acetone
Ag	Silver
Al	Aluminum
ANOVA	Analysis of variance
As	Arsenic
Avery	Avery Technical Center
B2EHP	Bis(2-ethylhexyl) phthalate
Ba	Barium
BAF	bioaccumulation factor
BAPYR	Benzo(a)pyrene
Be	Beryllium
BIS	Bis(2-ethylhexyl) hydrogen phosphite
BW	Body weight
Ca	Calcium
Cd	Cadmium
CH2CL2	Methylene chloride
CHCL3	Chloroform
CHRY	Chrysene
cm^2	square centimeters
Co	cobalt
COPC	chemical of potential concern
Cr	chromium
Cu	Copper
Ditto	Ditto Technical Center
DPG	Dugway Proving Ground
ED	Exposure duration
EF	Exposure frequency
ENDRNK	Endrin ketone
EPA	U.S. Environmental Protection Agency
EPC	exposure point concentration
ERA	ecological risk assessment
ESFSO4	endosulfan sulfate
Fe	Iron
ft	foot or feet
ft^2	square foot/feet
ha	hectares
HEAST	Health Effects Assessment Summary Table
Hg	Mercury

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HI	hazard index
HQ	hazard quotient
IMPA	Isopropylmethyl phosphonic acid
IRIS	Integrated Risk Information System
K	Potassium
kg	Kilogram
MEC6H5	Toluene
mg/kg/day	milligrams per kilogram per day
Mg	Magnesium
Mn	Manganese
msl	(Above) mean sea level
Na	Sodium
NEI	Nature and Extent Investigation
Ni	Nickel
OCP	organochlorine pesticide
Pb	Lead
PCB	Polychlorinated biphenyl
POL	petroleum, oil, and lubricants
ppb	parts per billion
ppm	parts per million
PPDDT	2,2-Bis(p-chlorophenyl)-1,1,1-trichloroethane
PYR	Pyrene
RBSL	risk-based screening level
RCRA	Resource Conservation and Recovery Act
RDX	Cyclonite/cyclotrimethylene trinitramine/ hexahydro-1,3,5-trinitro-1,3,4-triazine
RfD	reference dose
RFI	RCRA Facility Investigation
RME	reasonable maximum exposure
Sb	Antimony
Se	Selenium
SF	slope factor
SVOC	semivolatile organic compound
SWMU	Solid waste management unit
TCLP	Toxicity characteristic leaching procedure
TCR	target cancer risk
Tl	Thallium
TPHC	total petroleum hydrocarbon
UAC	Utah Administrative Code
UCL	upper confidence limit
UDEQ	Utah Department of Environmental Quality
USAEC	U.S. Army Environmental Center
UTL	upper tolerance limit

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UXO	unexploded ordnance
V	Vanadium
VOC	volatile organic compound
XYLEN	Xylenes
Zn	Zinc
°F	degrees Fahrenheit

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EXECUTIVE SUMMARY

This Closure Plan Module 2 is one of four that describe the activities required for closure of 41 solid waste management units (SWMUs) at Dugway Proving Ground (DPG) in Utah. These SWMUs are among 45 units listed in a Stipulation and Consent Order (No. 8909884) between DPG and the state of Utah, its amendment, and a second Stipulation and Consent Order (9303065). Final Closure Plan Module 1 (EBASCO 1995b) includes recommendations for administrative closure of three units (SWMUs 30, 160, and 161) that were not used to manage hazardous wastes. Module 2 (this document) addresses four units (SWMUs 20, 164, 166, and 170) for which sampling conducted through 1993 is adequate to plan closure. Closure Plan Module 3 will address 33 SWMUs that were included in additional field investigations completed during 1995. Module 4 will address SWMU 47, which is being investigated under a separate effort. No closure plans will be prepared for the remaining four SWMUs (SWMUs 27, 120, 121, and 125), which were not listed in the Second Consent Order.

Comparison of human health risk assessment results to corrective action criteria in state of Utah Administrative Rule R315-101 indicates that corrective action at the Module 2 SWMUs is not required. Ecological risks associated with organic compounds and metals above background are negligible. There are no explosive risks anticipated at these SWMUs because no unexploded ordnance (UXO) were known to have been handled or disposed of at these units. No future releases to the environment are expected from these SWMUs as no land disposal of hazardous waste or material occurred there, and no contaminated soil or residue is present at these units that poses a significant risk to human health or the environment. No groundwater monitoring at these SWMUs will be required.

For SWMU 20, the human health evaluation identified two organic constituents and two inorganic constituents as soil chemicals of potential concern (COPCs). The total cancer risk is 2.5×10^{-9} and the total hazard index (HI) is 2.5×10^{-5} under a hypothetical residential land-use scenario. Both values are well within the risk range—a cancer risk less than 10^{-6} and an HI less than 1.0 for noncancer endpoints—for which state of Utah Administrative Rule R315-101 allows risk-

based closure. The preliminary ecological risk assessment shows that the predicted HQs exceed 1.0 for the deer mouse (antimony, 2; barium, 21) and the horned lark (barium, 3). However, corrective action based on potential ecological risk predictions is not recommended due to the low magnitude of the HQ values and the degree of conservatism inherent in the risk estimates.

For SWMU 164, the human health evaluation identified five organic constituents as soil COPCs, and the ecological evaluation identified five inorganic constituents as potential soil COPCs. The risk assessment results indicated that, under a hypothetical residential land-use scenario, the total cancer risk is 2.4×10^{-8} and the HI is 4.5×10^{-4}. Both values are well within the risk range—a cancer risk less than 10^{-6} and an HI less than 1.0 for noncancer endpoints—for which state of Utah Administrative Rule R315-101 allows risk-based closure. The results of the preliminary ecological risk assessment show that the predicted incremental risk HQ values do not exceed 1.0 for the deer mouse, but do exceed 1.0 for the horned lark (chromium, 2; lead, 14). However, corrective action based on potential ecological risk predictions is not recommended due to the low magnitude of the HQ values and the degree of conservatism inherent in the risk estimates.

For SWMU 166, the human health evaluation identified four organic constituents as soil COPCs, and the ecological evaluation identified no constituents as soil COPCs. Under the hypothetical residential land-use scenario, the total cancer risk is 7.4×10^{-10} and the total HI is 2.4×10^{-5}. Both values are well within the risk range—a cancer risk less than 10^{-6} and an HI less than 1.0 for noncancer endpoints—for which state of Utah Administrative Rule R315-101 allows risk-based closure. The results of the preliminary ecological risk assessment show that there were no predicted incremental risk HQs that exceeded 1.0 for either the deer mouse or the horned lark. Corrective action based on potential ecological risk predictions is not recommended due to the very low magnitude (i.e., below 1.0) of the HQ values and the degree of conservatism inherent in the risk estimates.

For SWMU 170, the human health evaluation identified five organic constituents as soil COPCs and the ecological evaluation identified two inorganic constituent as a soil COPC. Under a

hypothetical residential land-use scenario, the total cancer risk is 1.5×10^{-3} and the total HI is 5.4×10^{-4}, which qualifies this SWMU for risk-based closure. The results of the preliminary ecological risk assessment show that the predicted incremental risk HQ values were less than 1.0 for the deer mouse but do exceed 1.0 for the horned lark (chromium, 3; lead, 3). However, corrective action based on potential ecological risk predictions is not recommended due to the low magnitude of the HQ values and the degree of conservatism inherent in the risk estimates.

Schedules of closure activities and certifications of closure are not required as there is no planned future use of these units for hazardous waste storage or disposal and there are no corrective action or closure activities planned at SWMUs 20, 164, 166, or 170. These units will be considered clean-closed upon approval of this closure plan by the Utah Department of Environmental Quality, Division of Solid and Hazardous Waste.

1.0 INTRODUCTION

This closure plan module outlines the activities required for closure of four solid waste management units (SWMUs) at Dugway Proving Ground (DPG) in Utah. It was prepared to satisfy part of the requirements for closure under a Stipulation and Consent Order (No. 8909884) issued by the state of Utah Solid and Hazardous Waste Committee on Se ber 13, 1990, and amended on December 22, 1993. Stipulation and Consent Order 3065, issued on September 30, 1994, confirmed that closure plans would be required for these units.

The Closure Plan for 40 of the 45 SWMUs listed in the Consent Order is being prepared in three modules:

Module 1: SWMUs 30, 160, and 161

Module 2: SWMUs 20, 164, 166, and 170

Module 3: SWMUs 2, 7, 9 and 9A, 14, 33, 34, 36, 37, 38, 39, 40, 42, 43, 46, 47, 48, 51, 55, 58, 59, 63-1 and 63-2, 90, 99, 124, 128, 130, 158, 162, 163, 165, 168, 169, and 190

Final Closure Plan Module 1 (SWMUs 30, 160, and 161) was issued in May 1995 (EBASCO 1995b). It included a recommendation for administrative closure of three units that were not used to manage hazardous wastes. This plan, prepared as Module 2, addresses four units (SWMUs 20, 164, 166, and 170) for which sampling conducted through 1993 is adequate to plan closure. Closure Plan Module 3 will address 33 other SWMUs for which data collection continued through 1995. One remaining SWMU (47) is being addressed by a separate effort. No closure plans will be prepared for SWMUs 27, 120, 121, and 125, because these units were not listed in Consent Order 9303065.

The objectives of Module 2 are to provide an overview of the nature and extent investigation at DPG, summarize the SWMU-specific investigations, and recommend closure activities. Because DPG is a federal installation, the closure cost estimate and financial assurance requirements of 40 CFR Part 264, Subpart H are not applicable (cf. 40 CFR 264.140(c)).

2.0 BACKGROUND AND TECHNICAL APPROACH

This section describes the history and physical setting of DPG and summarizes the results of the field investigations that have been conducted at the installation. Included in this overview are a discussion of background soil geochemistry and an outline of the methodologies used in the human health and ecological risk assessments. Supplemental information on any topic presented in this section can be found in the Final Interim Report on SWMU Closures at DPG, hereafter referred to as the Final Interim Report (EBASCO 1995a).

This document refers to three sets of SWMUs at DPG. The term "Consent Order SWMUs" refers to the 45 units listed in the original Consent Order (8909884) and the Amendment; Foster Wheeler Environmental Corporation has been tasked with developing closure plans for 40 of these. These 40 SWMUs include one incinerator, 7 treatment units, 5 impoundments, 9 landfills, 6 storage areas, and 12 operations areas that generated solid waste. Another unit listed in the Consent Order (SWMU 47) is being addressed under a separate effort. No closure plans will be prepared for SWMUs 27, 120, 121, and 125, which were not listed in the second Consent Order (9303065). The term "Module 2 SWMUs" refers to the units addressed by this closure plan module, i.e., SWMUs 20, 164, 166, and 170, which are all operations areas. SWMU 20 was originally described as a landfill, but was later found to be associated with an adjacent test facility. The term "Corrective Action SWMUs" refers to the units listed in a Resource Conservation and Recovery Act (RCRA) Part B permit with a corrective action requirement. The corrective action program is being conducted by Parsons Engineering-Science and is described in the draft final Phase I RCRA Facility Investigation (RFI) Report, DPG (Parsons ES 1995). A description of waste management practices, including waste volumes, is presented in the Installation Environmental Impact Assessment (Army 1982).

Information on activities and characteristics of each SWMU was obtained from a variety of sources. Previous environmental investigations and historical data were used to describe the purpose of each unit and the function of each subsection of the unit. This information was verified with DPG employees and former employees who worked at the specific units. Observations made during previous site visits, along with historical aerial photographs and photographs of current conditions at each SWMU, were used to describe site conditions at each

unit. Dimensions of the units were taken from as-built drawings wherever possible, and were confirmed using field measurements.

2.1 FACILITY DESCRIPTION

This section describes the physical setting of DPG, including its location, history, geology, soils, hydrology, climate, vegetation, wildlife, and demography.

2.1.1 Facility Location

DPG covers approximately 840,000 acres (or approximately 18 percent of the total land area) in Tooele County in western Utah (Figure 2.1-1). The entrance to DPG is situated 56 miles due west of Provo, at the southern tip of Skull Valley. DPG is approximately rectangular in shape and is bordered to the northeast by the Cedar Mountains and to the north-northwest by Wendover Air Force Range. The installation includes both mountains and valleys, but the majority of the installation lies within a large, flat, sparsely vegetated area that extends westward into the southern reaches of the barren salt flats of the Great Salt Lake Desert (USATHAMA 1988a).

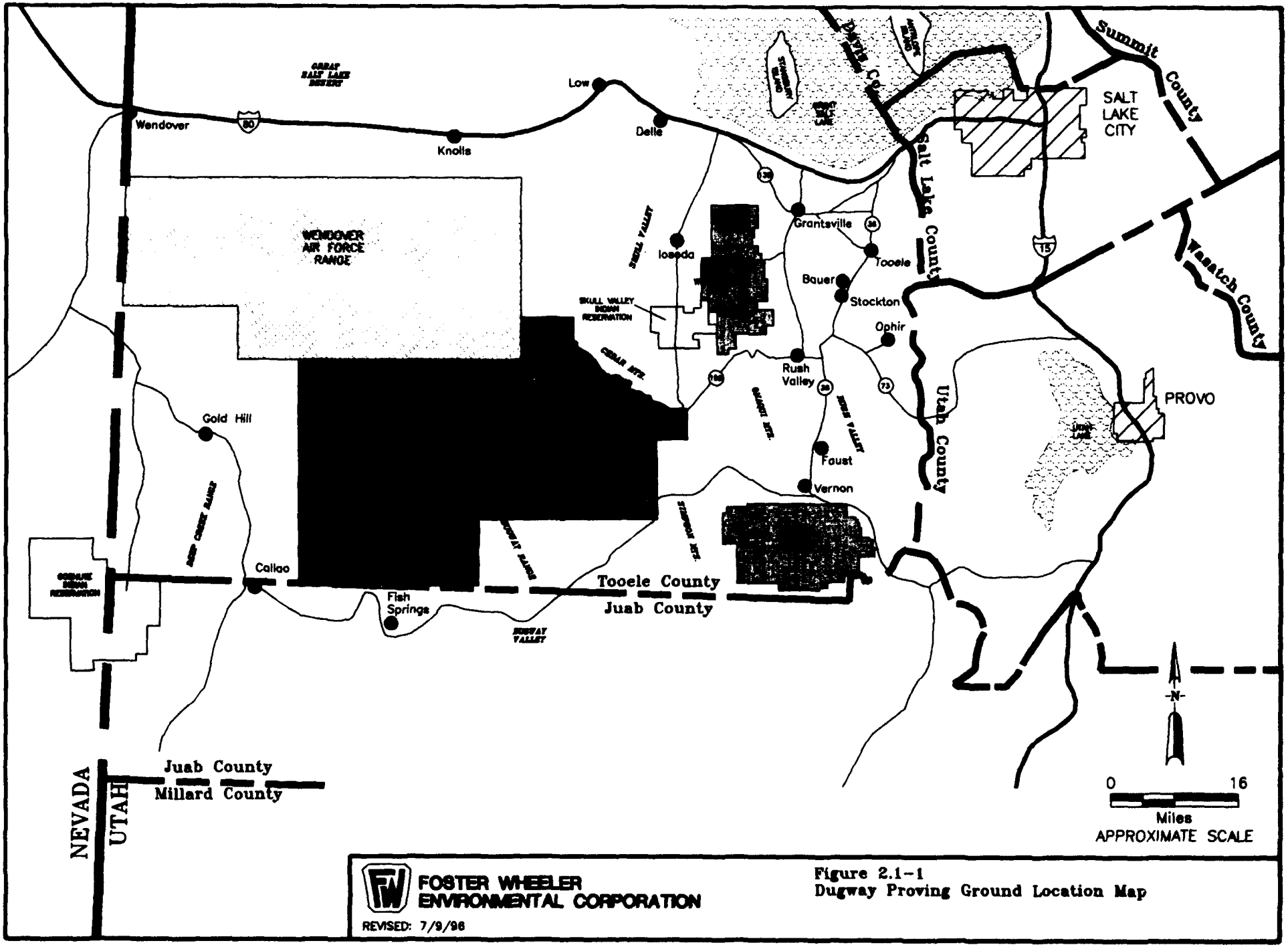
SWMU 20 is located on the northwest side of Camels Back Ridge in the southeastern portion of DPG. SWMUs 164 and SWMU 166 are located in the east-central portion of DPG on the northeastern and eastern side of Avery Technical Center (Avery), respectively. SWMU 170 is located in the southwestern portion of English Village, near the eastern perimeter of DPG (Figure 2.1-2).

2.1.2 Facility History

DPG was activated on March 1, 1942, with military weapons testing commencing shortly thereafter. Limited testing of biological warfare materials was initiated in 1945. DPG was deactivated in January 1947 and became a satellite post of the Deseret Chemical Storage Depot (now called Deseret Chemical Depot).

On July 1, 1950, DPG was designated a Class II installation under the jurisdiction of the Chief Chemical Officer. The Chemical Warfare Division and the Biological Warfare Division were

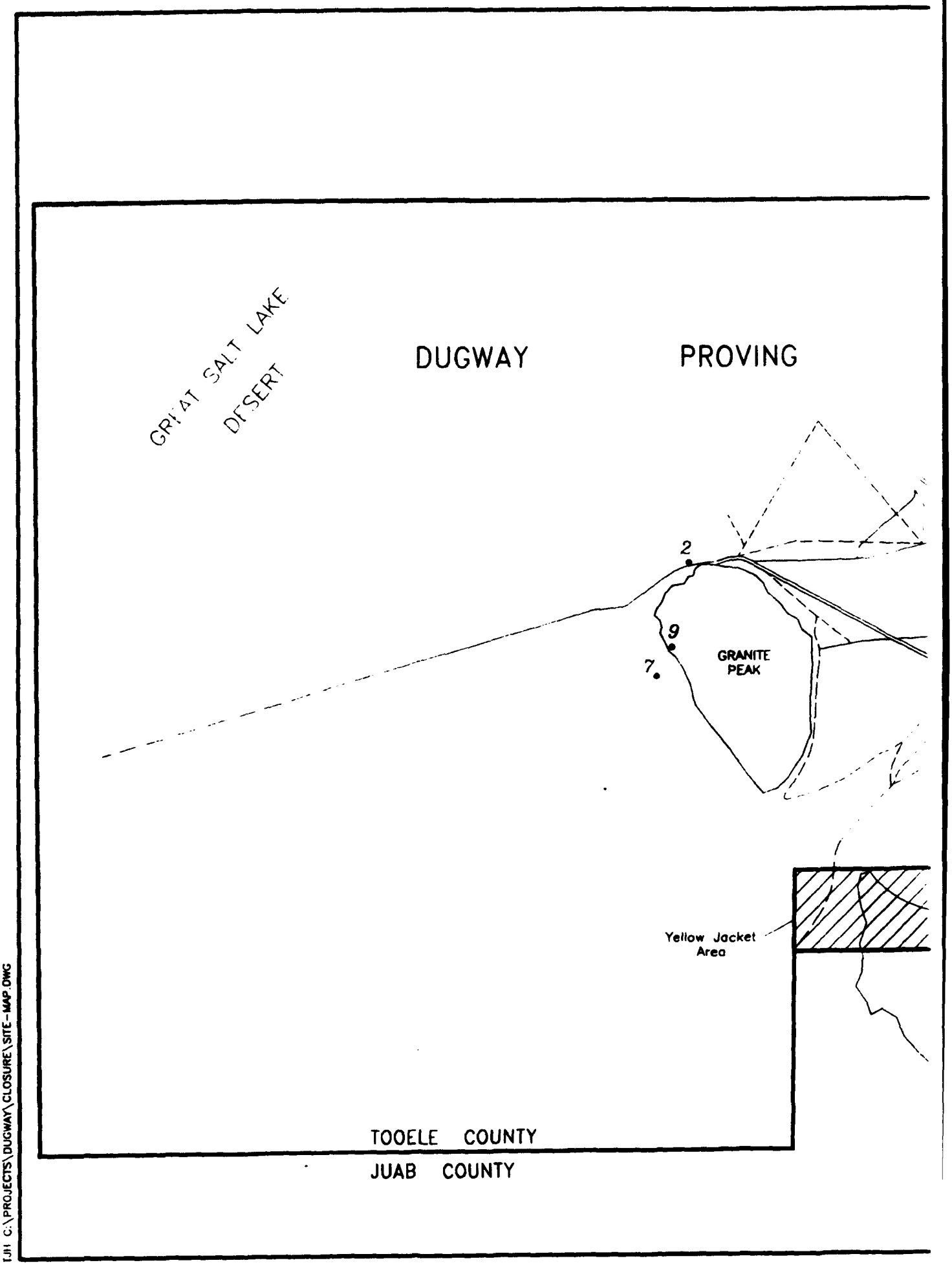
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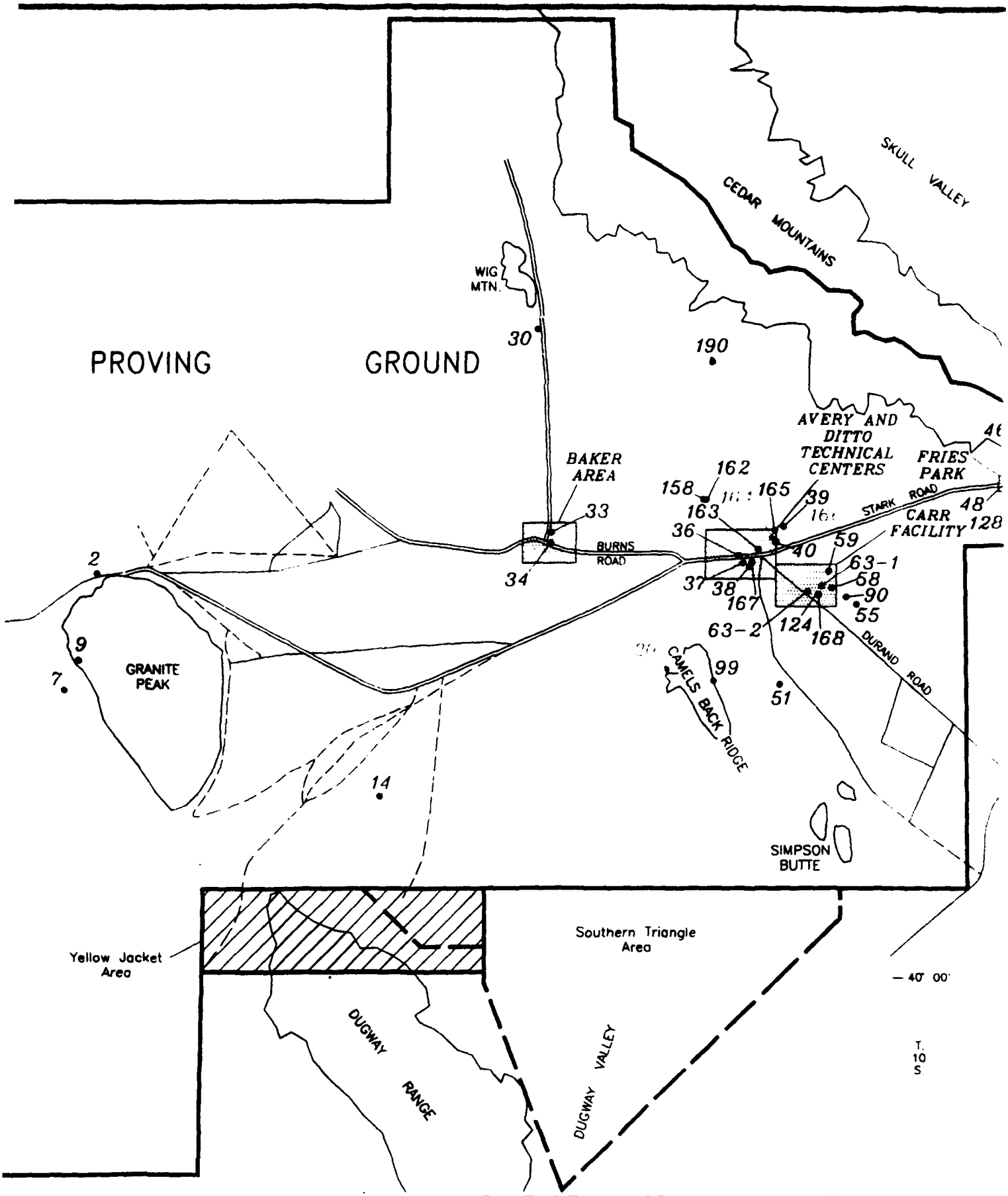
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Figure 2.1-1
Dugway Proving Ground Location Map



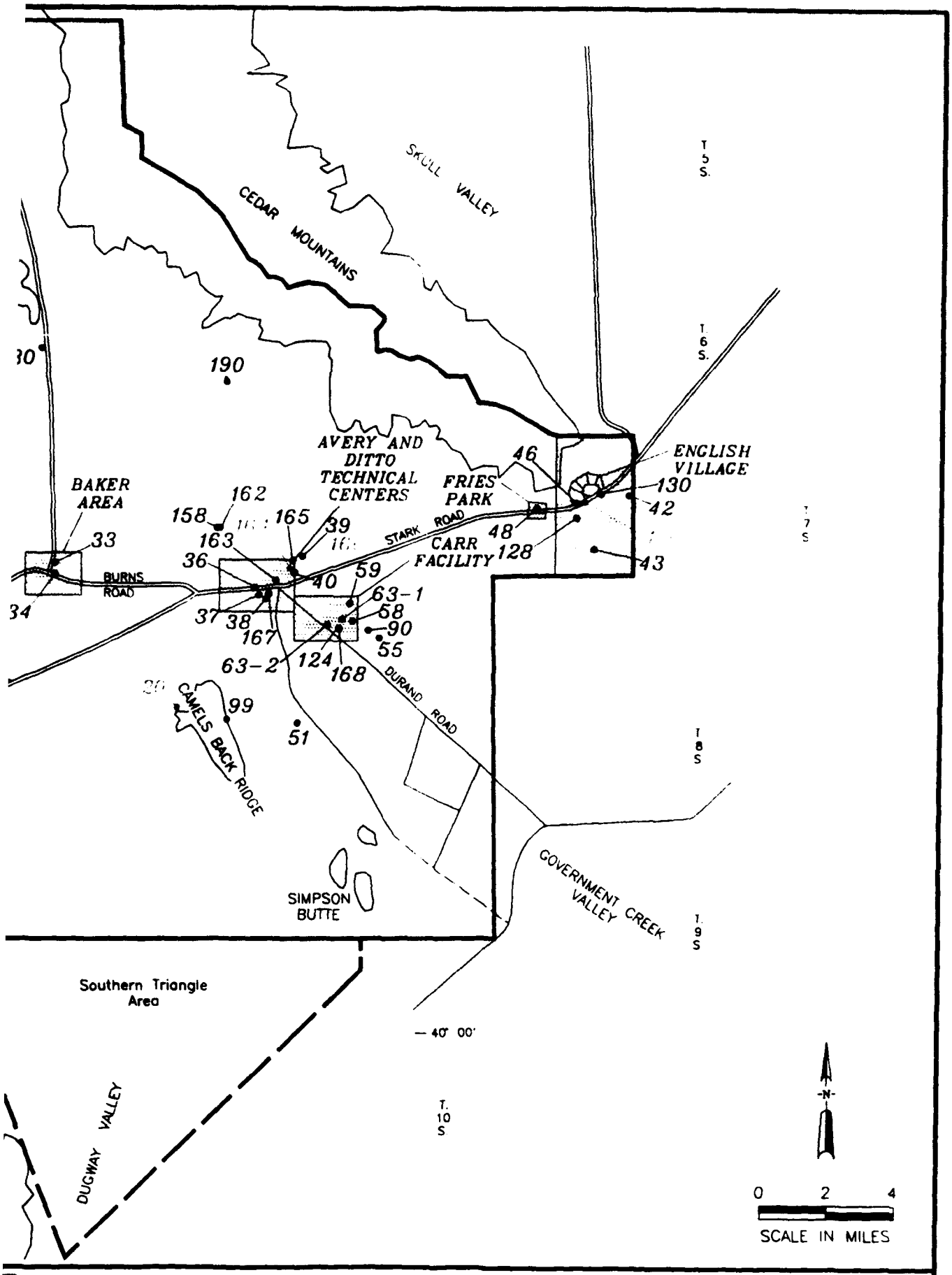
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Figure 2.
Site Loca
SWMUs 20



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Figure 2.1-2
Site Location of DPG and SWMUs 20, 164, 166, and 170

activated at Ditto Technical Center (Ditto) in January 1951. In 1952, Post Headquarters was moved from Ditto to English Village, and the Biological Warfare Division was moved from Ditto to Baker Laboratory (EBASCO 1995a).

DPG was combined with the Deseret Test Center, which also was a facility associated with chemical and biological testing, on July 1, 1968. The Deseret Test Center, which formerly occupied the western half of DPG, was decommissioned in June 1973. At that time, responsibility for testing chemical and biological weapons for the Department of Defense was assigned to the Department of the Army, and DPG was assigned to the Commanding General, Testing and Evaluation Command, on October 11, 1973 (EBASCO 1995a).

DPG includes several geographically distinct activity areas (Figure 2.1-2). In addition, there are scattered test and range areas that are not associated with developed activity areas. The names and uses (both past and present) of the activity areas are described below.

Since Avery and Ditto are located in close proximity, the descriptions of these two areas are presented together. Avery is located to the northeast of Ditto, approximately 3 miles northeast of Camels Back Ridge (Figure 2.1-2). This area, formerly known as Able Area, was used in the 1950s for radiological research. Currently, Avery is used primarily for administration; it is secondarily used for maintenance of generators and nickel-cadmium batteries. The U.S. Air Force also utilizes a portion of Avery. Ditto, known as Dog Area when DPG was activated, was the original DPG headquarters that included barracks for DPG personnel. Ditto now contains facilities for operations administration, material testing, decontamination, meteorology, photography, instrument calibration and storage, and chemical, ecological, and epidemiological laboratories.

The Baker Area is located on Burns Road, approximately 7 miles due west of the Avery and Ditto areas. This area contains biological defense facilities, limited animal-rearing facilities, holding facilities, and decontamination buildings.

The Carr Facility is located on Durand Road, approximately 2 miles southeast of Ditto. Carr, formerly known as the Toxic Gas Yard and Charlie Area, was used for storage and handling of chemical agent containers and munitions. Facilities for munitions assembly, handling, and storage, and equipment for environmental testing are currently located there.

English Village, originally known as Easy Area, is located near the eastern border of DPG, at the southern tip of the Cedar Mountains. Community housing, a school, recreation facilities, the clinic, and some administrative offices are located there.

Fries Park is located on Stark Road, approximately 1 mile west of English Village. This area is used for logistic-supply activities and formerly included a mobile home park.

2.1.3 Geology and Soils

DPG lies within the Basin and Range Province of the western United States. This area is characterized by a series of longitudinal, block-faulted mountain ranges and intervening down-dropped basins. The basins are partially filled with sediments derived from erosion and volcanism in the adjacent upthrown ranges of sedimentary, metasedimentary, and igneous rock.

DPG is bordered to the northeast by the Cedar Mountains, to the east by the Onaqui Mountains, and to the south by a series of ranges and valleys, the closest of which is the Dugway Range. The Deep Creek Range lies to the west and marks the western boundary of the Great Salt Lake Desert. Within the confines of the installation, the extensive flat plains are broken by Granite Peak, Sapphire Mountain, Camels Back Ridge, Simpson Butte, and Wig Mountain. Basins in the area include Government Creek Valley and Skull Valley. Elevations range from 4,225 feet (ft) above mean sea level (msl) on the desert floor in the northwestern part of DPG to 7,068 ft msl at the summit of Granite Peak in the center of DPG (USGS 1962). With the exception of English Village and Fries Park, most of the developed areas of the installation are located within the extensive flat areas of the Dugway Valley and the Great Salt Lake Desert.

Most of the relative movement between the basins and ranges occurred during the late Miocene and early Pliocene epochs, 4 million to 7 million years ago. The mountain blocks within and

adjacent to DPG consist of sedimentary, metasedimentary, and igneous rocks ranging in age from Precambrian to Tertiary (Figure 2.1-3). In the younger sediments and volcanic rocks, faulting is concentrated along the mountain fronts. This movement ended by late Pliocene time, as evidenced by undisturbed late Pliocene and younger strata.

Sediments of Tertiary to Quaternary age were deposited in the basin by streams and wind. Since late Pleistocene time, the basin has been occupied intermittently by Lake Bonneville, an immense lake that covered much of what is now western Utah and adjacent parts of Nevada and Idaho. Geologic indicators show that Lake Bonneville reached a maximum elevation of 5,135 ft msl, which indicates nearly complete inundation of the DPG area. Changes in sedimentary environments caused by fluctuations in lake level have resulted in a mixture of lithologies in the subsurface.

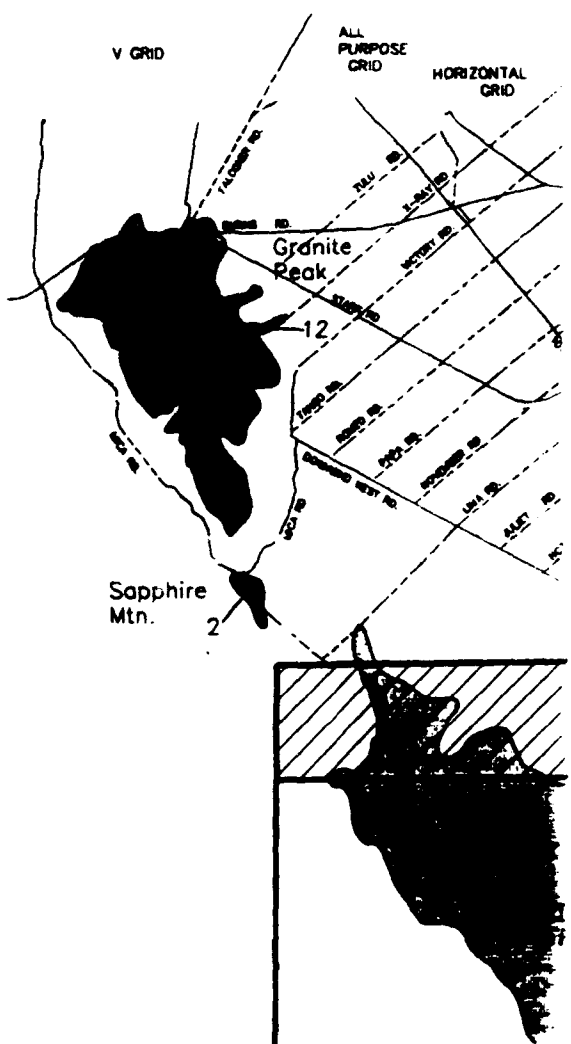
The basin sediments within DPG, consisting of clay and marl, sand and gravel, tuff, and conglomerate, are believed to reach thicknesses of several thousand feet. Younger alluvial and eolian deposits locally overlie Lake Bonneville sediments. In general, the sediments at English Village, which is located adjacent to the Cedar Mountains, are coarser-grained than the basinal sediments.

Soils at DPG are divided into four major types: lacustrine sediments, alluvial deposits, active and inactive dunes, and alpine colluvium and weathered bedrock or residual soils (Figure 2.1-4). SWMU 20 is located on residual soil, SWMUs 164 and 166 are located on lacustrine sediments, and SWMU 170 is located on alluvial sediments. In general, soils at DPG are thin and poorly developed as a result of the dry climate, sparse vegetation, and high evapotranspiration potential typical of this region. High evapotranspiration potential produces a negative water balance and has resulted in the deposition of soluble salts in near-surface horizons.

2.1.4 Surface Water and Groundwater

The general direction of surface drainage at DPG is to the northwest, onto the Great Salt Lake Desert, and then into the Great Salt Lake (Figure 2.1-5) (Gates and Kruer 1981). The streams that cross DPG flow intermittently. Most of the runoff that reaches the low valleys or basins

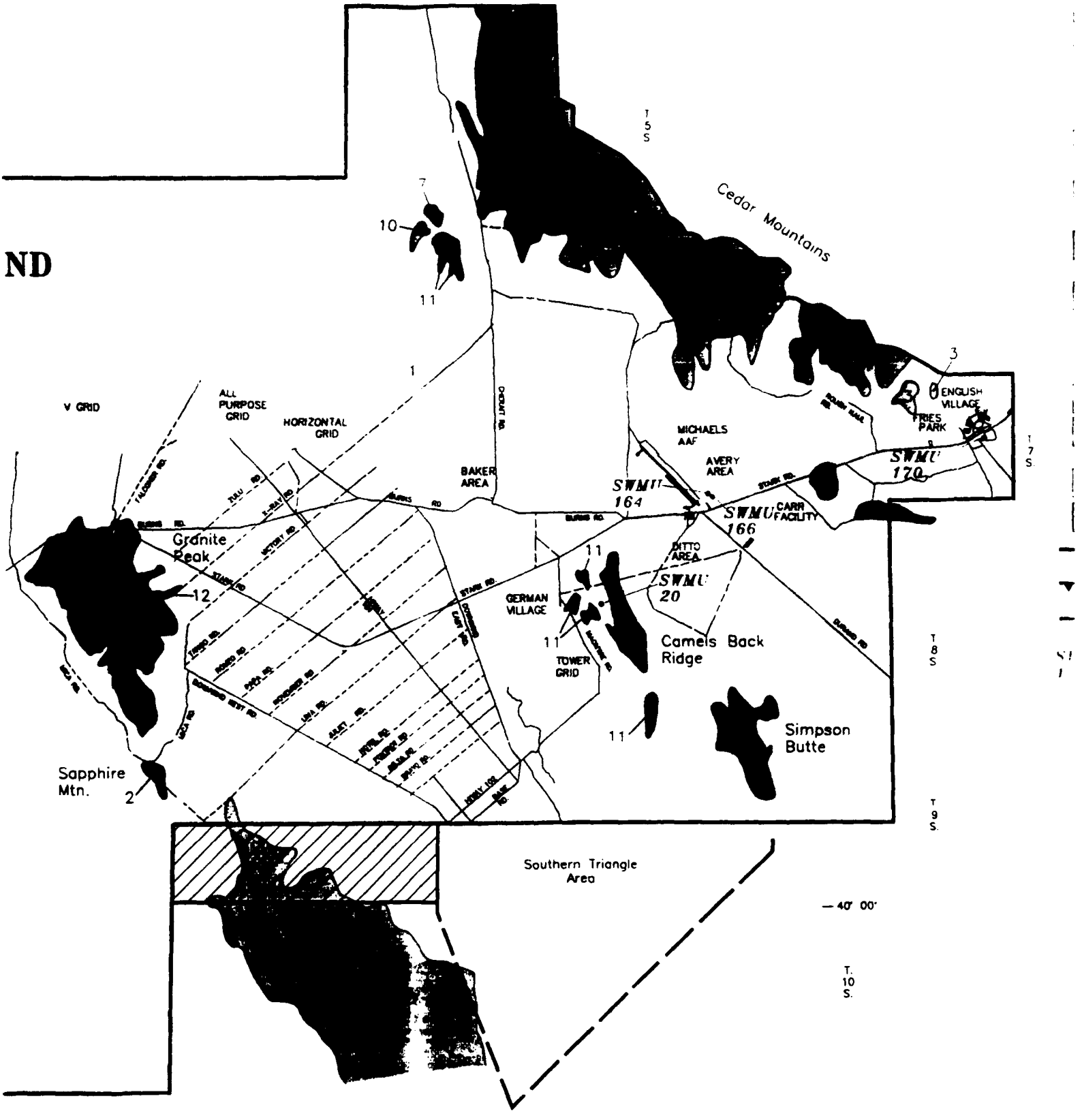
DUGWAY PROVING GROUND



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SOURCE: W. J. Moore, et al, 1979

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Figure 2.1
Generalize

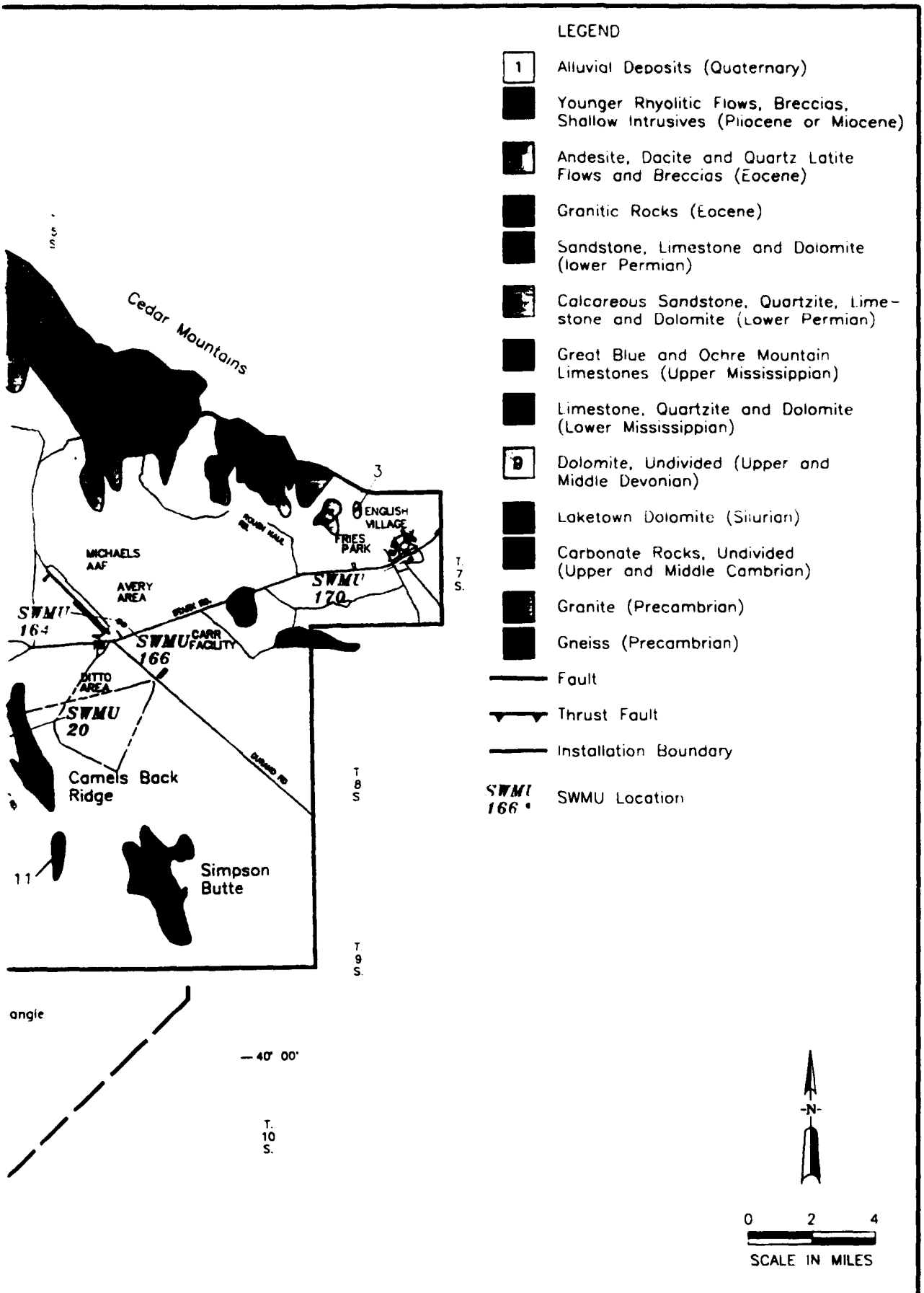


Figure 2.1-3
Generalized Geologic Map

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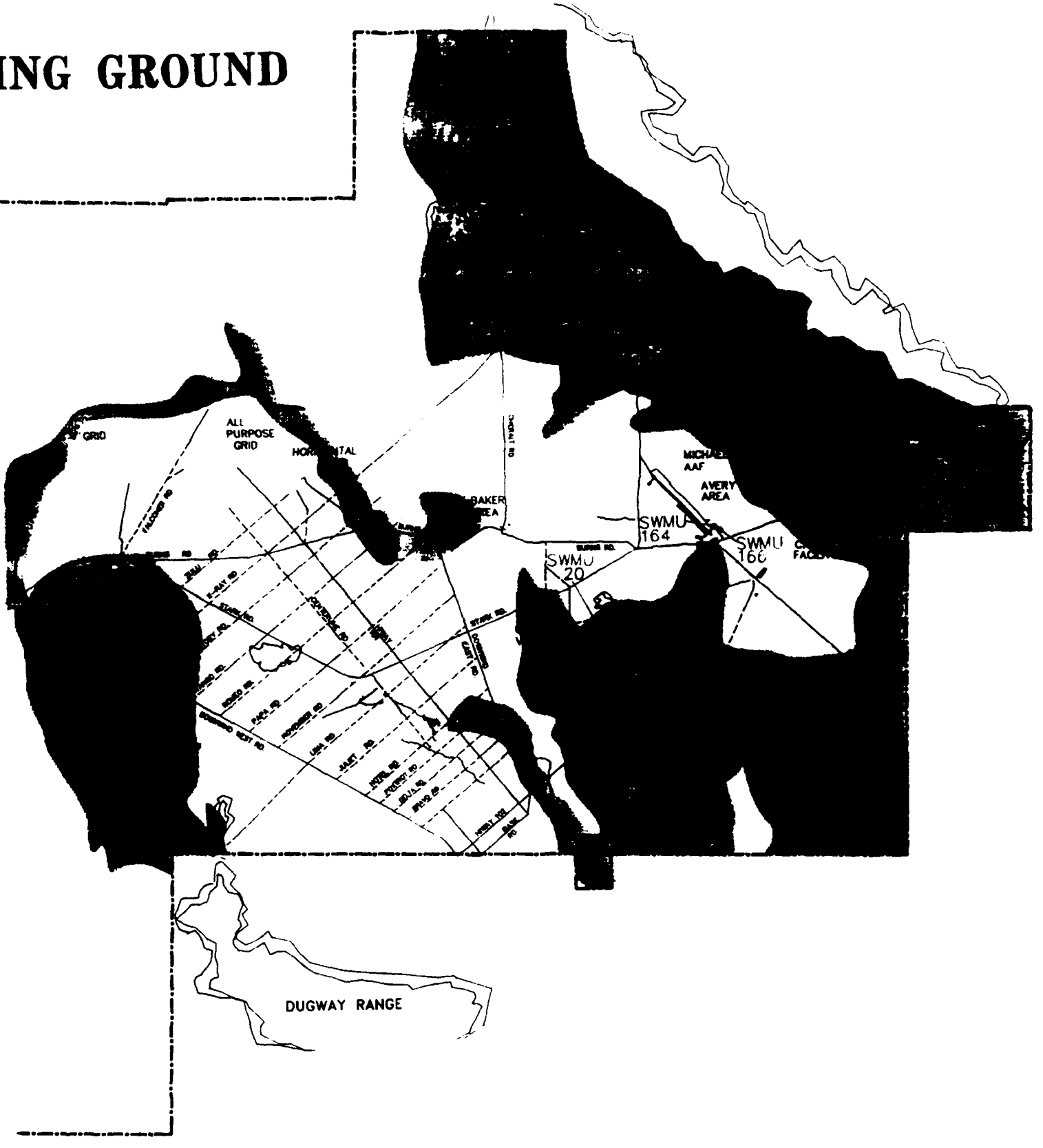
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DUGWAY PROVING GROUND



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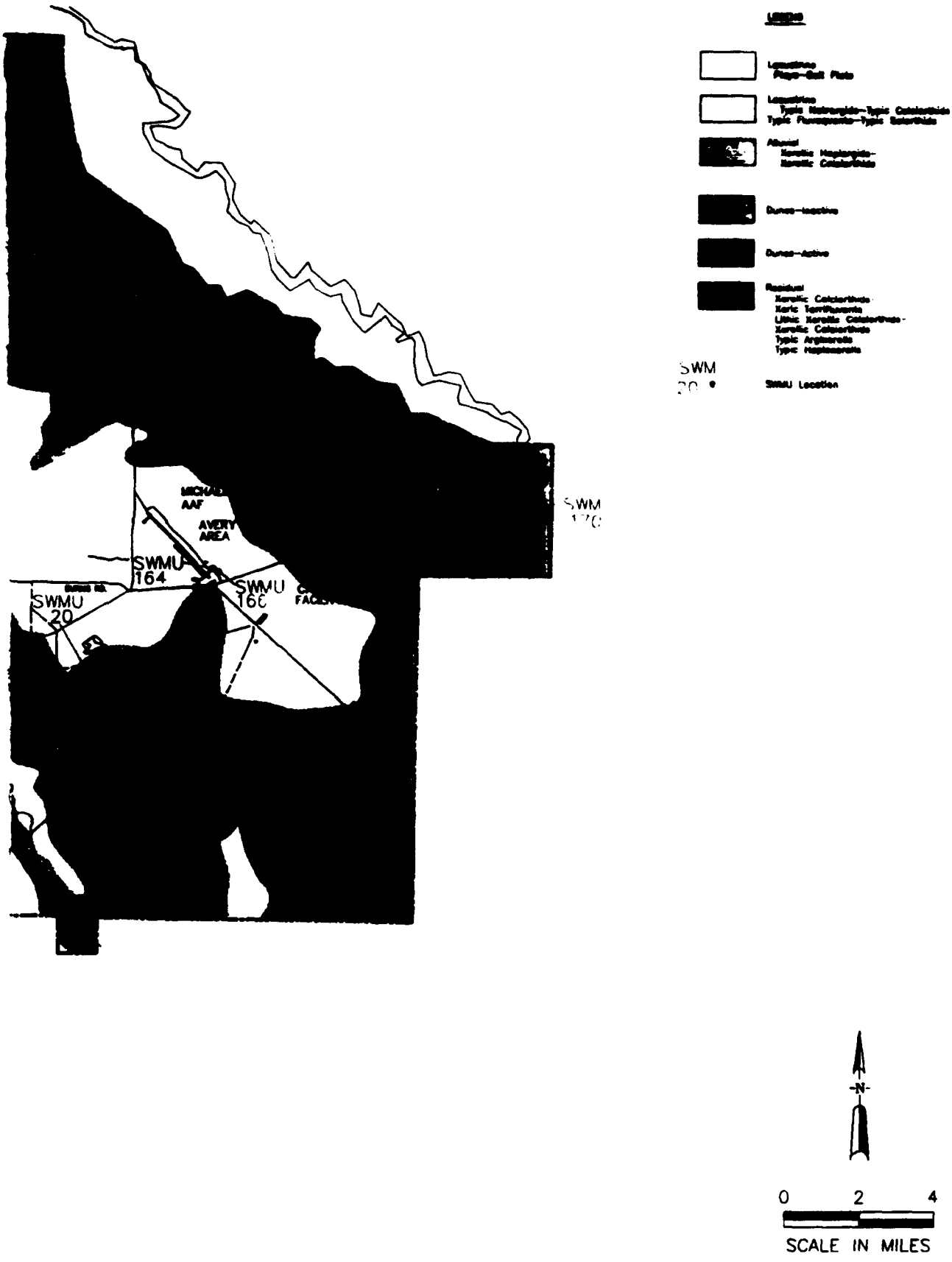
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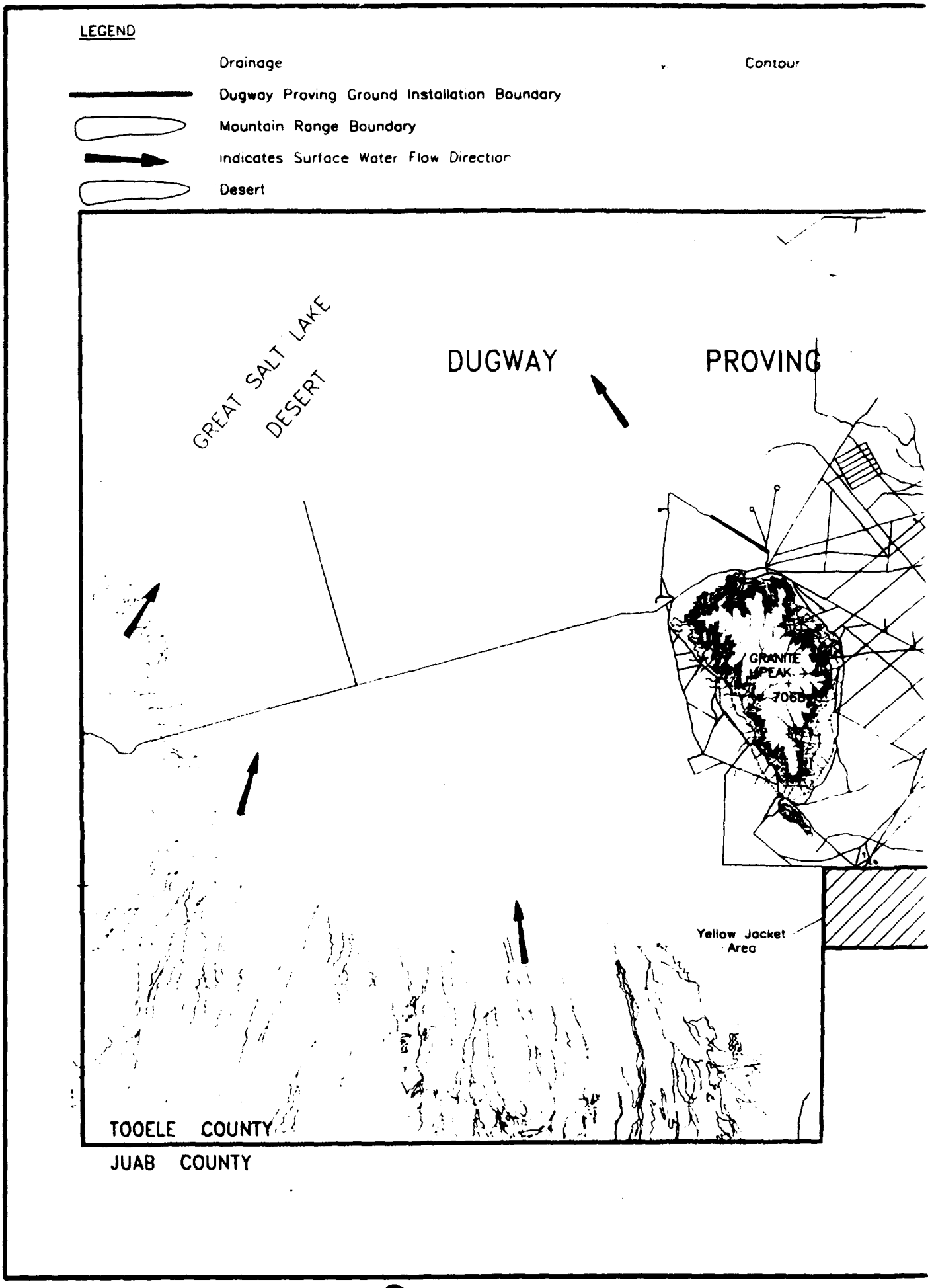
Figure 2.1-4
Dugway Prov



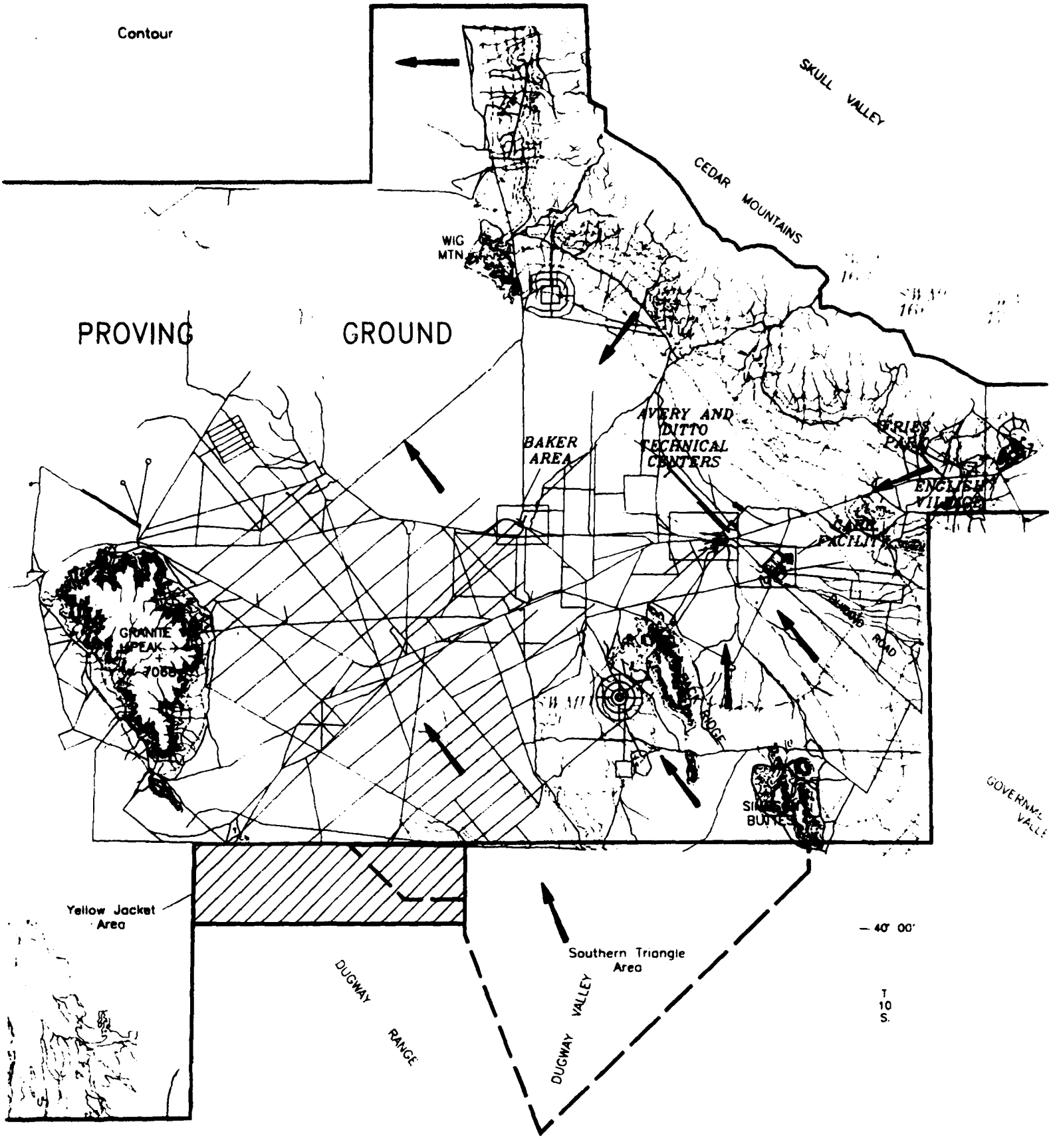
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Figure 2.1-4
Dugway Proving Ground Soils Map

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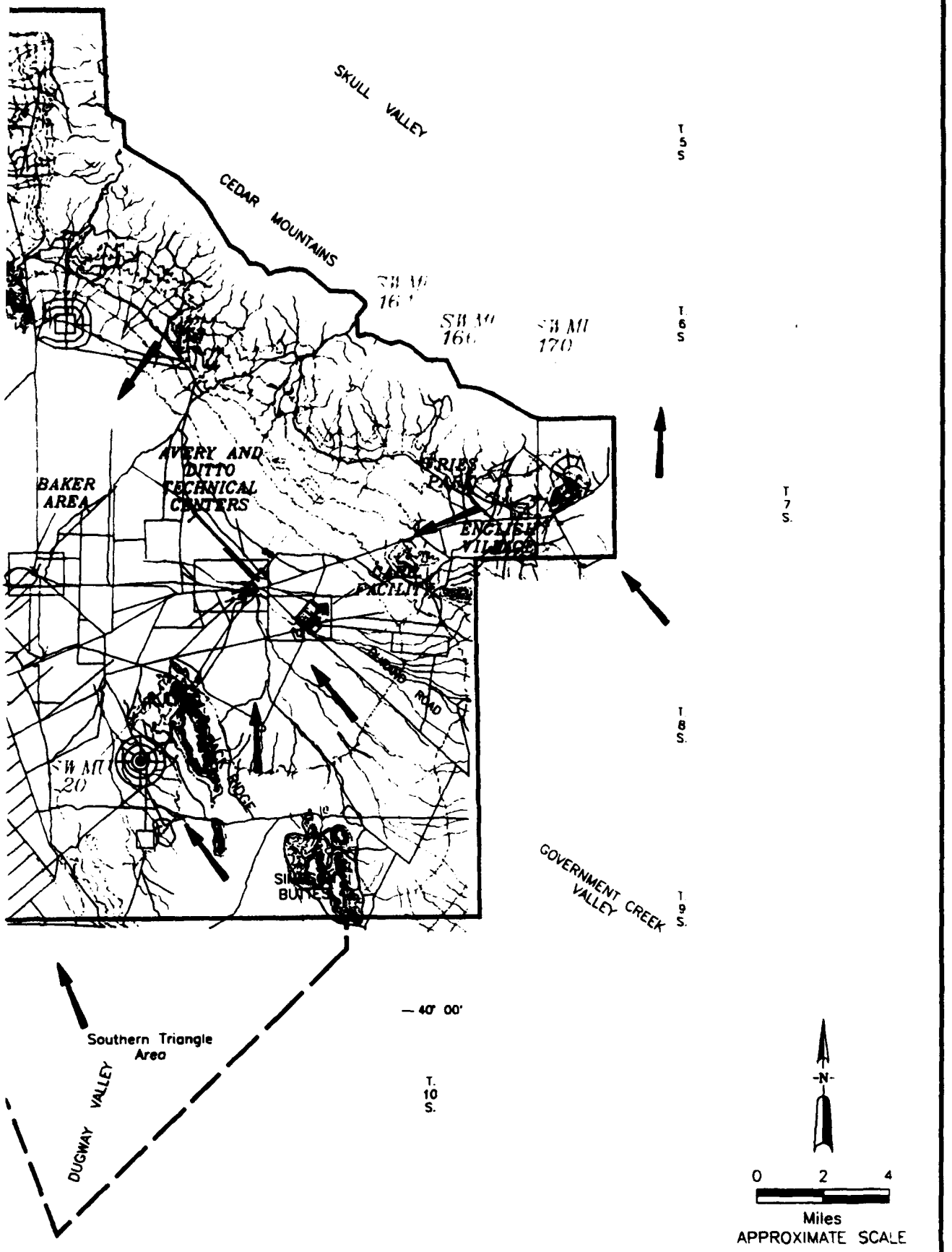
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Figure 2.1-5
Dugway Proving G
Surface Drainage



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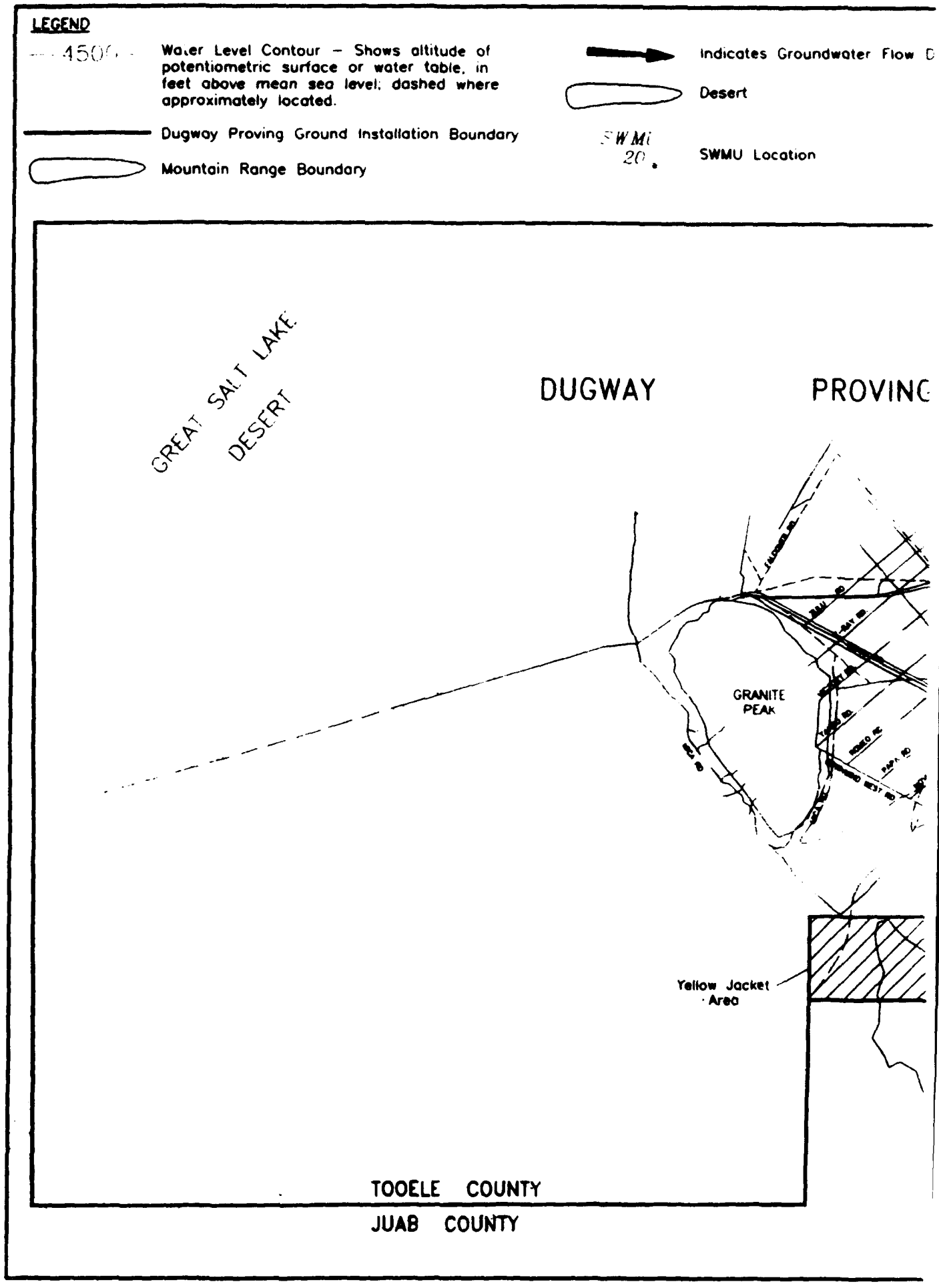
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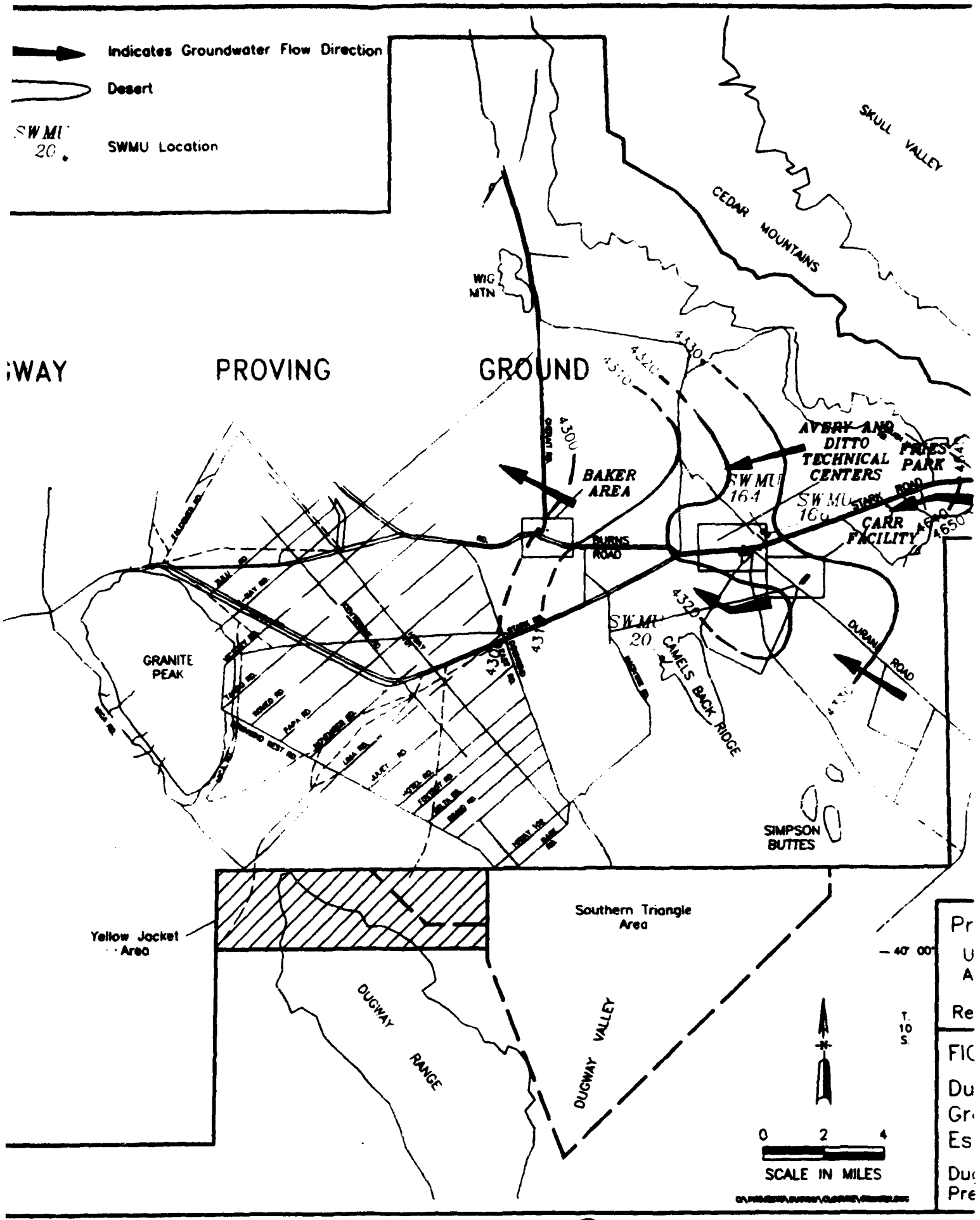
Figure 2.1-5
Dugway Proving Ground
Surface Drainage and Topography

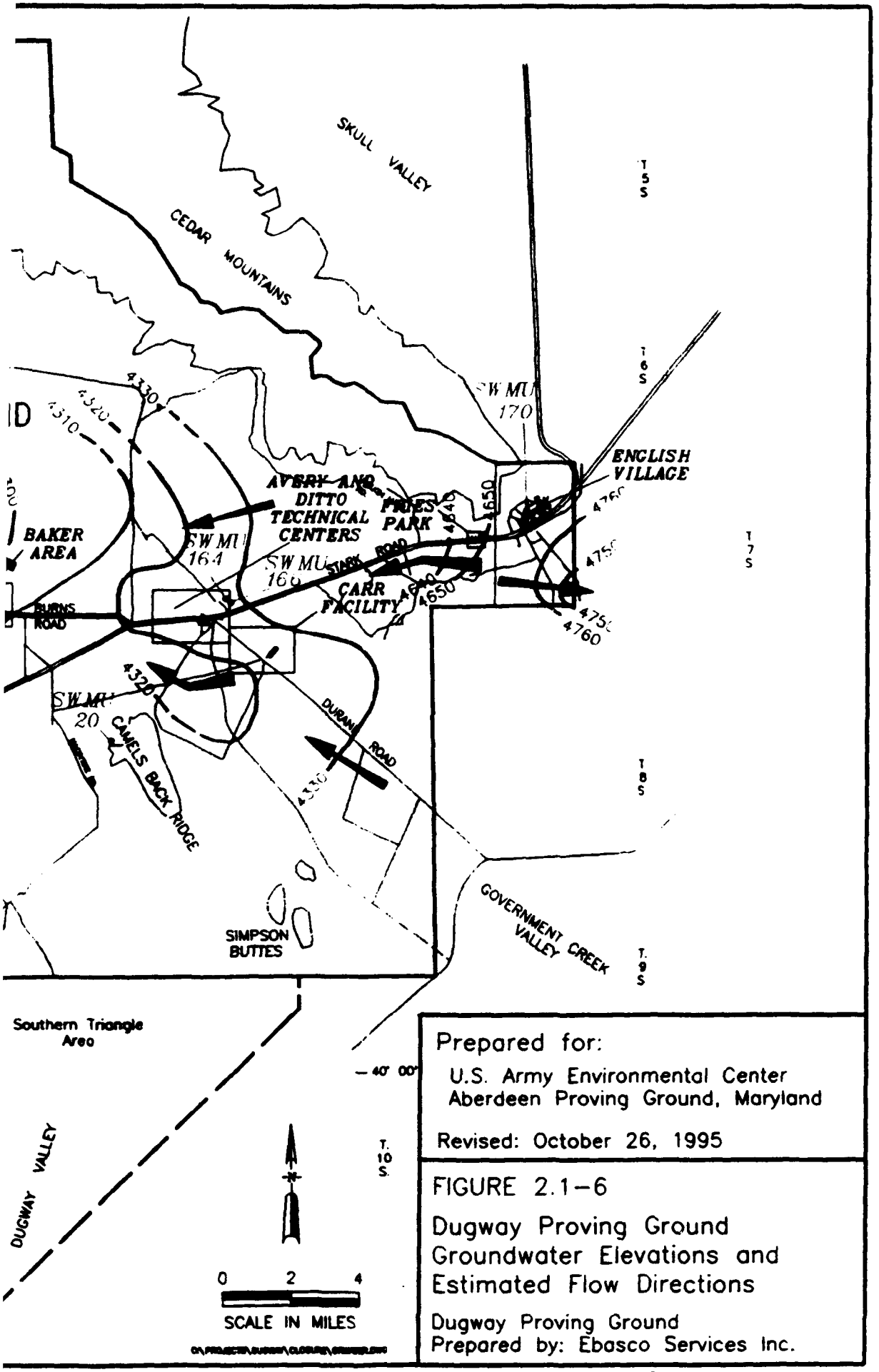
evaporates; a small amount infiltrates to be transpired by plants or to recharge the groundwater system. In this arid environment, surface runoff, which normally originates as precipitation at the higher elevations, does not usually reach the basin areas (Stephens and Sumison 1978). All runoff from the improved and built-up areas is conveyed by drainage ditches that parallel the roads. There are no storm sewers except in portions of Avery and Ditto. All of the Module 2 SWMUs are at least one-half mile from Government Creek, the primary natural drainage system in the basin. Additional information on surface-water drainage at DPG is presented in Section 2.1.4.1 of the Final Interim Report (EBASCO 1995a).

Groundwater recharge in the DPG area originates from precipitation in the mountainous areas around the desert and in the upper part of the alluvial fans (USAEHA 1987). Water flowing in the intermittent streams from the mountains infiltrates into the alluvial fans and recharges the aquifers in the alluvial fan and the basin-fill aquifers. Groundwater in the alluvium in and near the recharge areas near the mountains is unconfined. In the basin areas, the aquifers are generally under artesian conditions due to the overlying lacustrine silt and clay, which act as confining layers. Shallow groundwater, which is generally nonpotable, is also present in the basin areas above these confining layers.

A groundwater divide between Skull Valley and Dugway Valley occurs west of English Village (Figure 2.1-6). Groundwater in the English Village area is part of the aquifer system of Skull Valley. In this part of Skull Valley, lenticular sand and gravel units are hydraulically unconfined (Hood and Waddell 1968). Groundwater rises to 75 ft to 110 ft below ground surface in the water supply wells for English Village and is produced from coarse sediments at depths between 85 ft and 550 ft (USAEHA 1987). Groundwater in Skull Valley flows generally to the north (Hood and Waddell 1968), although extraction of water from supply wells at DPG may alter that flow pattern in the vicinity of English Village.


LEGEND
4500
Water Level Contour – Shows altitude of potentiometric surface or water table, in feet above mean sea level; dashed where approximately located.
Dugway Proving Ground Installation Boundary
Mountain Range Boundary
Indicates Groundwater Flow D
Desert
SWMU 20
SWMU Location
GREAT SALT LAKE DESERT
DUGWAY
PROVING
GRANITE PEAK
Yellow Jacket Area
TOOELE COUNTY
JUAB COUNTY


Indicates Groundwater Flow Direction
Desert
SWMU Location
SKULL VALLEY
CEDAR MOUNTAINS
WIG MTN
PROVING GROUND
BAKER AREA
AVERY AND DITTO TECHNICAL CENTERS
FRIES PARK
CARR FACILITY
BURNS ROAD
DUGWAY ROAD
CAMELS BACK RIDGE
GRANITE PEAK
SIMPSON BUTTES
Southern Triangle Area
Yellow Jacket Area
DUGWAY RANGE
DUGWAY VALLEY
SCALE IN MILES



Prepared for:

U.S. Army Environmental Center
Aberdeen Proving Ground, Maryland

Revised: October 26, 1995

FIGURE 2.1-6

Dugway Proving Ground
Groundwater Elevations and
Estimated Flow Directions

Dugway Proving Ground
Prepared by: Ebasco Services Inc.

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The depth to groundwater in water supply wells penetrating the deeper confined aquifers in the basin is generally less than 25 ft, or between 50 ft and 150 ft for coarser sediments that occur below a depth of 85 ft (USAEHA 1987). Groundwater generally flows to the northwest into the Great Salt Lake Desert, then to the north (Gates and Kruer 1981). At the saline mudflats of the Great Salt Lake Desert, groundwater discharges through transpiration by phreatophytes at the margins of the mudflats or by evaporation within the mudflats.

2.1.5 Climate

The climate of DPG is generally arid, as is much of the rest of the Basin and Range Province. In the vicinity of the installation, precipitation is greatest along the ridges of the Dugway and Cedar Mountains, where isolated storms yield moisture that does not reach the surrounding desert basins.

Pronounced temperature differences of more than 40 degrees Fahrenheit (°F) between day and night are characteristic of DPG and the vicinity. Records of meteorological data collected for the National Weather Service at DPG's Michael Army Airfield indicate that during the period of record (January 1951 through December 1975), the highest temperature was 105°F, and the lowest temperature was -22°F (Bauers 1995). The average annual number of frost-free days per year (for 1951 to 1964, inclusive) is 151 days.

Average annual precipitation ranges from less than 6 inches on the desert floor of the basin to 20 inches in the Cedar Mountains. DPG averages 45 inches of snowfall per year in its valleys and flats. This form of precipitation occurs primarily from October through April. During the summer months widely scattered cloudbursts can cause significant erosion; road washouts and rock debris slides are common.

Winds at DPG are generally southeasterly at night and northwesterly during the day, with an average speed of 6 miles per hour. However, high winds are common in the area from March to June and November to December, with gusts as high as 75 miles per hour.

2.1.6 Vegetation and Wildlife

The biota of west-central Utah are influenced by the physical geography, soil, seasonal distribution and amount of moisture, temperature, and topography. DPG falls within the Bonneville Basin, a desert area once covered by Lake Bonneville during the Pleistocene.

The soil derived from Lake Bonneville sediments is an important determinant of the present ecological setting of the DPG area. At DPG, the soil often creates conditions that support a variety of communities ranging from those characteristic of the mountains and canyons (e.g., mixed brush) to those characteristic of salt flats (e.g., pickleweed and barren areas). At certain locations, sand dunes are present. Fourteen plant communities have been recognized at DPG (Table 2.1-1). As shown in the table and in Figure 2.1-7, salt desert scrub, pickleweed barrens, and barren areas are the most prevalent vegetation types, followed by grassland and greasewood. The vegetation communities of the Module 2 SWMUs have been classified as salt desert scrub, sagebrush, and greasewood.

Plant communities can be used as indicators of topographic characteristics and soil types and the wildlife species that may occur there. Carnivores such as the badger, skunk, coyote, bobcat, and kit fox are highly mobile, ranging throughout DPG and nearby areas. Less wide-ranging wildlife, such as rodents and lagomorphs, tend to have their species, numbers, and distributions more limited by the kinds of plants, topography, and soil types collectively present in a habitat. Table 2.1-1 summarizes the wildlife observed and the SWMU and habitat where they occurred.

At the time DPG was established, the environmental effects of chemical warfare and biological defense testing were unknown. As a precaution against possible changes in patterns of succession or the possible introduction of microorganisms into the environment, an assessment of the current status of all indigenous organisms, including pathogens, was conducted in December 1952 by the Division of Biological Sciences at the University of Utah (Woodbury 1956). This study's findings are summarized in Table 2.1-2.

Table 2.1-1 Plant Communities and Biota Present at Dugway Proving Ground and Noted at Module 2 SWMUs

Plant Communities	SWMU 20 Shown on Figure 2.1-7	SWMU 20 Mapped in Field	SWMU 164 Shown on Figure 2.1-7	SWMU 164 Mapped in Field	SWMU 166 Shown on Figure 2.1-7	SWMU 166 Mapped in Field	SWMU 170 Shown on Figure 2.1-7	SWMU 170 Mapped in Field
Salt Desert Scrub (SD)	X		X					
Pickleweed Barrens (PB)								
Barren (BA)				X		X		X
Grassland (GR)								
Greasewood (GW)		X	X	X	X	X		
Sagebrush (SB)		X						
Juniper (JU)								
Pinyon-Juniper (PJ)								
Sagebrush/Perennial Grass (SG)								
Lowland Riparian (LR)							X	
Wetland (WT)								
Water (WA)								
Desert Grassland (DG)								
Agriculture (AG)								

Wildlife Observed	Season Observed in SWMU 20	164	166	170	Habitat in Which Observed
Grasshoppers					GR, SD, SW, BA
Crickets					BA, GW
Dragonflies					WT, GR, SD
Beetles					GR, BA
Bees		Su			BA, GW
Scorpions					GR, SD, GW, BA
Tadpoles					WT, PB, GR, GW, BA
Western bullsnake					GW, SD
Western racer					GR, BA
Striped whipsnake					BA, JU
Desert horned lizard					GW, BA
Side-blotched lizard	Su				GW, BA, GR
Longnose leopard lizard					GW
Western whiptail					JU

Wildlife Observed	Season Observed in SWMU 20	164	166	170	Habitat in Which Observed
Horned grebe					WT, BA, PB
Eared grebe					WT, BA, PB
Pied-billed grebe					WT, BA, PB
Mallard					WT, BA, PB
Blue-winged teal					WT, BA, PB
Redhead					WT, BA, PB
Ruddy duck					WT, BA, PB
Turkey vulture					SD, GW
Sharp-shinned hawk					GR
Northern harrier					LR, GW, BA, PB
Ferruginous hawk					BA, JU
Red-tailed hawk					SD, GW, Roadside
Golden eagle					GW, Roadside, Flyover
Osprey					Roadside
American kestrel					Roadside
Prairie falcon			Sp		GW, GR, JU, Roadside, BA, Flyover
Common egret					GR
Great blue heron					BA, PB
American coot					WT, BA, PB
Killdeer				Su	BA, GR, WT, GW, SD, BA
Lesser yellowlegs					WT, GW, GR, SD, BA
Common snipe					WT, GW, GR, SD, BA
Mourning dove		Su	Sp, Su	Su	PB, WT, BA, GW, GR, JU, LR
Great horned owl					GR, JU, BA
Short-eared owl					LR, BA, PB
Common nighthawk					GW, GR, SD, BA
Red-shafted flicker					LR, GR, JU, BA
Yellow-bellied sapsucker					LR, GR, JU, BA
Western kingbird		Su			GR, BA, GW, PB, SD
Say's phoebe					BA, PB, GW, GR, SD
Empidonax flycatcher					LR, BA, PB, WT, GW, GR, SD
Western flycatcher					GR

Table 2.1-1 Plant Communities and Biota Present at Dugway Proving Ground and Noted at Module 2 SWMUs

Wildlife Observed	Season Observed in SWMU 20	164	166	170	Habitat in Which Observed
Western wood pewee					GR, JU, BA
Horned lark					GW, GR, BA, SD, JU
Barn swallow		Su	Su		BA, GW, PB, GR, SD, Flyover
Raven	Su				GW, GR, JU, BA, SD, Roadside, Flyover
Wren spp.					GW, GR, SD, BA
Northern mockingbird	Su				GW, LR, GR, BA
Sage thrasher					GW, GR, BA, Roadside
American robin					GR, JU, BA
Shrike spp.					PB, WT, GW, Roadside
Starling			Fa, Su		GW, GR, JU, BA, PB, Roadside
Virginia's warbler					GR, JU, LR, WT, BA, GW, SD
Yellow-rumped warbler					GR, JU, GW, LR, WT, BA, SD
Black-throated gray warbler					LR, GW
MacGillivray's warbler					WT, GW, GR, SD, BA
Wilson's warbler					LR, GW
Western meadowlark					GW, GR, JU, BA, SD, Roadside
Yellow-headed blackbird					PB, WT, GW, BA, GR, SD
Red-winged blackbird					WT
Common grackle					GW, GR, JU, SD, BA
Brown-headed cowbird					GR, Roadside
Western tanager					LR, BA, PB
Blue grosbeak					LR, BA, PB
House finch					GW, GR, JU, CR, BA, SD, LR
American goldfinch					Flyover, BA, PB
Black-throated sparrow	Su				GW, GR, SD, BA
Sage sparrow					GW, GR, SD
Dark-eyed junco					WT, BA, PB, GR, JU
Tree sparrow					GW, GR, SD, BA
Brewer's sparrow					BA, PB
White-crowned sparrow			Fa		LR, GR, JU, BA, GW, WT, PB, Roadside, SD
Song sparrow					WT, BA, PB, GW

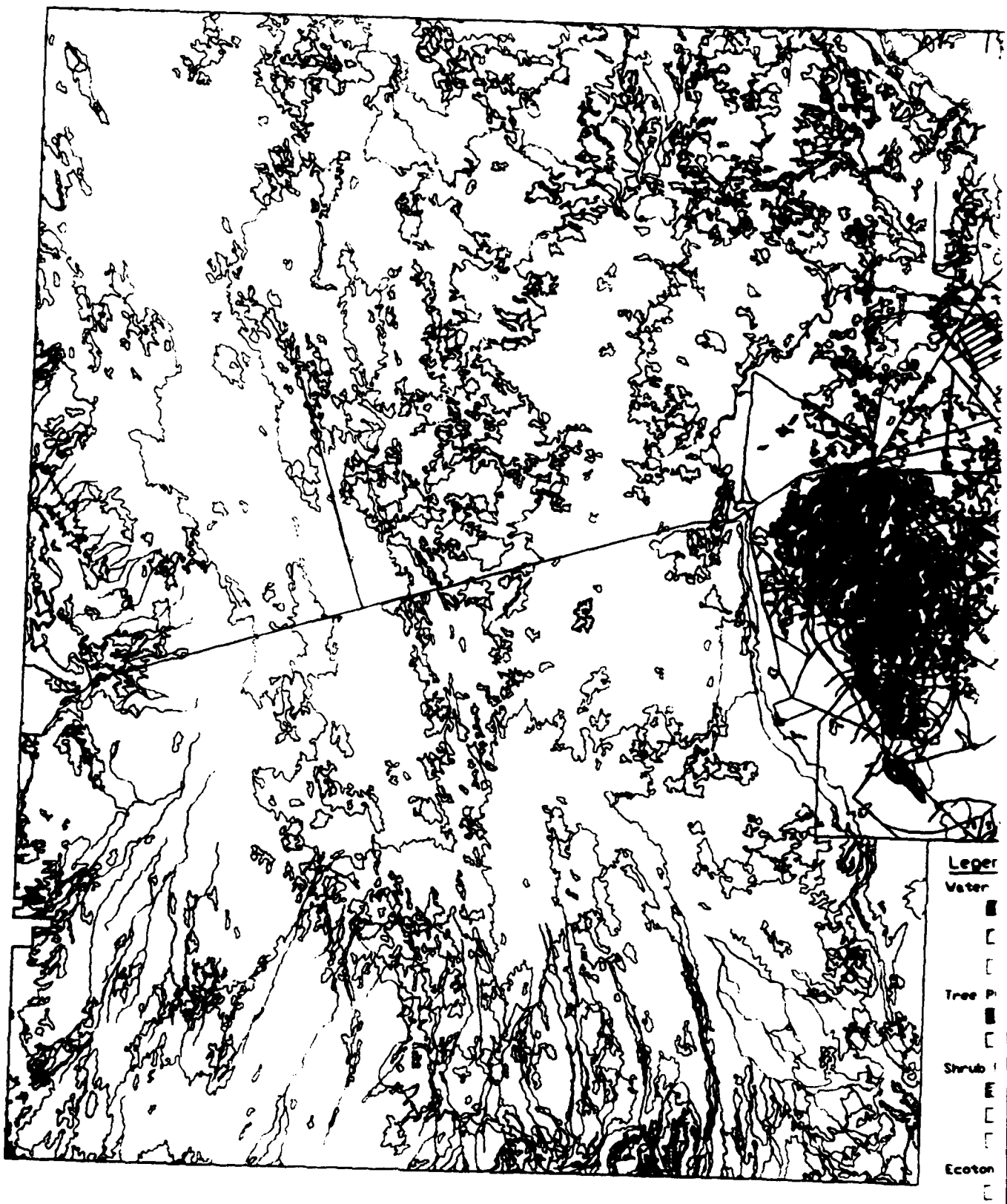
Table 2.1-1 Plant Communities and Biota Present at Dugway Proving Ground and Noted at Module 2 SWMUs

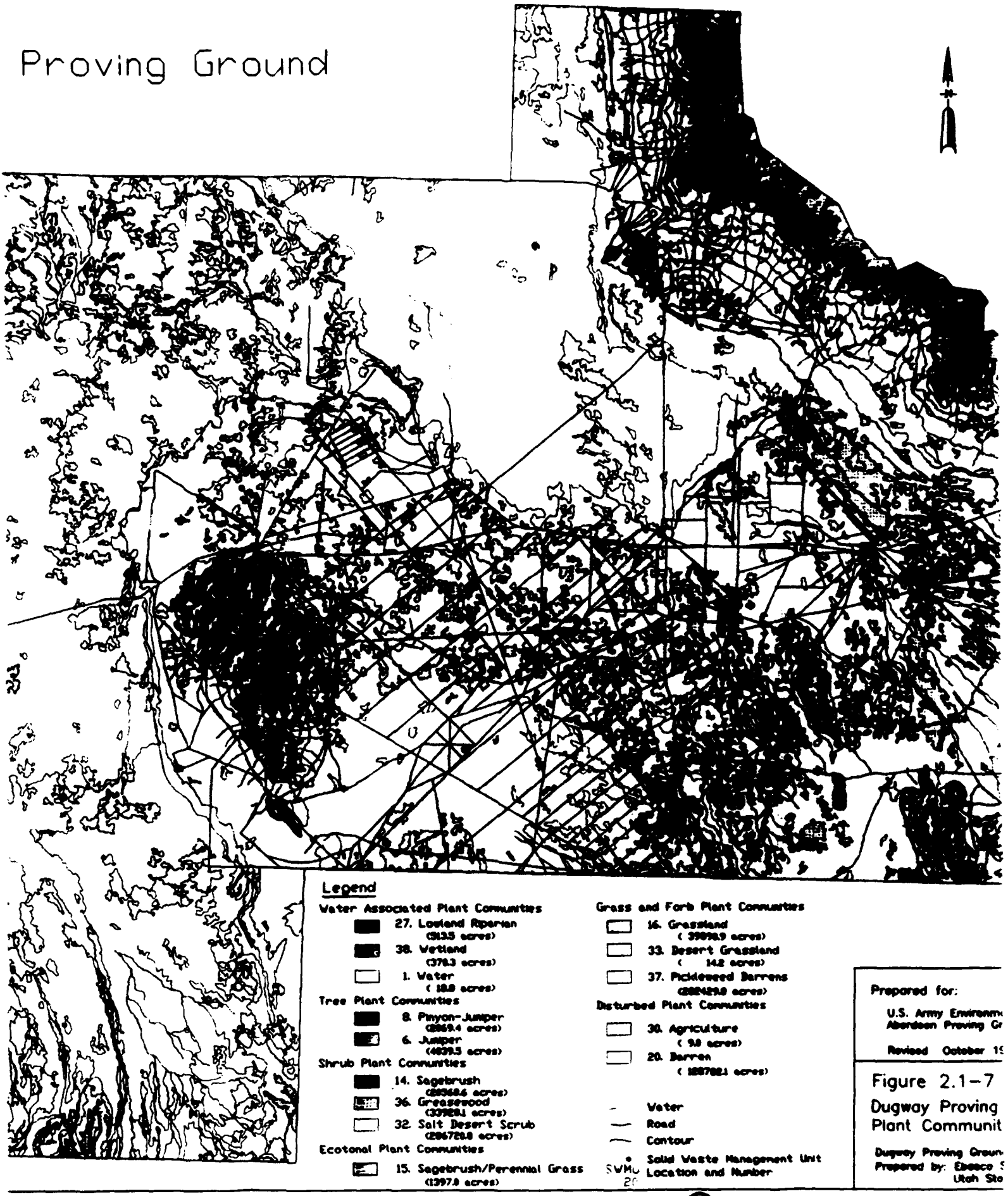
Wildlife Observed	Season Observed in SWMU 20	164	166	170	Habitat in Which Observed
Longtail weasel					SD, GW
Badger	Wi-sign				GW, SB, GR, BA
Coyote	Wi-sign				GR, BA, SD, JU, GW
Red fox					GR, GW, BA
Kit fox					GW, BA, GR
Townsend ground squirrel					GW, GR, BA
Whitetail antelope squirrel					GW, BA, PB, SD, GR
Ground squirrel					GW
Least chipmunk					GW
Ord's kangaroo rat					GR, GW, BA, SD
Great Basin kangaroo rat					GW, SD
Western harvest mouse					GW
Deer mouse					GW, GR, BA, JU, SD
Desert woodrat					GW
Mountain vole					GW
Blacktail jackrabbit			Su		GW, GR, BA, JU, WT
Desert cottontail	Wi				GW, SB
Mule deer					GR, SD
Pronghorn	Su		Fa-sign		GW, GR, SD, BA, Roadside
Feral horse					GW

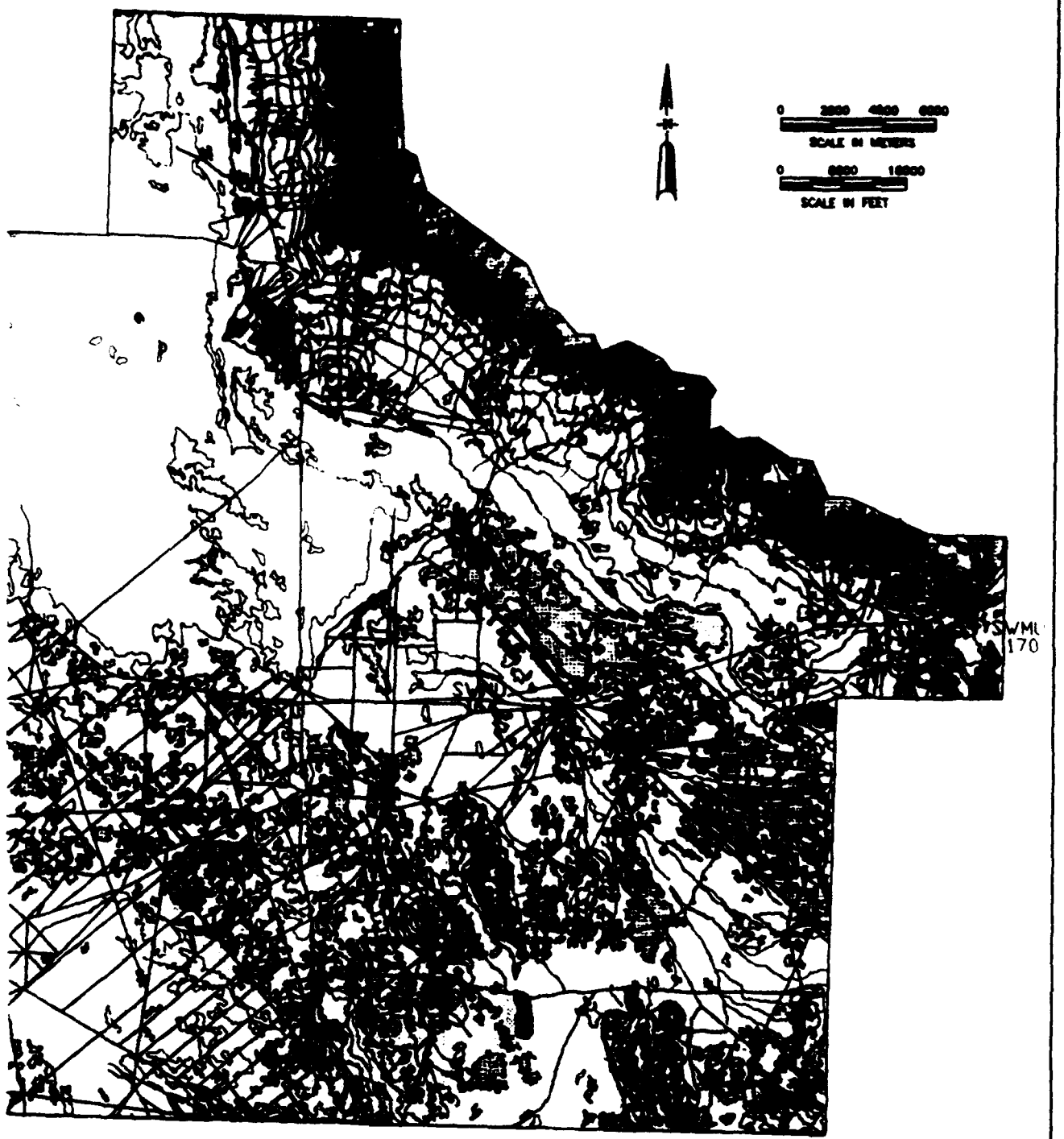
Note: Varying amounts of time were spent in individual SMWUs and plant communities

SU Summer
WI Winter
FA Fall
SP Spring

Dugway Proving Ground




Proving Ground
Legend
Water Associated Plant Communities
27. Lowland Riparian (3135 acres)
38. Wetland (378.3 acres)
1. Water (18.8 acres)
Tree Plant Communities
8. Pinyon-Juniper (2869.4 acres)
6. Juniper (4639.5 acres)
Shrub Plant Communities
14. Sagebrush (283668.6 acres)
36. Greasewood (33988.1 acres)
32. Salt Desert Scrub (286728.8 acres)
Ecotonal Plant Communities
15. Sagebrush/Perennial Grass (13997.8 acres)
Grass and Forb Plant Communities
16. Grassland (39878.9 acres)
33. Desert Grassland (14.2 acres)
37. Pickleweed Barrens (28824.8 acres)
Disturbed Plant Communities
30. Agriculture (9.8 acres)
20. Barren (1887.1 acres)
Water
Road
Contour
Solid Waste Management Unit Location and Number
SWMU 20
Prepared for:
U.S. Army Environm
Aberdeen Proving Gr
Revised October 19
Figure 2.1-7
Dugway Proving
Plant Communit
Dugway Proving Groun
Prepared by: Ebasco
Utah Sta



Plant Communities

...land Riparian
...3.5 acres)
...tland
...78.3 acres)
...ter
...18.8 acres)
...rties
...yon-Juniper
...869.4 acres)
...nper
...439.5 acres)
...rties
...eterush
...acres)
...easwood
...acres)
...t Desert Scrub
...acres)
...munities
...eterush/Perennial Grass
...397.8 acres)

Grass and Forb Plant Communities

- 16. Grassland (398958.9 acres)
- 33. Desert Grassland (14.2 acres)
- 37. Pickleweed Barrens (2884429.8 acres)

Disturbed Plant Communities

- 30. Agriculture (9.8 acres)
- 20. Barren (1887882.1 acres)

- Water
- Road
- Contour
- Solid Waste Management Unit Location and Number (SWMU 20)

Prepared for:

U.S. Army Environmental Center
Aberdeen Proving Ground, Maryland

Revised October 1995

Figure 2.1-7

Dugway Proving Ground
Plant Communities

Dugway Proving Ground
Prepared by: Ebasco Services Inc. using data from the Utah State Automated Geographic Reference Center

(3)

Table 2.1-2 Inventory of Flora and Fauna Found in the DPG Area[1]

Group	Description
Arthropod-Borne Viruses	Encephalomyelites, a California group virus carried by mosquitos, was most frequently recovered. Western, Hart Park, and Cache Valley arboviruses were less frequently recovered. Antibodies against the psittacosis-LGV group of viruses were occasionally found in jackrabbits.
Bacteria, Rickettsia, and Related Organisms	Parasitic bacteria reported from wildlife at DPG include species of *Micrococcus sp.* and *Sarcina sp.* and those species that cause spotted fever, Q-fever, brucellosis, tularemia, and plague.
Fungi (Basidiomycetes)	There are 114 species of fungi that occur in soils, animal dung, and other materials. Eleven types of mushrooms and six parasitic rusts and molds that occur naturally on native plants have also been reported.
Lichens	Twenty-six species of lichens in nine families have been reported.
Algae	Five species of blue-green algae, 8 species of diatoms, and 14 species of green algae have been identified in springs and ponds in the area.
Mosses (Bryophyta)	Eighteen species in six families have been reported.
Ferns	No ferns have been identified in the area.
Seed Plants (Spermatophyta)	There are 259 species of spermatophytes in 51 families that have been identified in the area. Conifers include junipers (or cedars), pinion pines, and jointfir (or Mormon tea). Flowering plants include pickleweed, fourwing saltbrush, shadscale, mound saltbrush, winterfat, gray molly, Russian thistle (or tumbleweed), greasewood, inkweed, and halogeton in the Goosefoot family, and sagebrush, rabbitbrush, and horsebrush in the Sunflower family. Also, 28 species of grasses are found in the area.

[1] Summarized from Woodbury 1956.

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Group	Description
Invertebrates	Protozoans occur as intestinal parasites in native rodents.
	Worms about 20 species of parasitic trematodes (flatworms of the Platyhelminthes phylum), 79 species of free-living roundworms (Nematoda class) were identified in the soil and as parasites. Four species of spiny-headed worms (in the Acanthocephala phylum) were found as parasites in birds and mammals.
	Mollusks are represented by 36 species of snails and slugs, and 1 species of clam. The Clinton's cave snail is known only from Clinton's cave.
	Arthropods in the Crustacean class include brine shrimp, fairy shrimp, seed shrimp, scuds, and sideswimmers. Arthropods in the Acarina order include more than 150 species of mites, ticks, spiders, pseudoscorpions, solpugids, and scorpions.
	Insects More than 1,300 species of insects in 157 families and 22 orders have been identified. Grasshoppers, bugs, beetles, mosquitos, gnats, flies, bees, wasps, ants, lice, and fleas are particularly noticeable.
Vertebrates	Fishes (Pisces superclass) include four native species of chub, minnows, and dace. Additionally, rainbow trout, brown trout, brook trout, largemouth bass, and bluegill have been introduced in the area. The numbers of the least chub are declining significantly.
	Amphibians The only native amphibian found in the area is the Great Basin spadefoot toad. Bullfrogs have been introduced.
	Reptiles Eight species of lizards and six species of snakes, including the Utah lizard, western collared lizard, Great Basin rattlesnake, and the Skull Valley pocket gopher snake, are found in the DPG area.
	Birds About 217 species of birds found; 53 species are year-around residents, 70 are summer residents, 11 are winter residents, 71 are migrants, and 12 species are occasional or accidental. Migratory birds sighted in the area include ducks, geese, bald eagles, and prairie falcons. The peregrine falcons appear to have been extirpated from the DPG area. Sandhill cranes have been reintroduced and introduced chukars have become established.
	Mammals are represented by about 50 species of rabbit, ground squirrel, weasel, badger, skunk, pronghorn antelope, mule deer, wild horse, and mountain lion.

[1] Summarized from Woodbury 1956.

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2.1.7 Demography, Land Use, and Water Use

Several small towns are located within a 30-mile radius of DPG. Based upon 1990 census figures, Tooele and Grantsville, in Tooele Valley, have populations of 13,887 and 4,500, respectively. The town of Dugway and the surrounding area have a population of 1,761, based on the 1990 census. Other smaller communities in the DPG area include Stockton, Ophir, Faust, Vernon, Rush Valley, Ioseda, Fish Springs, Callao, and Gold Hill (Figure 2.1-1). In 1990, the approximate population of these widely spaced, smaller communities, including ranches, totaled about 1,100.

The average population density in Tooele County is 3.8 people per square mile. However, the population density of the area between Dugway and Wendover, which includes nearly 83 percent of the county land area, is about 0.6 people per square mile, or just 14 percent of the county's population.

Two Native American reservations are located near DPG, the Goshute Indian Reservation on the Utah-Nevada border and the Skull Valley Indian Reservation near the eastern entrance of DPG (Figure 2.1-1). These two reservations comprise 18,844 acres, which is 0.4 percent of the total area in Tooele County. It is reported that 32 Native Americans reside in the Skull Valley reservation and 76 in the Goshute Reservation.

Agricultural activity near DPG is confined almost exclusively to Rush, Tooele, and Skull valleys. Tooele County ranks second out of the state's 29 counties in total area, but seventh in the percentage of area used for agriculture. Less than 1 percent of Toole County is either cropland or pasture, and approximately one-tenth of its land is range and one-fifth is forest. Irrigation is required in Skull Valley and most of Tooele Valley, but it is less important in Rush Valley, where typical agricultural practices are better suited to the seasonal rainfall patterns (USATHAMA 1979). In 1981, the major cash crops in the Dugway area consisted of wheat (21,400 bushels), barley (50,463 bushels), and alfalfa (21,300 tons). These crops were supplemented that year by the raising of 18,300 beef cattle and calves (Army 1982).

Sheep grazing is common in the unirrigated, higher portions of the valleys. Large semiannual migrations occur as flocks are moved between their summer ranges in the mountains of Nevada and Utah and their winter ranges in the deserts of western Utah. Since 1949, grazing on DPG has been restricted to the Southern Triangle, a 42,690-acre tract of land leased from the Bureau of Land Management. The terms of the lease permit grazing by the North Dugway Graziers Association for no more than 3 months from November 1 to April 30.

In the foreseeable future, agriculture will continue to play a minor role in the DPG area. More than 50 percent of DPG is without agricultural economic value, as most of the area is covered by salt flats and sand dunes. The remainder of DPG could support limited agricultural activity, primarily grazing along the slopes of the Cedar Mountains and grazing and other possible agricultural uses of the area between the eastern facility boundary and Little Granite Mountain. According to Fowkes (1964), the economic potential of the Granite Peak area "does not appear to be great." Other areas may be useful as gravel or soil borrow areas, but no other significant economic deposits are known within DPG.

2.2 PRE-CONSENT ORDER AND MOBILIZATION 1 AND 2 FIELD PROGRAMS

The Consent Order was issued by the state of Utah Solid and Hazardous Waste Committee on September 13, 1990, and amended on December 22, 1993. In anticipation of this order, the Army initiated pre-Consent Order assessment activities to delineate the boundaries of designated SWMUs, to determine whether hazardous wastes were disposed of at these sites, and to assess the potential for releases of hazardous substances. Preliminary investigations, including a review of historical records, assessment of available analytical data, and aerial photograph interpretation, were completed in 1989. Field activities, including site inspections, mapping, soil gas surveys, geophysical surveys, and limited source sampling, were completed in 1990.

Following the issuance of the Consent Order, a more detailed environmental investigation at 40 SWMUs was completed. The investigation was divided into three phases designated Mobilizations 1, 2, and 3.

The Mobilization 1 field program, which was conducted from August to December 1992, included soil gas and geophysical surveys, waste sampling, soil sampling, monitoring well installation, and groundwater sampling. Activities completed at each of the Module 2 SWMUs during the pre-Consent Order and Mobilization 1 programs are summarized in Table 2.2-1. The results of these activities at each SWMU were presented in the nature and extent investigation (NEI) plans. In general, the pre-Consent Order samples were analyzed using USAEC performance-demonstrated, which are based on and equivalent to U.S. Environmental Protection Agency (EPA) SW-846 analytical methods. The NEI plans also presented the sampling and analysis plans for Mobilization 2 investigations.

The Mobilization 2 field program, conducted from May to September 1993, was initiated to further define apparent contamination detected during the pre-Consent Order and Mobilization 1 field activities. The Mobilization 2 field activities were generally the same as the Mobilization 1 activities, but included more extensive sampling. These samples were analyzed using EPA SW-846 methods and validated to achieve data quality objective level III. The activities completed at each of the Module 2 SWMUs during Mobilization 2 are summarized in Table 2.2-2.

The chemical analyses completed on soil samples collected at each of the Module 2 SWMUs during Mobilizations 1 and 2 are presented in Table 2.2-3. One material sample was taken at SWMU 170 during Mobilization 2. The sample was analyzed for the RCRA characteristics of toxicity, reactivity, corrosivity, and ignitability. None of the analytes detected or the ignitability or corrosivity of the sample exceeded hazardous waste characterization limits as specified in 40 CFR 261.2, Subpart C. No groundwater samples were collected at the Module 2 SWMUs during any field mobilization, and no material, soil, or water sampling was conducted at these SWMUs during Mobilization 3, which was conducted from February to July 1995. Mobilization 3 sampling was not required at these SWMUs because evaluation of data available from previous sampling showed no data gaps.

2.3 DETERMINATION OF SOIL BACKGROUND GEOCHEMISTRY

This section describes the development of the background geochemistry dataset, the use of an analysis of variance (ANOVA) to compare SWMU sample data to this background dataset, and

Table 2.2-1 Mobilization 1 Activities at Module 2 SWMUs[1]

SWMU	Geophysical Survey	Soil Gas Survey	Total Number of Surface Soil Samples	Total Number of Composite Soil Samples	Total Number of Soil Samples	Total Number of Wells Installed	Total Number of Groundwater Samples
20	X		-	-	-	-	-
164			2	-	2	-	-
166			2	-	2	-	-
170			2	-	2	-	-
Total	1		6		6		

[1] This table includes geophysical and soil gas surveys that were completed during Mobilization 1, as well as surveys completed during the pre-Consent Order activities.

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Table 2.2-2 Mobilization 2 Activities at Module 2 SWMUs

SWMU	Geophysical Survey	Soil Gas Survey	Materials Samples	Surface Soil Samples	Total Number of Soil Borings Drilled[1]	Total Number of Soil Boring Samples	Total Number of Soil Samples	Total Number of Wells Installed	Total Number of Groundwater Samples
20			-	-	10	20	20	-	-
164			-	1	3	4	5	-	-
166			-	-	2	4	4	-	-
170			1	-	5	6	6	-	-
Total			1	1	20	34	35		

[1] Includes borings that were completed as wells.

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SWMU No.	Volatile Organics		Semivolatile Organics		Pesticides/ PCBs		Explosives		Agent Breakdown Products		Total Metals (>Background)		Cyanide		Total Petroleum Hydrocarbons	
20	●	◆	●	◆	■		●		●	◆	●	◆	●			
164	●	◆	●	◆	■		■				●	◆	●		●	◆
166	●	◆	●		■		■				●	◆	●		●	◆
170	●	◆	●		●	◆	■				●	◆				

DPG Mod2 Rev 7.15.97 jb

Table 2.2-3 • Summary of Soil Sample Analyses and Detections, Mobilizations 1 and 2

PCBs Polychlorinated biphenyls

- ● Analytes analyzed for
- ◆ Analytes detected
- ■ Analyzed under EPA Method 8270 (Semivolatile organics)

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the calculation of an upper tolerance limit (UTL) at 95 percent confidence for each metal in the background dataset. This evaluation is important because even natural levels of some metals in soil and groundwater at DPG result in human health risk calculations that exceed regulatory guidelines for clean closure. Therefore, objective methods are necessary to determine accurate background concentrations of metals and for comparing SWMU sample concentrations to the background dataset to identify contaminant releases, select chemicals of potential concern (COPCs) in the risk assessment, and help establish cleanup goals if corrective action is required.

Ideally, the ANOVA is used to identify metals above background at each SWMU, and the background UTL values are used as a tool for identifying metal concentrations in individual SWMU samples that are near or above background. In some cases, the effectiveness of these methods may be limited by a small sample size. A summary of the methodology used for the development and evaluation of background geochemical data is presented in the following section, while a detailed discussion is provided in Appendix A.

2.3.1 Development of the Background Dataset

The background dataset used in the ANOVA and in the UTL calculation must be prepared carefully to ensure that the dataset is truly representative of background conditions. The dataset was prepared in the following steps which are explained below:

1. All available data for background samples collected at DPG in both the Consent Order and Corrective Action programs were combined.
2. Data from samples collected at depths greater than 20 ft were deleted.
3. Using a nonparametric ANOVA, the surface soil and subsurface soil datasets for each metal were compared to ensure they were not statistically different and then the data from all depth intervals were combined into one dataset.
4. Nondetection results from inductively coupled plasma methods were removed for those metals also analyzed by graphite furnace methods with lower detection limits.
5. Statistical outliers were removed.
6. All results for samples with at least one detection of an organic compound were deleted.
7. Spatial trends across DPG in the data were evaluated.

The background soil dataset is composed of analytical data collected at Consent Order SWMUs during Mobilization 2 in 1993 and in the Phase I RFI conducted at Corrective Action SWMUs in 1994. Analytical results for the background samples collected during the two programs are included in Appendix A of this report.

A total of 69 background samples were collected from uncontaminated areas outside of the boundaries of 35 Consent Order SWMUs. Consent Order SWMU background soil borings were sampled at the 0- to 6-inch and 3- to 4-ft intervals and analyzed for volatile organic compounds (VOCs), semivolatile organic compounds (SVOCs), polychlorinated biphenyls (PCBs), pesticides, explosives, agent breakdown products, and metals. A supplemental background sampling program took place during Mobilization 3 in 1995 to verify the absence of mercury in background samples using a lower method reporting limit for mercury than was used in the previous programs. Eighteen additional samples were collected outside of 10 SWMUs and analyzed for mercury only in this supplemental program.

During the Phase I RFI of the Corrective Action SWMUs, 50 samples were collected outside of areas of known or suspected contamination at 8 separate study areas to a maximum depth of 87 ft in soil borings and upgradient monitoring well borings. These samples were analyzed for parameters consistent with SWMU-specific sampling in the eight study areas.

Most of the background and SWMU samples collected at the Consent Order SWMUs were collected at depths less than 20 ft. Background samples collected at the Corrective Action SWMUs were collected from the ground surface to a maximum depth of 87 ft. Since a depth of 20 ft corresponds to a likely remedial excavation depth and a maximum exposure depth for ecological receptors, the 10 background samples collected more than 20 ft below ground surface were eliminated from consideration so that the final background dataset would not be biased by samples from lower stratigraphic units. Appendix A, Table A10, presents the analytes detected in the 10 background samples collected deeper than 20 ft below ground surface.

Once the two datasets were combined and data from samples collected more than 20 ft below ground surface deleted, a nonparametric ANOVA was performed to test whether there was a

statistical difference between the metal concentrations in surface (0-ft to 0.5-ft interval) and subsurface (deeper than 0.5 ft) soil samples. The Wilcoxon rank-sum test was selected for this determination because it is valid for comparing two data populations, regardless of the population distributions. A detailed discussion on the use and evaluation of the Wilcoxon rank-sum test in comparing surface soil and subsurface soil populations is presented in Appendix 1B. The results of the ANOVA indicated that there is no significant statistical difference between the surface and subsurface data populations, so the data from all samples collected from depths less than or equal to 20 ft were recombined into one dataset.

A supplemental background sampling program took place during Mobilization 3 in 1995 to verify the absence of mercury in background samples. The method reporting limit for mercury was lower during the Mobilization 3 field program than in the previous programs, therefore eighteen additional samples were collected outside of 10 SWMUs and analyzed for mercury only.

The analytical methods used for metals analysis were different in the Consent Order and Corrective Action programs. Samples from the Consent Order SWMUs were analyzed using EPA SW-846 methods, and samples from the Corrective Action SWMUs were analyzed using U.S. Army Environmental Center (USAEC) performance-demonstrated methods. The reporting limits for both programs were compared and shown to be significantly different for antimony and thallium. The difference in reporting limits caused distributional skewness and high bias in the background datasets for these two metals because values assig l to nondetection results were higher than some actual detections. To eliminate this problem, the antimony and thallium results of the USAEC performance-demonstrated methods were deleted from the dataset. Lead was not treated like antimony and thallium because the detection limits of the different methods were comparable.

Upper extreme statistical outliers were identified and removed from the dataset to introduce additional conservatism and enhance the validity of the ANOVA and the UTL calculations. The outliers were identified using a statistical protocol provided by EPA (1989d, 1992b), and are significantly higher than the other values for the same metals in the dataset. Table A-2 in Appendix A lists the values that are statistical outliers. Once the outliers were identified, they

were plotted on a DPG base-wide map with the remaining background data and evaluated for spatial trends.

The outliers did not indicate any significant spatial trends, but elevated concentrations of lead in background samples occurred in the six background samples from the northwest side of Granite Peak at SWMUs 2, 7, and 9, as presented in Plate A-1 in Appendix A. Therefore, when lead concentrations in samples from these units exceeded the lead background value, they were compared to the higher lead levels in the six background samples from this part of DPG.

Finally, the data for all background samples with detections of organic compounds were deleted from the background dataset. These organic compound detections are listed by sample in Table A-1 of Appendix A. No attempt was made to distinguish anthropogenic from naturally occurring organic compounds since a clear case could not be made for most of these analytes; any organic detection disqualified the sample results. However, the occurrences of these organic compounds were compared to the occurrences of the upper-extreme outliers among the metal concentrations to evaluate whether organic contamination could be correlated with elevated metal concentrations. Since the two occurrences could not be correlated, the very conservative practice of deleting all samples from every boring in which an organic analyte was detected was not followed to avoid unnecessary reduction in the size of the background dataset.

The spatial variation of metals in background samples was evaluated for soil types and geographic areas at DPG. Soil types were taken from maps provided by the US Department of Agriculture, Natural Resources Conservation Service (NRCS, formerly the Soil Conservation Service) for the DPG vicinity. Geographic areas at DPG were defined by technical facilities or geographic localities where DPG SWMUs are clustered. A detailed discussion of the methodology used to evaluate spatial variation of background metals is presented in Appendix A. This process is summarized below.

Background samples were assigned to a soil type or geographic area and were then evaluated using box-and-whisker plots. The box-and-whisker plots graphically depict the distribution of data for a metal in each category. In general, the whiskers, or vertical lines in these plots, show

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the range of values in the category. Outliers are shown as single points beyond the length of the lines. The boxes show the range of values between the 25th and 75th percentile, and the horizontal line within each box shows the mean value. For purposes of evaluating trace metal concentrations between soil types or geographic locations, the most important information provided by the plots is probably the 75th percentile and maximum values. These results were evaluated subjectively by inspection, and indicated that, with the exception of lead in samples collected northwest of Granite Peak, metals concentrations in background soil samples collected to date do not vary significantly with soil type or geographic location.

2.3.2 Evaluation of the Final Background Dataset

Once the background dataset was finalized, site-specific assessments were conducted to identify potential inorganic contamination within each SWMU. The evaluation of the site-specific contamination assessment was performed in two ways.

In one statistical approach, each SWMU-specific dataset was compared to the background dataset using a nonparametric ANOVA, the same that was used to compare surface and subsurface soil data in developing the background dataset. The use of a nonparametric ANOVA does not require the data to be normally distributed. If the background population is determined to be significantly different from the SWMU population, and the mean rank of SWMU results exceeds the mean rank of background results, the SWMU concentrations may be above background.

In the other approach, the 95 percent UTL was calculated for each of the 23 metals in the background dataset (Table 2.3-1). A detailed discussion of how the UTL values were derived is presented in Appendix A. The UTL value was used as a conservative approximation of the maximum background concentration of each metal; approximately 5 percent of any normally distributed background concentrations are predicted to fall above that limit. The results of the UTL calculations are used as a statistical tool to identify potential contamination. These are the results presented on the analyte figures for each SWMU.

Table 2.3-2 presents a summary of metals that exceed the background UTLs for the Module 2 SWMUs, and Table 2.3-3 the summary of the results of the Wilcoxon rank-sum nonparametric

Table 2.3-1 Summary of Results and Upper Tolerance Values for the Background Soil Data Set

Analyte	Number of Samples	Number of Detections	Percent Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Arithmetic Mean	Standard Deviation	Background 95% Upper Tolerance Limit (µg/g)
Aluminum	86	86	100	2300	27000	DTO999SB03	11000	4700	19,000
Antimony	39	6	15	6.1	9.1	059BG01	3.1	2	9.1 MAX
Arsenic	85	79	93	1.3	21	046BG01	6.5	3.7	13
Barium	86	86	100	19	630	046BG01	210	110	400
Beryllium	86	49	57	0.5	1.1	TWR017MW01	0.53	0.3	1
Cadmium	86	1	1	0.68	0.68	124BG01	0.44	0.19	0.46 RL
Calcium	86	86	100	2300	240000	BKR999SB02	100000	51000	190,000
Chromium	86	86	100	2.9	20	DTO999SB03	10	3.9	17
Cobalt	86	72	84	2.2	17	EGL075SB04	4.1	2.3	7.9
Copper	86	83	97	1.9	69	036BG01	11	8.2	25
Iron	85	85	100	3400	28000	EGL075SB04	11000	4600	19,000
Lead	80	80	100	2.7	29	EGL075SB04	7.4	3.8	14
Magnesium	86	86	100	2000	33000	165BG01	16000	8000	29,000
Manganese	86	86	100	47	1100	APG999SB02	280	140	520
Mercury	104	1	1	0.061	0.061	EGL069MW03	0.037	0.012	0.05 RL
Nickel	86	80	93	3.0	36	EGL075SB04	8.6	5.2	17
Potassium	86	86	100	40	10000	APG999SB03	3900	2100	7,400
Selenium	63	4	6	0.61	2.9	EGL075SB04	0.29	0.42	2.9 MAX
Silver	86	0	0	ND	ND	- -	0.32	0.086	0.94 RL
Sodium	86	86	100	460	15000	DTO999SB03	4600	3600	11,000
Thallium	39	25	64	10	46	165BG01	16	11	35
Vanadium	86	86	100	9.0	57	EGL075SB04	20	6.9	29
Zinc	86	86	100	9.3	70	EGL075SB04	35	15	59

µg/g - Micrograms per gram
ND - Not detected
RL - Reporting limit
MAX - Maximum value

Table 2.3-2 Summary of Metals Exceeding the Background UTL in Module 2 SWMUs

Analyte	SWMU 20	SWMU 164	SWMU 166	SWMU 170
Aluminum				
Antimony	X A	X		
Arsenic	X	X		
Barium	X A		X A	
Beryllium				
Cadmium		X	X	
Calcium	X A		X A	
Chromium		X		X A
Cobalt				
Copper		X		
Iron				
Lead		X A	X	X A
Magnesium	X A		X	
Manganese			X	
Mercury		X		X
Nickel			X	
Potassium				
Selenium				
Silver				
Sodium	X			X
Thallium		X		
Vanadium				X
Zinc		X	X	X A

X Analyte exceeds the background 95 percent upper tolerance limit value

A Nonparametric analysis of variance supports evidence of potential site contamination

Table 2.3-3 Results of the Analysis of Variance Comparison for SWMU-Specific Data and the Background Dataset

Analyte	SWMU 20	SWMU 164	SWMU 166	SWMU 170
Aluminum				
Antimony	X		X	
Arsenic			X	
Barium	X		X	
Beryllium				
Cadmium				
Calcium	X		X	
Chromium		X		X
Cobalt				
Copper		X		
Iron				
Lead		X		X
Magnesium	X			
Manganese				
Mercury				
Nickel				
Potassium				
Selenium				
Silver				
Sodium				
Thallium		X		
Vanadium				
Zinc		X		X

X SWMU-specific data indicates potential site contamination compared to background data.

ANOVA. These results are described in detail in Appendix A and are discussed further in subsequent sections on the nature and extent of contamination at each SWMU.

2.4 CONTAMINATION ASSESSMENT METHODOLOGY

The nature and extent of chemical constituents detected in soil and material samples was evaluated using data from pre-Consent Order, Mobilization 1, and Mobilization 2 field activities at the Module 2 SWMUs. Screening methods were applied to the data to facilitate review and interpretation of the analytical results.

Soil COPCs discussed in the contaminant assessment were selected based on the comparison to DPG background values. All validated organic detections were considered COPCs, as were inorganic constituents for which the ANOVA results suggested a statistically significant difference with respect to background (presented in Section 2.3). The above criteria as well as subjective decision-making factors shown in Figure 2.5-2 (see page 2-42) were used to focus the discussions presented in the contamination assessments of each of the Module 2 SWMUs (Sections 3.1.4, 4.1.4, 5.1.4, and 6.1.4) on chemicals selected for evaluation in the risk assessments. The spatial distribution of COPC concentrations in soil that exceed the DPG background values and organic analytes detected in soil are shown in separate figures for inorganic and organic COPCs at each Module 2 SWMU. A brief discussion of the potential sources of these COPCs and their general fate and transport characteristics is included in the contamination assessment for each SWMU.

2.5 HUMAN HEALTH RISK ASSESSMENT METHODOLOGY

This section presents the methods used to evaluate human health risks associated with potential exposures to soil constituents at the Module 2 SWMUs. The objectives of the human health risk assessment were to estimate risk-based screening levels (RBSLs) that would be protective of human receptors for both current site use (approximated by evaluating an industrial land-use scenario) and hypothetical residential use and to characterize potential human health risks posed by the COPCs based on the RBSLs.

2.5.1 General Approach

2.5.1.1 Methodology

The risk assessments for DPG Module 2 SWMUs were developed using the streamlined approach outlined in the flow diagram shown in Figure 2.5-1. This approach is similar to the risk-based corrective action process developed by the American Society for Testing and Materials (ASTM 1994). In that process, conservative generic assumptions (e.g., hypothetical residential land use) are initially evaluated. The results and recommendations are reviewed and a determination is made whether a more detailed analysis is required using site-specific assumptions (e.g., for an actual industrial land-use scenario).

The risk calculations were performed after developing RBSLs. These RBSLs are the back-calculated soil concentrations of each COPC that would result in an excess cancer risk of 10^{-6} or noncancer risk (a hazard quotient [HQ]) of 1.0. The back-calculation is performed using standard default exposure parameters for a given exposure (or land-use) scenario. The RBSL of each COPC is therefore the minimum level of that chemical that would result in risk above the threshold values for risk-based closure of the SWMU if that COPC were the only contaminant at the unit. The RBSLs are useful in the contamination assessment for comparison to the actual concentrations detected in samples from the SWMUs. Given appropriate standard default exposure parameters for each exposure scenario, the risk associated with each COPC can be calculated from the RBSLs in a streamlined manner.

For this analysis, the initial phase involved deriving generic RBSLs for a conservative, hypothetical residential land-use scenario (Figure 2.5-1). This scenario assumes unrestricted land use and represents the most conservative analysis of potential soil exposures for the Module 2 SWMUs. According to state of Utah Administrative Code (UAC) R315-101, this conservative analysis is used to determine whether a SWMU qualifies for clean, or risk-based, closure. The second phase involves deriving RBSLs for an industrial land-use scenario—a more likely, but still conservative, scenario given the projected uses of DPG SWMU locations. This evaluation is used to determine whether corrective action is necessary according to the rule. For all the Module 2 SWMUs, risks associated with current site conditions (corresponding to minimal human exposure) would be lower than those estimated for future residential or industrial land-use scenarios.

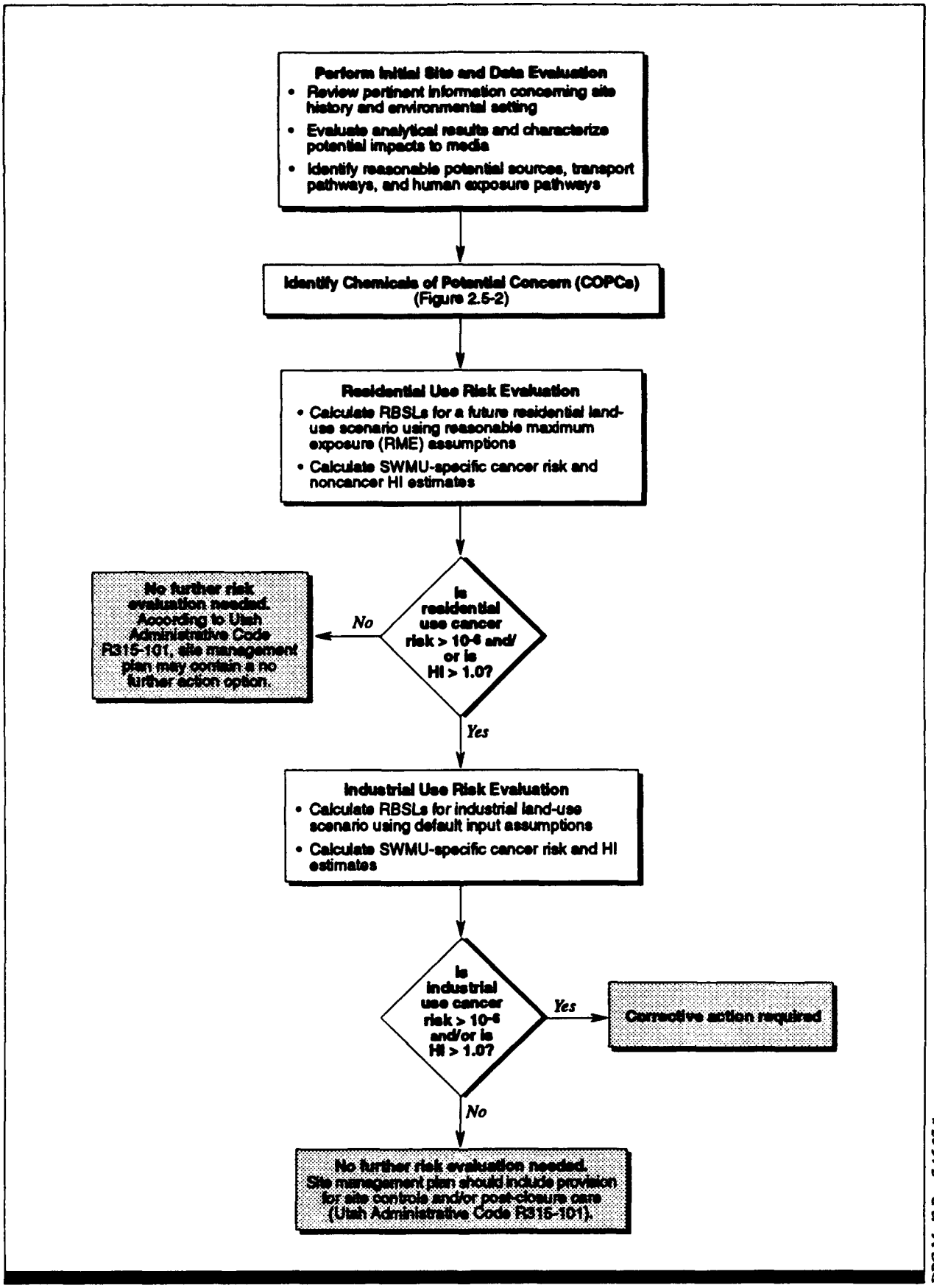


Figure 2.5-1 • Approach Used to Conduct Human Health Risk Assessment

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land-use RBSLs are below target risk criteria (i.e., a cancer risk range of 10^{-6} to 10^{-4} and an HI less than 1.0), then further risk evaluation is not warranted (Figure 2.5-1).

The reason for using this phased approach is twofold. First, because the exposure assumptions used in the risk calculations are the same for all SWMUs, it results in a more streamlined assessment (i.e., it eliminates redundancy in summarizing exposure assumptions and risk calculations). Second, it provides a worst-case, screening-level point of reference against which observed concentrations of soil COPCs can be compared. All methods used in this evaluation are in general accordance with guidelines presented in the state of Utah Hazardous Waste Management Rules, the Proposed EPA Guidance for RFIs (EPA 1989a), and Risk Assessment Guidance for Superfund (EPA 1989b).

2.5.1.2 Assumptions

As required by UAC R315-101, the human health risk assessments of the Module 2 SWMUs evaluate risks associated with hypothetical residential land use; the calculations are based on default exposure parameters recommended by EPA guidance listed in the previous section; and the toxicity information used for the COPCs is based on current data retrieved from EPA's Integrated Risk Information System (IRIS) database (EPA 1995b) and Health Effects Assessment Summary Table (HEAST) (EPA 1994c). Although the risk assessment calculations are based on a relatively low number of samples, and only soil samples, these risk assessments are adequately conservative because they are based primarily on EPA default exposure parameters which generally represent the 90th to 95th percentile value for a particular exposure variable (body weight is the 50th percentile because this variable is normally used in the denominator of exposure equations). Additionally, the industrial scenario used conservative assumptions to estimate risk (e.g., although it is more probable with federal employment, it is rare in today's society that a person will be employed at the same location for 25 years).

Surface water was not evaluated at these SWMUs because there is insufficient surface water at any of these SWMUs to be an exposure pathway or medium. The low levels of contaminants

detected in soil did not indicate a need for groundwater monitoring, as the detected concentrations of all contaminants were too low to be sources of contaminant leaching to groundwater.

The limited amount of soil sampling conducted at SWMUs 164, 166, and 170 reflects the small size of these units (500 to 750 square feet [ft^2]) and the lack of evidence of contaminant releases on a scale larger than traces of engine oil washed from uncontaminated vehicles at the wash racks during cleaning with water and steam. At SWMU 20, which is larger, only a limited amount of sampling was warranted because the site history indicates that no wastes of any kind were disposed of there. For these reasons, although the number of sample analyses is relatively low for calculating exposure concentrations of the COPCs (many COPCs were above background or detected in only one sample at a SWMU), these data are considered adequate for the risk assessment of these units. In fact, for COPCs that are truly site-related, risks may be overestimated because the wash racks are smaller than a reasonable exposure area. EPA Region VIII defines a reasonable exposure area consisting of 1-acre plots (43,560 ft^2) for use in risk assessments at CERCLA sites.

As discussed in later sections for each SWMU, chemical analytical results for the Module 2 SWMU samples indicated concentrations of inorganic constituents generally comparable to background and a limited number of organic constituents detected at very low concentrations. Since only low levels of soil contamination were detected in very small areas, and no other media are expected to be contaminated at the Module 2 SWMUs, the risk assessment evaluated only soil exposure pathways, and agricultural pathways were not included. This approach is conservative since even the industrial land-use scenario evaluated for some SWMUs substantially overestimates risks that could be associated with the actual occasional uses of these small units.

The food pathway was omitted from consideration because site-specific data (e.g., chemical- and media-specific partition coefficients) were not available. There is great variability in plant species uptake of chemicals, which is highly dependent on site-specific factors. Literature data are often limited, when available. The use of laboratory generated data would not be appropriate based on the generally harsh soil conditions at DPG. The use of non-site-specific parameters would have produced unquantifiable uncertainty in the results, making the findings useless for risk

management decisions. Additionally, most of the chemicals of concern at DPG are inorganics which do not tend to biomagnify in the food chain (although some may bioaccumulate in plant tissue). Furthermore, for many of the metals (e.g., lead), the soil/forage/cattle/people scenario is not significant given the tendency for these metals to concentrate in the liver, kidneys, and bone rather than meat and fat. Although some areas on DPG may support crop (garden) production, it is our understanding that the soil is generally very alkaline or saline and would require extensive amendment to support productive plant growth. Consequently, the potential for the food pathway to be a significant contributor to human exposure is very low, particularly for inorganic constituents.

2.5.2 Identification of Human Health COPCs

Human health COPCs are those chemicals that are to be evaluated in the human health risk assessment process. Soil COPCs for the Module 2 SWMUs were selected primarily on the basis of three factors: toxicity, frequency of detection, and comparison to DPG background levels. These factors were applied using the decision framework outlined in Figure 2.5-2 and are defined as follows:

- Toxicity—Chemicals considered essential nutrients and with negligible toxicity to humans were excluded from further consideration. Calcium, iron, magnesium, potassium, and sodium were therefore not selected as COPCs.

- Frequency of Detection—Chemicals detected at frequencies greater than 5 percent were retained for further evaluation. This criterion was not a factor for the Module 2 SWMUs, with sample sizes less than 20.

- Comparison to DPG Background Levels—SWMU-specific distributions of inorganic constituents were compared with DPG background data using the ANOVA described in Section 2.3. Based on the ANOVA, which is documented in Appendix A, constituents potentially present at concentrations greater than background were identified as potential COPCs.

- Historical Use—Records of use or disposal of inorganic constituents identified as COPCs by the ANOVA were reviewed. Suspected contaminants were evaluated further; inorganic COPCs that were not used or disposed of at the SWMU were not evaluated further.

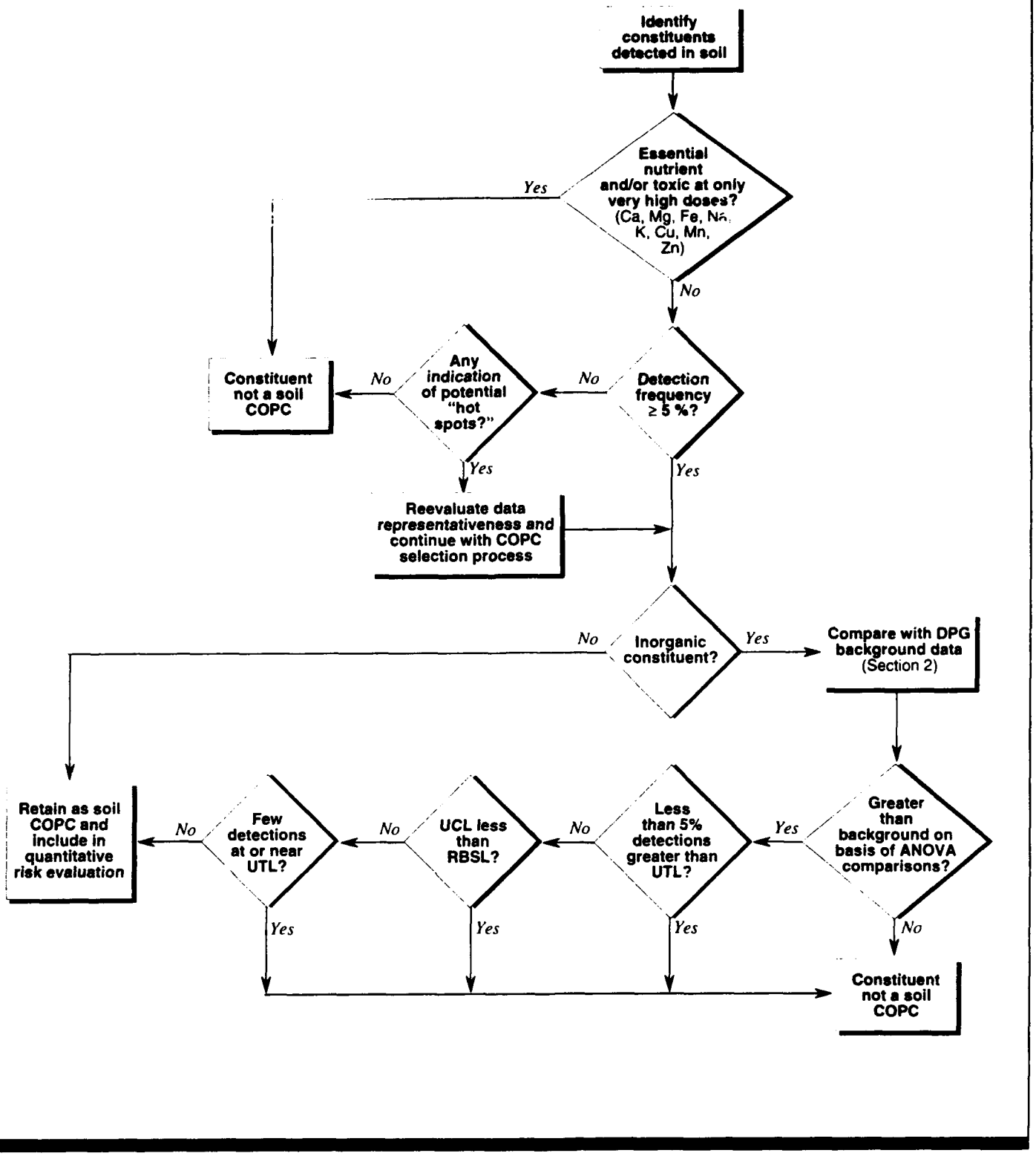


Figure 2.5-2 • Selection Process for Soil COPCs

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- Spatial Distribution—Plots depicting the spatial distribution of detections of a COPC were reviewed. If spatial patterns indicated a contaminant hot spot or other release, the COPC was retained.

- Comparison to UTL—Individual detections of inorganic constituents identified as COPCs by the ANOVA were compared to the DPG-specific background UTL value. If the concentration of an inorganic constituent exceeded the UTL, that analyte was included in further evaluation. If the COPC had only a few detections exceeding the UTL by a small margin, then the COPC was excluded from further evaluation.

- Comparison to RBSL—exposure point concentrations (EPCs), equivalent to the 95 percent upper confidence limit (UCL) of inorganic COPCs were compared to RBSLs. For carcinogenic constituents, RME concentrations and 10^{-6} target risk RBSLs were compared directly. For noncarcinogenic constituents, EPCs were compared to one-tenth of the noncarcinogenic RBSL. If both comparisons showed the EPCs less than the RBSL, the inorganic COPC was excluded from further evaluation.

2.5.3 Exposure Assessment

The following sections identify the exposure pathways that were evaluated in the risk assessment (Section 2.5.3.1) and specify the exposure parameters used to estimate pathway-specific RBSLs (Section 2.5.3.2).

2.5.3.1 Exposure Pathway Evaluation

In order for an individual to be exposed to COPCs, the exposure pathway must be complete. A complete pathway consists of the following elements:

- A source and mechanism of chemical release to the environment
- An environmental transport medium (e.g., soil)
- A point of contact with the medium (an exposure point)
- An exposure route at the contact point (e.g., ingestion, dermal contact, or inhalation)

As discussed in Section 2.5.1 and outlined in the flow diagram (Figure 2.5-1), two scenarios were evaluated in the Module 2 risk assessments, hypothetical residential land use and industrial land use. The receptors associated with these scenarios are residents (adults and children) and site workers (adults only). The pathways through which these receptors could be exposed to soil COPCs are summarized below:

- Hypothetical Resident—The residential-use scenario assumes that homes would be built in the immediate vicinity of the SWMU and that adult and child residents would be exposed to surface or subsurface soil 350 days per year for 30 years. Exposure to soil would occur through incidental ingestion, dermal contact, inhalation of particulates, and inhalation of volatilized constituents. This exposure scenario, which was evaluated for all the Module 2 SWMUs, represents the greatest potential exposure to soil COPCs. The evaluation is hypothetical for SWMUs 20, 164, and 166, which are located in remote or industrial areas. Residential use of the area surrounding SWMU 170 is somewhat more likely since this SWMU is located in English Village, which is partly residential.

- Future Industrial Worker—The industrial-use scenario assumes that an adult works at the SWMU 250 days per year for 30 years and comes into contact with surface or subsurface soil through incidental ingestion, dermal contact, inhalation of particulates, and inhalation of volatilized constituents. As indicated in Figure 2.5-1, this scenario was evaluated only if the cancer risk for residential use exceeded 10^{-6} or the noncancer HI was greater than 1.0.

These receptor groups and associated pathways were identified based on regulatory criteria and conservative assumptions regarding potential exposures associate with actual site uses. Exposures associated with current site uses and conditions are expected to be negligible. Consequently, the RBSLs derived for the two land-use scenarios defined above would also protect the site workers who are the current receptors.

2.5.3.2 Exposure Assumptions

To calculate chemical intakes, RBSLs, and corresponding risks, the following factors must be estimated:

- The amount of chemical taken up by the body through ingestion, dermal absorption, and inhalation

- The frequency and duration of exposures

- The constituent concentration in the medium at the point of exposure (the EPC)

The input parameters used to estimate exposures for the residential and industrial land-use scenarios are summarized in Table 2.5-1. The exposure point concentration for each constituent is the 95% UCL concentration term. Additionally, the use of default input parameters (e.g., breathing rate, ingestion rate, etc.) representing the 90th or 95th percentile, considered upper bound exposure variables, with the 95% UCL concentration term results in a maximum

Table 2.5-1 Assumptions Used to Calculate Soil RBSLs and Corresponding Risk Estimates for Residential and Industrial Land-Use Scenario Evaluations

	Residential Land-Use Scenario [1]		Industrial Land-Use Scenario [2]	
Input Parameter	Assumed Value: Carcinogenic Endpoints	Assumed Value: Noncarcinogenic Endpoints	Assumed Value: Carcinogenic and Noncarcinogenic Endpoints	Basis/Data Source(s)
Target Cancer Risk (TCR), Target Noncancer HQ	$TCR = 10^{-6}$	$HQ = 1.0$	$TCR = 10^{-6}$, $HQ = 1.0$	According to State of Utah Hazardous Waste Management Rules (USHWCB 1994), if the cancer risk level estimated for a site is below 10^{-6} and the HI is less than 1.0 for noncarcinogens, the site management plan may contain a no further action option (given no indication of adverse health effects). Cancer risk estimates between 10^{-6} and 10^{-4} are considered within the range of risks requiring only site controls. This interpretation is similar to the one established by EPA for the Superfund program (EPA 1989b) under the National Contingency Plan (EPA 1990). (See text Section 2.5.5.5.)
Age During Exposure	Adult	Child (ages 1–6)	Adult	Standard EPA default assumptions.[3]
Body Weight (BW)	70 kg	15 kg	70 kg	Standard EPA default assumptions. [3]
Exposure Frequency (EF)	350 days per year	350 days per year	250 days per year	Standard EPA default assumptions. [3] For both current and projected future uses of Module 2 SWMUs, these assumptions are considered very conservative. The SWMUs are either remote, have limited access, and/or the paved surface would preclude exposure to underlying soil.
Exposure Duration (ED)	30 years	6 years	25 years	Standard EPA default assumptions [3]

Table 2.5-1 Assumptions Used to Calculate Soil RBSLs and Corresponding Risk Estimates for Residential and Industrial Land-Use Scenario Evaluations

	Residential Land-Use Scenario [1]		Industrial Land-Use Scenario [2]	
Input Parameter	Assumed Value: Carcinogenic Endpoints	Assumed Value: Noncarcinogenic Endpoints	Assumed Value: Carcinogenic and Noncarcinogenic Endpoints	Basis/Data Source(s)
Soil Ingestion Rate	114.3 mg-yr/kg-dy	200 mg/day	50 mg/day	Standard EPA default assumptions. [3] Value assumed for residential use adult receptors is age-adjusted soil ingestion rate (to account for earlier childhood exposures).
Skin Surface Area Exposed	5,800 cm^2	2,650 cm^2	5,800 cm^2	Upper-bound default values recommended by EPA, assuming that dermal exposure occurs at the hands, legs, arms, neck, and head, and that approximately 25% of the total skin surface area is exposed (EPA 1992a).
Adherence Factor	1 mg/cm^2/event	1 mg/cm^2/event	1 mg/cm^2/event	Reasonable upper-bound value for soil-to-skin adherence rate reported in EPA's Dermal Exposure Assessment Guidance (1992a).
Dermal Absorption Factors				
Inorganic Constituents	0.001 (unitless) or 0.1 percent, based on absorption data for cadmium chloride			Appendix Table B.1-1 (pg. 3); EPA 1992a
VOCs	0.05 or 5 percent			Appendix Table B.1-2 (pg. 3); EPA 1992a
SVOCs	0.05 or 5 percent, based on absorption data for benzo(a)pyrene			Appendix Table B.1-3 (pg. 3); EPA 1992a
Pesticides/PCBs	0.05 or 5 percent, based on absorption data for PPDDT			Appendix Table B.1-4 (pg. 3); EPA 1992a
Inhalation Rate	11.66 m^3-yr/kg-day	12 m^3/day	20 m^3/day	Standard EPA default assumptions. [3] Value assumed for an adult resident is the age-adjusted inhalation rate (to account for earlier childhood exposures).
Particulate Emission Factor (PEF)	6.79E+08 m^3 air/kg soil			Emission factor recommended in EPA's Technical Background Document for Soil Screening Level Guidance (EPA 1994a).

Table 2.5-1 Assumptions Used to Calculate Soil RBSLs and Corresponding Risk Estimates for Residential and Industrial Land-Use Scenario Evaluations

Input Parameter	Residential Land-Use Scenario [1] Assumed Value: Carcinogenic Endpoints	Residential Land-Use Scenario [1] Assumed Value: Noncarcinogenic Endpoints	Industrial Land-Use Scenario [2] Assumed Value: Carcinogenic and Noncarcinogenic Endpoints	Basis/Data Source(s)
Volatilization Factors (VFs) and Inhalation RBSLs for Volatile Parameters	The equation used to calculate volatilization factors is very complex and requires definition of numerous chemical-specific input parameters (EPA 1994a, Appendix B, Table B.1-2(c)). In many cases, the RBSLs calculated using the VFs exceed the soil saturation limit. Given the uncertainties underlying VF derivation, and the changing guidance reflecting this pathway's complexity, inhalation RBSLs for volatile, semivolatile, and pesticide parameters are the inhalation soil screening levels (SSLs) recommended by EPA (EPA 1994a).			EPA 1994a. Also, see inhalation RBSL documentation provided in Appendices B.1 and B.2.
Chemical-Specific Soil Exposure Point Concentrations (EPCs)	95 percent upper confidence limit (UCL) of the arithmetic mean concentration, except for COPCs for which the 95% UCL exceeds the maximum concentration, in which case the maximum was used.			EPA's Supplemental Guidance to RAGS: Calculating the Concentration Term (EPA 1994e)

Note:

[1] Appendix B.1 documents the input parameters, equations, and calculations for residential land-use RBSLs.

[2] Appendix B.2 documents the input parameters, equations, and calculations for industrial land-use RBSLs.

[3] The standard EPA default assumptions referenced above are found in EPA 1991a, 1994a, and 1995a.

reasonable exposure estimate. These assumptions form the basis of the RBSLs listed in Tables 2.5-2 and 2.5-3, and for the SWMU-specific risk assessments presented in the following sections.

2.5.4 Toxicity Assessment

In the risk assessment two categories of chemical toxicity are considered—carcinogenic and noncarcinogenic effects. These effects are quantified using chemical-specific toxicity values in EPA's IRIS database (EPA 1995b) and HEAST (EPA 1994c). The following sections summarize the methods used to evaluate the carcinogenic and noncarcinogenic endpoints. Appendix B.3 details these methods and includes toxicity profiles for the COPCs evaluated in the SWMU-specific risk assessments. The toxicity values are also summarized in Table 2.5-4 for all Module 2 COPCs.

2.5.4.1 Evaluation of Carcinogenic Effects

Carcinogenic effects are assessed using cancer slope factors (SFs). These factors are defined as upper bound estimates of the probability of an individual developing cancer resulting from chronic exposure to a specified level of a potential carcinogen. SFs are derived assuming that exposure to a chemical can induce changes in a single cell or a small number of cells that eventually lead to the formation of tumors (EPA 1989c). This mechanism is described as a no-threshold mechanism because it conservatively assumes that there is no level of exposure below which disease (i.e., cancer) will not result. SFs are usually derived based on animal studies, but in some cases are based on human studies involving occupational exposures. Because these studies vary in applicability, the dosages used, the species and number of test organisms, and other factors, EPA assigns weight-of-evidence classifications corresponding to the likelihood that an agent is a human carcinogen. Appendix B.3 presents a more detailed discussion of carcinogenic endpoints and describes the weight-of-evidence categories.

2.5.4.2 Evaluation of Noncarcinogenic Effects

When the effects of noncarcinogenic chemicals are evaluated, it is assumed that an organism can tolerate a range of exposures from just above zero to some finite threshold value without appreciable risk of an adverse effect (EPA 1989d). This threshold value is generally expressed

	Soil RBSLs for Residential Land-Use Scenario (µg/g) [1]	
Analyte	Cancer Risk Endpoints	Noncancer Endpoints
Acetone	--	4,700
Antimony	--	31
Arsenic	0.36	2.3
Barium	--	5,200
Bis(2-ethylhexyl) phthalate	--	940
Chromium	140	390
Copper	--	2,900
Lead	--	400 [2]
Methylene Chloride	6.0	7.0
PPDDT	0.89	24
Thallium	--	6.2
Toluene	--	490
Total Petroleum Hydrocarbons	--	940
Trichloroethylene	0.32	--
Xylenes	--	320
Zinc	--	23,000

[1] This table lists RBSLs for all constituents detected in DPG Module 2 soil samples, with the exception of essential nutrients. RBSLs for cancer risk endpoints were derived assuming a target cancer risk of 10^{-6}. RBSLs for noncancer endpoints assume HQ of 1.0. The RBSLs were calculated based on the assumptions detailed in Table 2.5-1 and Appendix B.1 for soil ingestion, dermal contact, and inhalation exposure routes.

Analyte	Cancer Risk Endpoints	Noncancer Endpoints
Acetone	--	30,000
Antimony	--	730
Arsenic	2.9	550
Barium	--	100,000
Bis(2-ethylhexyl) phthalate	60	6,000
Chromium	230	9,200
Copper	--	68,000
Lead	--	400 [2]
Methylene Chloride	11	12
PPDDT	2.4	150
Thallium	--	150
Toluene	--	860
Total Petroleum Hydrocarbons	--	6,000
Trichloroethylene	0.59	--
Xylenes	--	540
Zinc	--	550,000

[1] RBSLs for cancer risk endpoints were derived assuming a target cancer risk of 10^{-6} except for arsenic (see Table 2.5-2). RBSLs for noncancer endpoints assume an HQ of 1.0. The RBSLs were calculated based on the assumptions detailed in Table 2.5-1 and Appendix B.2 for soil ingestion, dermal contact, and inhalation exposure routes.

[2] The RBSL for lead is the 400 mg/kg value recommended in EPA guidance (1994b).

-- Endpoint not applicable

Table 2.5-4 Toxicity Values for Module 2 COCs

	Carcinogenic effects				Noncarcinogenic effects					
Chemical	EPA carcinogen classification	Cancer slope factor $(mg/kg/day)^{-1}$	Type of cancer	Basis/source	Reference dose (RfD) (mg/kg/day)	Confidence level	Critical effect	Basis/source	Uncertainty/ modifying factors	Comments
ACET	--	--	--	--	0.1 (oral)	Low	Increased weight, nephrotoxicity	IRIS	100	Inhalation RfC not availab
B2EHP	B2	0.014 (oral)	Hepatocellular carcinomas and adenomas	oral administration	0.02 (oral)	Medium	Increased relative liver weight	Diet/IRIS 5-10-94	1000/1	No inhalation RfC data av Statistically significant inc relative liver weight obse treated female guinea pig
Ba	Not evaluated by EPA	--	--	--	0.07 (oral)	Medium	Increased blood pressure	Drinking Water/IRIS 5-9-94	3/1	Bantosis. Higher incidenc hypertension. Animals at dose exhibited ultrastructu changes in kidney glomer presence of myelin figure
CH2CL2	B2	0.0075	Hepatocellular adenomas or carcinomas (NTP) and hepatocellular cancer and neoplastic nodules	Inhalation/ Drinking water IRIS 5-10-94	0.06 (oral) 0.857 (inhalation)	Medium	Liver toxicity	Drinking water/IRIS 5-10-94	100/1	
CHRY	B2	0.0073 (oral)	Carcinoma, malignant lymphoma	Injection, Dermal/ IRIS 5-9-94 EPA	Not available	--	--	--	--	No RfC data available. P carcinomas and malignant lymphoma in mice. Chro abnormalities in hamster germ cells after gavage. Component of mixtures a with human cancer. Qu estimate of carcinogenic oral and inhalation expos available. See Health Ef Assessment Summary Ta
Cr	A	42 (inhalation)	Lung	Injection/IRIS 5-9-94	0.005 (oral) Limited to *metallic* chromium (VI) of soluble salts	Low	No effects reported	Drinking Water/IRIS 5-9-94	500/1	Based on chromium VI. concentrations accumulat considerable quantities of before pathological chan RfC under review. Oral not available. *Chromium* (hexavalent) compounds produced lung tumors in ingestion.

Table 2.5-4 Toxicity Values for Module 2 COCs

	Carcinogenic effects				Noncarcinogenic effects					
Chemical	EPA carcinogen classification	Cancer slope factor $(mg/kg/day)^{-1}$	Type of cancer	Basis/source	Reference dose (RfD) (mg/kg/day)	Confidence level	Critical effect	Basis/source	Uncertainty/ modifying factors	Comments
MEC6H5	D	--	--	IRIS 2/1/94	0.2 (oral) 0.114 (inhalation)	Medium	Changes in liver and kidney weights	IRIS 4/1/94	1000/1	NOAEL: 312 mg/kg LOAEL: 625 mg/kg 13 week rat gavage stu
Pb	B2	Recommended that numerical estimate not be used	Bilateral renal carcinoma (lead salts), stomach, and lung	Oral/IRIS 5-9-94	Determined to be inappropriat e for lead	--	--	IRIS 5-9-94	--	RfC – no data. Signifi increases in renal tumo dietary and subcutaneo to soluble lead salts. E carcinoma in rats/mice. Quantitative estimate o carcinogenic risk from exposure or inhalation available.
PPDDT	B2	0.34 (oral)	Hepato-cellular adenomas and carcinomas, lung tumors	Diet/IRIS 5-9-94	0.0005 (oral)	Medium	Liver lesions	Diet/ IRIS 5-9-94	100/1	No inhalation RfC data Liver cell alteration an storage in fat of rat ind dietary levels of 1-50 Inhalation unit risk = 0 $\mu g/m^3$ Calculated from oral d SF for inhalation = 0.3 $(mg/kg/day)^{-1}$.
PYR	--	--	--	--	0.03 (oral)	Medium	Kidney effects	IRIS	300	Inhalation RfC not ava
Sb	--	--	--	--	0.0004 (oral)	Medium	Increased mortality, altered chemistries	IRIS	1000	Inhalation RfC not ava
XYLEN	D	--	--	IRIS 3/1/91	2.0 (oral)	Medium	Hyperactivity decreased body weight and increased mortality in males	HEAST 1993	100/1	Inhalation RfC pending
ZN	--	--	--	--	0.3 (oral)	Medium	Decreased blood enzyme	IRIS	3	Inhalation RfC not ava

Analyte acronyms are defined in the Acronym List.

subpopulations, can sustain without an unacceptable risk of deleterious effects during a lifetime (EPA 1989d). Like SFs, RfDs are usually derived from animal studies, but they are in some cases based on human studies involving occupational exposures. These experimental or epidemiological data are then adjusted using a range of uncertainty factors. The RfDs thereby provide a benchmark to which chemical intakes by various exposure routes may be compared.

2.5.4.3 Toxicity Values Used in the Risk Assessment

The chemical-specific SF and RfD values used in the risk assessment are documented in the RBSL calculation tables provided in Appendices B.1 and B.2. These tables list toxicity values for both oral and inhalation endpoints. In accordance with EPA guidance (1992a), the oral toxicity factors (SFs and RfDs) were also used to calculate dermal exposure pathway risks. Oral data were not adjusted for dermal exposures given the uncertainties in the chemical-specific absorption data and the fact that adjustment using default absorption factors often yields anomalous results.

Using the methods documented in Section 2.5.5 below, the toxicity values listed in Appendix B.1 and B.2 were combined with the exposure assumptions (Table 2.5-1) to calculate RBSLs and associated risks. Appendix B.3 presents a more detailed description of the methods used to assess carcinogenic and noncarcinogenic endpoints and provides toxicity profiles for the COPCs evaluated.

2.5.5 Risk Characterization

This section presents the methods used in the human health risk assessment that apply to all SWMU-specific evaluations. It describes the general methodology used to calculate risks; identifies the methods used to calculate the RBSLs that formed the basis for the cancer risk and noncancer (HI) calculations; and discusses the methods used to calculate cancer and noncancer risks using the RBSLs. Finally, it describes the context within which the results of the human health risk assessment should be interpreted.

2.5.5.1 General Methodology

As discussed in Section 2.5.1, risks for the Module 2 SWMU evaluations were calculated using RBSLs. RBSLs are chemical- and medium-specific concentrations that are considered protective of human health given a defined set of exposure and toxicity assumptions. For carcinogens, RBSLs are defined as concentrations protective of human health at a cancer risk level of 10^{-6}. For noncarcinogens, RBSLs are defined as concentrations unlikely to pose adverse health effects based on an HQ of 1.0. Tables 2.5-2 and 2.5-3 list the soil RBSLs developed for the residential and industrial land-use scenarios. These tables list RBSLs for every constituent that was detected in the Module 2 soil samples.

2.5.5.2 RBSL Development

The soil RBSLs listed in Tables 2.5-2 and 2.5-3 were calculated using the equations and assumptions documented in Appendix B.1 and B.2 for residential and industrial land uses, respectively. These equations incorporate factors that quantify the assumed intakes for the soil ingestion, dermal absorption, and vapor and particulate inhalation pathways. The parameters used in these equations are defined in Table 2.5-1.

2.5.5.3 Characterization of Cancer Risks Using RBSLs

Excess cancer risks associated with exposures to known or potentially carcinogenic COPCs are defined as risks in excess of the normal cancer "burden" in a population. These estimates represent the upper-bound probability that an individual exposed to a given level of contaminant over a lifetime will develop cancer as a result of those exposures. A 10^{-6} upper-bound excess lifetime cancer risk is an increase of 1 in 1 million in the probability that an exposed individual will develop cancer. For this evaluation, cancer risks were calculated using the following equation (Equation 2-1):

$$Risk_i = \frac{EPC_i}{RBSL_i} * TCR \qquad (2\text{-}1)$$

where:

$Risk_i$ = Cancer risk for chemical i, the unitless probability that an individual will develop cancer attributable to the assumed exposure scenario

EPC_i = Exposure point concentration for chemical i in soil (μg/g)

$RBSL_i$ = Risk-based screening level for chemical i in soil (μg/g)

TCR = Target cancer risk (10^{-6})

The 10^{-6} reference risk level included in this equation accounts for the fact that the RBSL term was originally calculated assuming a 10^{-6} risk level. This approach to calculating risk is equivalent to that used in standard risk assessment evaluations (i.e., as outlined in the Risk Assessment Guidance for Superfund [EPA 1989b]), in that risk is estimated as the product of the exposure point concentration, the intake rate, and the cancer SF (EPA 1989a). The term "1/RBSL * TCR" in Equation 2-1 is equivalent to the product of the intake rate and the SF; thus the two approaches yield the same result. The total cancer risk associated with a given SWMU is then calculated by adding the chemical-specific risks.

2.5.5.4 Characterization of Noncancer HIs Using RBSLs

As discussed in Section 2.5.4.2, potential noncarcinogenic effects associated with exposures to COPCs are evaluated using RfDs. These criteria are estimates of the daily chemical exposures that present an acceptably low risk of adverse effects to an individual over a specified exposure duration. The ratio of the chemical-specific intake to the RfD is referred to as the HQ. Mixtures of chemicals are assessed by means of the HI, which is defined as the sum of the chemical-specific HQs derived for each noncarcinogenic COPC. Any single chemical with an exposure level greater than the RfD would cause both the chemical-specific HQ and the cumulative HI to exceed 1.0, indicating potential health risks of concern.

In this evaluation, HQs are calculated by dividing the chemical-specific EPC by the soil RBSL, the chemical concentration in soil for which no adverse health effect is anticipated. An HQ is computed separately for each COPC as shown below (Equation 2-2):

$$HQ_i = \frac{EPC_i}{RBSL_i} \qquad (2\text{-}2)$$

where:

HQ_i = Hazard quotient for chemical i

EPC = Exposure point concentration in soil for chemical i (μg/g)

$RBSL_i$ = Risk-based screening level for chemical i (μg/g)

This calculation is equivalent to more standard approaches in which the hazard quotient is calculated as the ratio of the chronic daily intake rate to the RfD (EPA 1989a).

The total (additive) noncancer health threat is expressed by the HI and is computationally equivalent to the sum of the HQs. For multiple chemical exposures, the HI can exceed the 1.0 target criterion even if no single chemical HQ exceeds 1.0. However, the assumption of additivity reflected in the HI equation is properly applied only to compounds that induce the same effect by the same mechanism. Consequently, applying this equation to compounds that are not expected to induce the same type of effects could overestimate the potential for adverse health effects.

2.5.5.5 Interpretation of Risk Assessment Results

UAC R315-101 contains the following provisions and requirements according to the results of the human health risk evaluation:

- A SWMU qualifies for risk-based (clean) closure if and only if the excess cancer risk is less than 10^{-6} and the noncancer HI is less than 1.0 for residential use.

This provision means that if no appreciable health risks would result from exposures during residential use of a site, no site controls or corrective action is required before or after the unit is closed. This result is conclusive because other exposure scenarios would likely result in even lower risks. If the noncancer HI is 1.0 or greater, clean closure is not permitted. If the cancer

risk is greater than 10^{-6}, the closure requirements are based on the results of the human health risk evaluation for the actual, or current, land-use scenario:

- Site controls are required if the cancer risk associated with the actual land use is less than 10^{-4}, provided that the HI is less than 1.0. Corrective action is optional.
- Corrective action is required if the cancer risk is greater than 10^{-4} or the HI is greater than 1.0 for exposures due to actual use.

These requirements are similar to those established by EPA for the Superfund program under the National Contingency Plan (EPA 1990). This federal guidance states that the target risk range for carcinogens is a 10^{-6} to 10^{-4} incremental cancer risk, and that for noncarcinogens, where the HI exceeds 1.0, assumed exposures may present a health hazard and therefore warrant further evaluation.

2.5.6 Evaluation of Uncertainties

Risk assessment is an inexact but essential methodology used to characterize and quantify health effects potentially resulting from exposures to chemicals. The lack of relevant toxicity and exposure data, the uncertainty in chemical measurements in both the environment and the laboratory, and the need to extrapolate experimental endpoints to assumed human exposures make precise quantification of risk difficult and inherently uncertain. For example, the assumptions used to calculate exposure rates are by nature imprecise given variations in human behavior and physical characteristics. Given these uncertainties, which are described in detail in the preceding sections, the general approach applied in this assessment is to develop conservative RMEs of contaminant exposures and doses.

2.5.6.1 Uncertainties Associated with the Identification of Soil COPCs

As discussed in Section 2.5.1, analytical results for the Module 2 SWMUs do not indicate any significant contamination of soil or other environmental media. This general lack of contamination is not unexpected given that historical records do not indicate that any releases of hazardous substances have occurred at these SWMUs. Consequently, many of the COPCs defined for each SWMU, which were identified using the selection criteria outlined in Figure 2.5-2, are not likely to be site-related contaminants. The relatively small number of data points available

for SWMUs 20, 164, 166, and 170 is not considered a data gap given the small size of these SWMUs (500 to 750 ft^2) and the lack of evidence of contaminant releases.

2.5.6.2 Uncertainties Associated with the Exposure Assessment

Numerous uncertainties are associated with the exposure assessment. For example, assumptions regarding exposure frequency, duration, and anticipated contact with contaminated media are inherently uncertain since the behavior patterns of target receptors are not well known. These factors vary depending on site-specific characteristics and are extremely difficult either to measure or to verify. The values used in this analysis were developed to reflect the highest level of exposure and risk that could reasonably be expected to occur. Table 2.5-1 indicates that, for most parameters, default guidance values were used (EPA 1991; EPA 1992a; EPA 1994a).

One of the central tenets of risk assessment is that in order for a risk to be posed, a complete exposure pathway must exist. At the Module 2 SWMUs, there are many factors that would serve to preclude or markedly reduce potential exposures to constituents in soil (see Section 2.5.3.1). With few exceptions, this finding would also likely apply to future land uses at DPG. Consequently, the exposure assumptions used to derive RBSLs and risks (Table 2.5-1) are considered theoretical upper-bound estimates.

2.5.6.3 Uncertainties Associated with the Toxicity Assessment

The uncertainty associated with toxicity factors has a significant effect on the estimated RBSLs and risks. The toxicity values specified by EPA are based on numerous assumptions that tend to overestimate risks. For example, in deriving cancer SFs, the dose-response relationship is assumed to be the same for both test animals and humans. Carcinogenicity tests are performed on animals and the results are extrapolated to humans, thus introducing uncertainty and potential overestimation. In addition, the study protocol for some cancer toxicity studies is to select a test animal species known to be particularly sensitive (prone to develop cancer) to ensure that a carcinogenic effect will be observed, if it is to occur. Additionally, these factors represent upper-bound (95 percent UCL) estimates of potency (EPA 1994d). Thus, if an individual's exposure to a constituent is equivalent to the level that defines the potency, there is only a 5 percent chance that the actual risk to that individual will exceed the calculated risk, and a 95

percent chance that the risk is at or below the calculated level. Consequently, the actual risks associated with exposures to a potential carcinogen are not likely to exceed the risk estimated using these upper-bound slope factors, and in fact may be lower.

Similar conservatism is reflected in the RfDs developed for noncancer endpoints. As discussed in Section 2.5.4.2 and Appendix B.3, RfDs are derived by applying uncertainty factors to the underlying data. Application of these factors is often necessary (given data gaps), but may lead to an acceptable dose that is orders of magnitude less than is indicated on the basis of empirical evidence.

2.6 PRELIMINARY ECOLOGICAL RISK ASSESSMENT

2.6.1 General Approach

A preliminary ecological risk assessment is performed in order to determine the presence of potential risk and to identify critical exposure pathways and contaminants of concern. If necessary, this information can then be used to determine the extent of potential risk to appropriate receptors while evaluating more specific site information in a baseline ecological risk assessment. Preliminary assessments incorporate conservative assumptions to identify maximum levels of potential risk, and thus reduce the likelihood of overlooking potential risks. The objective of this preliminary assessment is to determine the presence or absence of potential risk to a conservative mammalian and avian representative receptor of DPG at the four Module 2 SWMUs.

2.6.2 Methodology

2.6.2.1 Receptor Identification

The Module 2 preliminary ecological risk assessment was designed to be conservative by assessing potential risk to wildlife species with the suspected highest potential for contaminant exposure. The deer mouse was considered an appropriate representative and conservative mammalian receptor for evaluation in the preliminary risk assessment because of its ubiquitous distribution, relatively small home range, intimate soil contact, varied diet, and average sensitivity to the contaminants of concern. The horned lark was considered an appropriate representative

and conservative avian receptor for evaluation in the preliminary risk assessment for the same reasons, with the exception of intimate soil contact. Top-level predators are not expected to be significantly exposed to the low levels of contamination at the Module 2 SWMUs due to the small size of these SWMUs relative to top-level predator home ranges, lack of suitable habitat, and the absence of suitable prey species.

The deer mouse is considered to be nearly ubiquitous at DPG, with occurrence in most habitats, except the salt flats of the western portion of the base. Compared to other potential mammalian receptors, this species has a relatively small home range of approximately 0.11 hectares (ha) (Bowers and Smith 1979). Such a small home range increases the likelihood that the deer mouse will be exposed to any chemical contamination when the home range is within contaminated areas, and reduces dilution of exposure from roaming in non-contaminated areas. Deer mice also have intimate contact with the soil surface as the result of their feeding behavior. This behavior is likely to increase their exposure to any contaminants in DPG soils. Also, this species has a varied diet consisting of grasses, seeds, and insects. The varied diet increases the potential exposure of contaminants to the deer mouse by increasing the number of potentially contaminated food items that are consumed. Finally, the deer mouse has an average sensitivity to the COPCs relative to other mammalian species based on available information. The toxicity benchmark values used in the preliminary ecological risk assessment were derived from either the lab mouse, lab rat, or mink as test animals. Although these conservative characteristics may apply to some other potential receptors at DPG, the combination of all four characteristics in the deer mouse support its selection for evaluation in the preliminary assessment.

The horned lark is also considered to be nearly ubiquitous at DPG, with occurrence in most habitats, except the salt flats of the western portion of the base. Additionally, the horned lark is a permanent year-round resident of DPG, which maximizes its potential exposure to chemical contamination. Compared to other potential avian receptors, this species has a relatively small home range of approximately 1.6 ha (Verbeek 1967). Such a small home range increases the likelihood that the horned lark will be exposed to any chemical contamination when the home range is within contaminated areas, and reduces dilution of exposure from foraging in non-contaminated areas. Horned larks also have a soil ingestion exposure route as the result of their

feeding behavior. The horned lark, as a granivorous passerine, ingests soil as part of its diet of seeds and insects. This behavior is likely to increase its exposure to any contaminants in DPG soils. The varied diet increases the potential exposure of contaminants to the horned lark by increasing the number of potentially contaminated food items that are consumed. Finally, the horned lark has an average sensitivity to the COPCs relative to other avian species based on available information. The toxicity benchmark values used in the preliminary ecological risk assessment were derived from various avian species used as test animals.

2.6.2.2 Selection of COPCs

To maintain conservatism, all detected chemicals in surficial soil were evaluated in the preliminary assessment (Tables 2.6-1 through 2.6-8). With few exceptions, flora and fauna receive the maximum exposure to soil contaminants within the first foot of soil.

2.6.2.3 Risk Calculation

SWMU-specific exposure estimates for the deer mouse and horned lark were made using the maximum detected surficial soil concentration at each SWMU and conservative estimates of food intake rate, exposure area, bioavailability, and body weight. The concentration term was made very conservative by assuming that the maximum detected soil concentration is the concentration in soil throughout the SWMU. The food intake rate was determined using a formula provided by the Wildlife Exposure Factors Handbook (EPA 1993; Eq. 3-8 for mammal, Eq. 3-4 for avian) that incorporates the body weight of the wildlife receptor species and two additional conversion factors. The exposure area represents the proportion of the home range that overlaps with the area of contamination. This proportion is assumed to be 100 percent; in other words, the animal's home range is completely contained within the contaminated area. Additional conservatism was thereby provided by eliminating any dilution from uncontaminated areas that could actually be within the animal's home range. Bioavailability was assumed to be complete for all of the COPCs, and a value of 1.0 (100 percent) was used in the equation. Toxicological studies have demonstrated that pre-adult life stages tend to be the most sensitive. Exposure is calculated for pre-adults, and an uncertainty factor of 2 was multiplied by the product of all other factors already described when the study did not include the most sensitive life stage. All of these exposure factors were divided by the body weight of the wildlife species in the following manner:

$$Exposure,\ mg\ /\ kg\ -\ BW\ /\ d = \frac{(SC_{max})\ x\ (FIR_r)\ x\ (ExpA)\ x\ (BA)\ x\ (LS)}{(BW_r)} \quad (2\text{-}2)$$

where:

SC_{max} = maximum soil concentration, mg / kg,

FIR_r = food ingestion rate for the receptor, kg / d,

$ExpA$ = exposure area (1),

BA = bioavailability (1),

LS = life stage used in study (1 = non-adult, 2 = adult), and

BW_r = body weight of the receptor, kg.

To determine a safe dose level specific to the deer mouse and horned lark, doses from scientific studies resulting in a no observable adverse effect level were used. All values were found in the document Toxicological Benchmarks for Wildlife: 1996 Revision (Sample et al. 1996). The HQ was computed by dividing the estimated chemical exposure dose rate from the site by a safe chemical dose rate from the scientific literature. The resulting quotient was used as a tool to identify potential risk to biological receptors. Computed HQs greater than 1 were considered to represent potentially harmful effects to the chosen biological receptors.

The HQs should not be presented as stand-alone values, but instead should be evaluated in light of site-specific conditions such as habitat abundance and quality, receptor occurrence and abundance, and human activity. The presence or absence of suitable habitat is an important consideration when interpreting potential risk. The presence of suitable habitat and its abundance relative to the size of the contaminated area, as well as the habitat quality within the contaminated area relative to outside, can influence the possibility that potential risk will be realized. Large-scale ecological risk assessments often consider potential risk to a species that has not been confirmed to occur in a particular locale. Information from local biologists and scientific literature can identify the likelihood of a species' occurrence in a specific locale and, thus, the plausibility of potential exposure and subsequent risk. Human activity can have a significant effect on the presence of a receptor in an area. Heavy machinery, vehicle traffic, and general proximity to

habitat.

For preliminary ecological risk assessments, conservative assumptions are used to safeguard against the underestimation of potential risk. In the preliminary assessment for the Dugway Module 2 SWMUs, conservatism was employed in the following components in the calculation of HQs:

1. The maximum soil concentration for each detected COPC was used to represent the concentration term for the entire SWMU, whereas, over most of the SWMU area, the contaminant levels are likely to be lower.

2. The chemical concentration in food was estimated to be equal to that in soil. Excluding those that bioaccumulate, concentrations of many contaminants in food (i.e., plant and animal matter) are typically only a small fraction of their concentrations in soil.
3. The receptor's home range, or foraging area, is assumed to be completely contained within the area of contamination or SWMU. This assumption may result in a conservative overestimation of risk, because an animal's true home range probably includes uncontaminated areas outside of the SWMU.

4. Bioavailability of a contaminant in ingested soil or food is assumed to be 100 percent in the determination of exposure for chemicals absorbed by an animal. This is extremely conservative for most metals, since metals typically become complexed to soil particles or become oxidized. The bioavailability of complexed and oxidized metals for mammals such as the deer mouse is normally considered to be 10 percent or lower based on some human studies. This conservative assumption, therefore, represents an overestimation of exposure.

Since they are intentionally calculated in a way that tends to overestimate risk, the HQs that are calculated in a preliminary ecological risk assessment are more appropriately used to discern which chemicals do not pose a substantive risk to the receptors (i.e., the HQs < 1), rather than providing conclusive information by which to judge those chemicals that may pose risks to the receptors (i.e., the HQs > 1). Considering the application of conservative exposure and toxicity assumptions, the HQs may be interpreted on the basis of professional judgment as follows: HQs less than or equal to 10 can be considered to pose negligible risk to the target receptor for that chemical; HQs that are greater than 10 but less than 100 may indicate some potential risk; and HQs that are greater than 100 are considered to indicate likely potential risk.

the HQs greater than unity must then be evaluated considering all available information pertaining to a site. This evaluation can ensure that recommendations and subsequent actions can be made to be adequately protective without being unnecessarily costly or disruptive to habitat. In addition, remedial measures that destroy habitat, such as scraping, excavating, or capping, may cause more harm to target receptors such as the deer mouse than the chemical contamination. For this reason, even a SWMU with HQs greater than 100 may not be recommended for corrective action if 1) the SWMU is not a wildlife attractant; 2) there is other suitable good-quality habitat in abundance within the local area that will support the receptor populations; and 3) the location is not surrounded by other nearby SWMUs with predicted risks that might create an unmeasured cumulative adverse impact. Conditions specific to particular SWMUs that need to be considered are discussed in the respective SWMU sections.

2.6.3 Ecological Risk Results

Risk was computed for the deer mouse and horned lark at the four Module 2 SWMUs (Tables 2.6-1 through 2.6-8). The only chemicals causing predicted potential total risk were naturally-occurring inorganic chemicals that did not appear to be related to the use history of any of the SWMUs. The highest potential total risk to the deer mouse from any one chemical from any SWMU was an HQ of 736, from aluminum at SWMU 170. Many HQs (50 percent) were less than 100 but greater than 10; some HQs (18 percent) were less than or equal to 10. All SWMUs had estimates of potential total risk to the deer mouse greater than 100 for some chemicals, always from aluminum and usually from thallium. As previously described, an HQ greater than 100 but less than 1000 is considered to indicate likely potential risk to the receptor. However, since all detected analytes were included in the risk characterization, a substantial component of total risk relates to trace metals that occur naturally in the environment.

Potential total risk to the horned lark from any one chemical from any SWMU never exceeded an HQ of 100 (highest HQ = 53, mercury, at SWMU 170), and some HQs were less than 50 but greater than 10 (21 percent); most HQs (75 percent) were less than or equal to 10. All SWMUs had estimates of potential total risk to the horned lark greater than 10 for some chemicals, always from aluminum. As previously described, HQs less than or equal to 10 can be considered to pose

Table 2.6-1. Preliminary Exposure Estimates and Risk Calculations Based on NOAEL Dose Rates (mg/kgbw/day) for the Deer Mouse at SWMU 20, Dugway Proving Ground, Dugway, Utah.

Chemical	Max. Soil Conc. (mg/kg)	Background Soil Conc. (mg/kg)	No. Detects > Bkgd	Exposure Estimate for Soil (mg/kg-BW/day)^	Screening-Level Value Diet (mg/kg-BW/day)^^ NOAEL - w	Hazard Quotient Diet~	Risk Due to Conc. > Bkgd?
AL	7800	19,000	0	1404	2.2	631	No
AS	17	13	3	3.1	0.15	21	Yes
B2EHP	0.24	NA	NA	0.043	21	0.002	NA
BA	650	400	4	117	2	55	Yes
BE	0.66	1	0	0.12	1.4	0.1	No
CA	210,000	190,000	1	37,800	NAv	NAv	NAv
CO	3.6	7.9	0	0.65	NAv	NAv	NAv
CR	7.1	17	0	1.3	5830	0.0002	No
CU	11	25	0	2.0	32	0.06	No
FE	7800	19,000	0	1404	NAv	NAv	NAv
K	3900	7400	0	702	NAv	NAv	NAv
MEC6H5	0.51	NA	NA	0.092	30	0.003	NA
MG	31,000	29,000	1	5580	NAv	NAv	NAv
MN	260	520	0	47	187	0.2	No
NA	7600	11,000	0	1368	NAv	NAv	NAv
NI	7.7	17	0	1.4	85	0.02	No
PB	6.8	14	0	1.2	17	0.07	No
SB	11	9.1	1	2.0	0.14	14	Yes
TL	20	35	0	3.6	0.008	452	No
V	22	29	0	4.0	0.42	10	No
ZN	26	59	0	4.7	341	0.01	No

NA = Not Applicable NAv = Not Available NOAEL = no observable adverse effect level

^ Exposure Estimates = product of maximum soil concentration, food intake rate (0.00306 kg/d, where chemical concentration in food is assumed to be the same as soil; food intake rate = [0.621 x (body wt. in grams)^0.564]/1000, Eq. 3-8, EPA 1993), exposure area/period (100%), bioavailability (100%), and life stage used (i.e., 1 if non-adult or during reproduction, 2 if adult); all divided by body wt. (0.017 kg, arith. mean of 46 individuals collected at DPG 1995).

^^ Screening-level Values (mg/kg-body wt./day) = extrapolated from the test animal (all body weights were provided in study summaries in Sample et al. 1996; mink = 1kg, EPA 1993) to the deer mouse (0.017 kg, arith. mean of 46 collected individuals from DPG 1995) using the formula NOAEL-w = NOAEL-t x (body wt.-t / body wt.-w)^1/4, where w = wildlife species and t = test species (Eq.4, Sample et al. 1996). The NOAEL-t values from Sample et al. 1996.

~ Hazard Quotient = exposure estimate divided by the screening-level ecotoxicity value.

Bold = metal concentrations above background on the basis of the ANOVA

Table 2.6-2. Preliminary Exposure Estimates and Risk Calculations Based on NOAEL Dose Rates (mg/kgbw/day) for the Horned Lark at SWMU 20, Dugway Proving Ground, Dugway, Utah.

Chemical	Max. Soil Conc. (mg/kg)	Background Soil Conc. (mg/kg)	No. Detects > Bkgd	Exposure Estimate for Soil (mg/kg-BW/day)^	Screening-Level Value Diet (mg/kg-BW/day)^^ NOAEL - w	Hazard Quotient Diet~	Risk Due to Conc Bkgd?
AL	7800	19,000	0	1883	110	17	No
AS	17	13	3	4	3	2	Yes
B2EHP	0.24	NA	NA	0.058	1.1	0.05	NA
BA	650	400	4	157	21	8	Yes
BE	0.66	1	0	0.16	NAv	NAv	No
CA	210,000	190,000	1	50,700	NAv	NAv	NAv
CO	3.6	7.9	0	0.87	NAv	NAv	NAv
CR	7.1	17	0	1.7	1.0	2	No
CU	11	25	0	2.7	47	0.06	No
FE	7800	19,000	0	1883	NAv	NAv	NAv
K	3900	7400	0	942	NAv	NAv	NAv
MEC6H5	0.51	NA	NA	0.12	NAv	NAv	NA
MG	31,000	29,000	1	7484	NAv	NAv	NAv
MN	260	520	0	63	977	0.1	No
NA	7600	11,000	0	1835	NAv	NAv	NAv
NI	7.7	17	0	1.9	77	0.02	No
PB	6.8	14	0	1.6	1.1	1	No
SB	11	9.1	1	2.7	NAv	NAv	Yes
TL	20	35	0	4.8	NAv	NAv	No
V	22	29	0	5.3	6	0.9	No
ZN	26	59	0	6.3	15	0.4	No

NA = Not Applicable NAv = Not Available NOAEL = no observable adverse effect level

^ Exposure Estimates = product of maximum soil concentration, food intake rate (0.00306 kg/d, where chemical concentration in food is assumed to be the same as soil; food intake rate = [0.398 x (body wt. in grams)^0.850]/1000, Eq. 3-4, EPA 1993), exposure area/period (100%), bioavailability (100%), and life stage used (i.e., 1 if non-adult or during reproduction, 2 if adult); all divided by body wt. (0.017 kg, arith. mean of 46 individuals collected at DPG 1995).

^^ Screening-level Values (mg/kg-body wt./day) = NOAELW = NOAELT (where w = wildlife, t = test animal, Eq. 6, Sample et al. 1996). All body weights were provided in study summaries in Sample et al. 1996.

~ Hazard Quotient = exposure estimate divided by the screening-level ecotoxicity value.

Bold = metal concentrations above background on the basis of the ANOVA

Table 2.6-3. Preliminary Exposure Estimates and Risk Calculations Based on NOAEL Dose Rates (mg/kgbw/day) for the Deer Mouse at SWMU 164, Dugway Proving Ground, Dugway, Utah.

Chemical	Max. Soil Conc. (mg/kg)	Background Soil Conc. (mg/kg)	No. Detects > Bkgd	Exposure Estimate for Soil (mg/kg-BW/day)^	Screening-Level Value Diet (mg/kg-BW/day)^^ NOAEL - w	Hazard Quotient Diet~	Risk Due to Cor Bkgd?
ACET	0.043	NA	NA	0.0077	11	0.0007	NA
AL	6800	19,000	0	1224	2.2	550	No
AS	14	13	1	2.5	0.15	17	Yes
B2EHP	0.69	NA	NA	0.12	21	0.01	NA
BA	270	400	0	49	2	23	No
BAPYR	0.065	NA	NA	0.012	1.2	0.01	NA
BE	0.84	1	0	0.15	1.4	0.1	No
CA	150,000	190,000	0	27,000	NA	NAv	NAv
CD	2.4	0.46	2	0.43	2.1	0.2	Yes
CHRYSENE	0.062	NA	NA	0.011	NA	NAv	NA
CO	3.3	7.9	0	0.59	NA	NAv	NAv
CR	23	17	2	4.1	5830	0.001	Yes
CU	27	25	1	4.9	32	0.1	Yes
FE	9500	19,000	0	1710	NA	NAv	NAv
HG*	0.07	0.073	0	0.013	2.8	0.1	No
K	2800	7400	0	504	NA	NAv	NAv
MEC6H5	0.0034	NA	NA	0.00061	30	0.00002	NA
MG	20,000	29,000	0	3600	NA	NAv	NAv
MN	300	520	0	54	187	0.3	No
NA	1600	11,000	0	288	NA	NAv	NAv
NI	14	17	0	2.5	85	0.03	No
PB	81	14	2	15	17	0.9	Yes
SB	9.2	9.1	1	1.7	0.14	11	Yes
TL	28	35	0	5.0	0.01	633	No
V	19	29	0	3.4	0.42	8	No
XYLENE	0.0016	NA	NA	0.00029	2.4	0.0001	NA
ZN	110	59	2	19.8	341	0.06	Yes

NA = Not Applicable NAv = Not Available NOAEL = no observable adverse effect level

^ Exposure Estimates = product of maximum soil concentration, food intake rate (0.00306 kg/d, where chemical concentration in food is assumed to be the same as soil; food intake rate = [0.621 x (body wt. in grams)^0.564]/1000, Eq. 3-8, EPA 1993), exposure area/period (100%), bioavailability (100%), and life stage used (i.e., 1 if non-adult or during reproduction, 2 if adult); all divided by body wt. (0.017 kg, arith. mean of 46 individuals collected at DPG 1995).

^^ Screening-level Values (mg/kg-body wt./day) = extrapolated from the test animal (all body weights were provided in study summaries in Sample et al. 1996; mink = 1kg, EPA 1993) to the deer mouse (0.017 kg, arith. mean of 46 collected individuals from DPG 1995) using the formula NOAEL-w = NOAEL-t x (body wt.-t / body wt.-w)^1/4, where w = wildlife species and t = test species (Eq.4, Sample et al. 1996). The NOAEL-t values from Sample et al. 1996.

~ Hazard Quotient = exposure estimate divided by the screening-level ecotoxicity value.

* Because Hg bioaccumulates, the HQ of 0.004 was multiplied by a calculated BAF of 22.5 from mink liver (Wren et al. 1987).

Bold = metal concentrations above background on the basis of the ANOVA

Table 2.6-4. Preliminary Exposure Estimates and Risk Calculations Based on NOAEL Dose Rates (mg/kgbw/day) for the Horned Lark at SWMU 164, Dugway Proving Ground, Dugway, Utah.

Chemical	Max. Soil Conc. (mg/kg)	Background Soil Conc. (mg/kg)	No. Detects > Bkgd	Exposure Estimate for Soil (mg/kg-BW/day)^	Screening-Level Value Diet (mg/kg-BW/day)^^ NOAEL - w	Hazard Quotient Diet~	Risk Due to Conc. > Bkgd?
ACET	0.043	NA	NA	0.010	NA	NAv	NA
AL	6800	19,000	0	1642	110	15	No
AS	14	13	1	3.4	3	1	Yes
B2EHP	0.69	NA	NA	0.17	1.1	0.2	NA
BA	270	400	0	65	21	3	No
BAPYR	0.065	NA	NA	0.016	NA	NAv	NA
BE	0.84	1	0	0.20	NA	NAv	No
CA	150,000	190,000	0	36,214	NA	NAv	NAv
CD	2	0.46	2	0.6	1	0.4	Yes
CHRYSENE	0.062	NA	NA	0.015	NA	NAv	NA
CO	3.3	7.9	0	0.8	NA	NAv	NAv
CR	23	17	2	5.6	1	6	Yes
CU	27	25	1	6.5	47	0.1	Yes
FE	9500	19,000	0	2294	NA	NAv	NAv
HG*	0.07	0.073	0	0.02	0.01	3	No
K	2800	7400	0	676	NA	NAv	NAv
MEC6H5	0.0034	NA	NA	0.0008	NA	NAv	NA
MG	20,000	29,000	0	4829	NA	NAv	NAv
MN	300	520	0	72	977	0.1	No
NA	1600	11,000	0	386	NA	NAv	NAv
NI	14	17	0	3.4	77	0.04	No
PB	81	14	2	20	1	17	Yes
SB	9.2	4.6	1	2	NA	NAv	NAv
TL	28	35	0	6.8	NA	NAv	No
V	19	29	0	4.6	6	0.8	No
XYLENE	0.0016	NA	NA	0.0004	NA	NAv	NA
ZN	110	59	2	27	15	2	Yes

NA = Not Applicable NAv = Not Available NOAEL = no observable adverse effect level

^ Exposure Estimates = product of maximum soil concentration, food intake rate (0.00306 kg/d, where chemical concentration in food is assumed to be the same as soil; food intake rate = [0.398 x (body wt. in grams)^0.850]/1000, Eq. 3-4, EPA 1993), exposure area/period (100%), bioavailability (100%), and life stage used (i.e., 1 if non-adult or during reproduction, 2 if adult); all divided by body wt. (0.017 kg, arith. mean of 46 individuals collected at DPG 1995).

^^ Screening-level Values (mg/kg-body wt./day) = NOAELw = NOAELt (where w = wildlife, t = test animal, Eq. 6, Sample et al. 1996).

All body weights were provided in study summaries in Sample et al. 1996.

~ Hazard Quotient = exposure estimate divided by the screening-level ecotoxicity value.

* Although it is acknowledged that mercury may bioaccumulate in some ecosystems, an avian BAF value greater than 1 was not readily available in the literature.

Bold = metal concentrations above background on the basis of the ANOVA

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Table 2.6-5. Preliminary Exposure Estimates and Risk Calculations Based on NOAEL Dose Rates (mg/kgbw/day) for the Deer Mouse at SWMU 166, Dugway Proving Ground, Dugway, Utah.

Chemical	Max. Soil Conc. (mg/kg)	Background Soil Conc. (mg/kg)	No. Detects > Bkgd	Exposure Estimate for Soil (mg/kg-BW/day)^	Screening-Level Value Diet (mg/kg-BW/day)^^ NOAEL - w	Hazard Quotient Diet~	Risk Due to Conc. > Bkgd?
ACET	0.045	NA	NA	0.0081	11	0.0008	NA
AL	7400	19,000	0	1332	2	599	No
AS	10	13	0	1.8	0.1	12	No
BA	300	400	0	54	2	26	No
BE	0.74	1	0	0.13	1	0.1	No
CA	200,000	190,000	1	36,000	NAv	NAv	NAv
CD	3	0.46	1	0.47	2	0.2	Yes
CH2CL2	0.0045	NA	NA	0.0008	12	0.0000650	NA
CO	4.4	7.9	0	0.79	NAv	NAv	NAv
CR	11	17	0	2.0	5830	0.0003	No
CU	14	25	0	2.5	32	0.1	No
FE	9300	19,000	0	1674	NAv	NAv	NAv
K	2900	7400	0	522	NAv	NAv	NAv
MEC6H5	0.013	NA	NA	0.0023	30	0.0001	NA
MG	42,000	29,000	1	7560	NAv	NAv	NAv
MN	310	520	0	56	187	0.3	No
NA	910	11,000	0	164	NAv	NAv	NAv
NI	9.5	17	0	1.7	85	0.02	No
PB	36	14	1	6.5	17	0.4	Yes
SB	8.6	9.1	0	1.5	0.1	11	No
TL	9.5	35	0	1.7	0.01	215	No
V	20	29	0	3.6	0.4	9	No
XYLEN	0.0083	NA	NA	0.0015	2	0.001	NA
ZN	110	59	1	20	341	0.1	Yes

NA = Not Applicable NAv = Not Available NOAEL = no observable adverse effect level

^ Exposure Estimates = product of maximum soil concentration, food intake rate (0.00306 kg/d, where chemical concentration in food is assumed to be the same as soil; food intake rate = [0.621 x (body wt. in grams)^0.564]/1000, Eq. 3-8, EPA 1993), exposure area/period (100%), bioavailability (100%), and life stage used (i.e., 1 if non-adult or during reproduction, 2 if adult); all divided by body wt. (0.017 kg, arith. mean of 46 individuals collected at DPG 1995).

^^ Screening-level Values (mg/kg-body wt./day) = extrapolated from the test animal (all body weights were provided in study summaries in Sample et al. 1996; mink = 1kg. EPA 1993) to the deer mouse (0.017 kg, arith. mean of 46 collected individuals from DPG 1995) using the formula NOAEL-w = NOAEL-t x (body wt.-t / body wt.-w)^1/4, where w = wildlife species and t = test species (Eq.4, Sample et al. 1996). The NOAEL-t values from Sample et al. 1996.

~ Hazard Quotient = exposure estimate divided by the screening-level ecotoxicity value.

Bold = metal concentrations above background on the basis of the ANOVA

Table 2.6-6. Preliminary Exposure Estimates and Risk Calculations Based on NOAEL Dose Rates (mg/kgbw/day) for the Horned Lark at SWMU 166, Dugway Proving Ground, Dugway, Utah.

Chemical	Max. Soil Conc. (mg/kg)	Background Soil Conc. (mg/kg)	No. Detects > Bkgd	Exposure Estimate for Soil (mg/kg-BW/day)^	Screening-Level Value Diet (mg/kg-BW/day)^^ NOAEL - w	Hazard Quotient Diet~	Risk Due to Conc. > Bkgd?
ACET	0.045	NA	NA	0.01	NAV	NAv	NA
AL	7400	19,000	0	1787	110	16	No
AS	10	13	0	2	2.6	0.9	No
BA	300	400	0	72	21	3	No
BE	0.74	1	0	0.2	NAV	NAv	No
CA	200,000	190,000	1	48286	NAV	NAv	NAv
CD	3	0.46	1	0.6	1	0.4	Yes
CH2CL2	0.0045	NA	NA	0.001	NAV	NAv	NA
CO	4.4	7.9	0	1	NAV	NAv	NAv
CR	11	17	0	3	1	3	No
CU	14	25	0	3	47	0.07	No
FE	9300	19,000	0	2245	NAV	NAv	NAv
K	2900	7400	0	700	NAV	NAv	NAv
MEC6H5	0.013	NA	NA	0.003	NAV	NAv	NA
MG	42,000	29,000	1	10140	NAV	NAv	NAv
MN	310	520	0	75	977	0.08	No
NA	910	11,000	0	220	NAV	NAv	NAv
NI	9.5	17	0	2	77	0.03	No
PB	36	14	1	9	1	8	Yes
SB	8.6	9.1	0	2	0.125	NAv	No
TL	9.5	35	0	2	NAV	NAv	No
V	20	29	0	5	5.7	0.8	No
XYLEN	0.0083	NA	NA	0.002	NAV	NAv	NA
ZN	110	59	1	27	15	2	Yes

NA = Not Applicable NAv = Not Available NOAEL = no observable adverse effect level

^ Exposure Estimates = product of maximum soil concentration, food intake rate (0.00306 kg/d, where chemical concentration in food is assumed to be the same as soil; food intake rate = [0.398 x (body wt. in grams)^0.850]/1000, Eq. 3-4, EPA 1993), exposure area/period (100%), bioavailability (100%), and life stage used (i.e., 1 if non-adult or during reproduction, 2 if adult); all divided by body wt. (0.017 kg, arith. mean of 46 individuals collected at DPG 1995).

^^ Screening-level Values (mg/kg-body wt./day) = NOAELw = NOAELt (where w = wildlife, t = test animal, Eq. 6, Sample et al. 1996).

All body weights used in risk calculations were borrowed from study summaries in Sample et al. 1996.

~ Hazard Quotient = exposure estimate divided by the screening-level ecotoxicity value.

Bold = metal concentrations above background on the basis of the ANOVA

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Table 2.6-7. Preliminary Exposure Estimates and Risk Calculations Based on NOAEL Dose Rates (mg/kgbw/day) for the Deer Mouse at SWMU 170, Dugway Proving Ground, Dugway, Utah.

Chemical	Max. Soil Conc. (mg/kg)	Background Soil Conc. (mg/kg)	No. Detects > Bkgd	Exposure Estimate for Soil (mg/kg-BW/day)^	Screening-Level Value Diet (mg/kg-BW/day)^^ NOAEL - w	Hazard Quotient Diet~	Risk Due to Conc. > Bkgd?
ACET	0.01	NA	NA	0.002	11	0.0002	NA
AL	9100	19,000	0	1638	2.2	736	[illegible]
AS	8.3	13	0	1.5	0.15	10	
BA	320	400	0	58	2	27	[illegible]
BE	0.68	1	0	0.12	1.4	0.1	No
CA	130,000	190,000	0	23,400	NAv	NAv	NAv
CO	4.2	7.9	0	0.76	NAv	NAv	NAv
CR	27	17	1	4.9	5830	0.001	Yes
CU	18	25	0	3.2	32	0.1	No
FE	12,000	19,000	0	2160	NAv	NAv	NAv
HG*	0.063	0.073	0	0.011	2.8	0.1	No
K	4200	7400	0	756	NAv	NAv	NAv
MEC6H5	0.003	NA	NA	0.0005	30	0.00002	NA
MG	16,000	29,000	0	2880	NAv	NAv	NAv
MN	360	520	0	65	187	0.3	No
NA	12,000	11,000	1	2160	NAv	NAv	NAv
NI	10	17	0	1.8	85	0.02	No
PB	29	14	1	5.2	17	0.3	Yes
V	35	29	1	6.3	0.42	15	Yes
XYLEN	0.0036	NA	NA	0.00065	2.4	0.0003	NA
ZN	75	59	1	[illegible]	341	0.04	Yes

NA = Not Applicable NAv = Not Available NOAEL = no observable adverse effect level

^ Exposure Estimates = product of maximum soil concentration, food intake rate (0.00306 kg/d, where chemical concentration in food is assumed to be the same as soil; food intake rate = [0.621 x (body wt. in grams)^0.564]/1000, Eq. 3-8, EPA 1993), exposure area/period (100%), bioavailability (100%), and life stage used (i.e., 1 if non-adult or during reproduction, 2 if adult); all divided by body wt. (0.017 kg, arith. mean of 46 individuals collected at DPG 1995).

^^ Screening-level Values (mg/kg-body wt./day) = extrapolated from the test animal (all body weights were provided in study summaries in Sample et al. 1996; mink = 1kg, EPA 1993) to the deer mouse (0.017 kg, arith. mean of 46 collected individuals from DPG 1995) using the formula NOAEL-w = NOAEL-t x (body wt.-t / body wt.-w)^1/4, where w = wiildlife species and t = test species (Eq.4, Sample et al. 1996). The NOAEL-t values from Sample et al. 1996.

~ Hazard Quotient = exposure estimate divided by the screening-level ecotoxicity value.

* Because Hg bioaccumulates, the HQ of 0.004 was multiplied by a calculated BAF of 22.5 from mink liver (Wren et al. 1987).

Bold = metal concentrations above background on the basis of the ANOVA

Table 2.6-8. Preliminary Exposure Estimates and Risk Calculations Based on NOAEL Dose Rates (mg/kgbw/day) for the Horned Lark at SWMU 170, Dugway Proving Ground, Dugway, Utah.

Chemical	Max. Soil Conc. (mg/kg)	Background Soil Conc. (mg/kg)	No. Detects > Bkgd	Exposure Estimate for Soil (mg/kg-BW/day)^	Screening-Level Value Diet (mg/kg-BW/day)^^ NOAEL - w	Hazard Quotient Diet~	Risk Due to Conc. > Bkgd?
ACET	0.01	NA	NA	0.002	NAV	NAv	NA
AL	9100	19,000	0	2197	109.7	20	No
AS	8.3	13	0	2.0	2.57	0.8	No
BA	320	400	0	77	21	4	No
BE	0.68	1	0	0.16	NAV	NAv	No
CA	130,000	190,000	0	31,386	NAV	NAv	NAv
CO	4.2	7.9	0	1.01	NAV	NAv	NAv
CR	27	17	1	6.5	1.00	7	Yes
CU	18	25	0	4.3	47	0.09	No
FE	12,000	19,000	0	2897	NAV	NAv	NAv
HG*	0.063	0.073	0	0.015	0.0	53	No
K	4200	7400	0	1014	NAV	NAv	NAv
MEC6H5	0.003	NA	NA	0.0007	NAV	NAv	NA
MG	16,000	29,000	0	3863	NAV	NAv	NAv
MN	360	520	0	87	977	0.1	No
NA	12,000	11,000	1	2897	NAV	NAv	NAv
NI	10	17	0	2.4	77	0.03	No
PB	29	14	1	7.0	1.13	6	Yes
V	35	29	1	8.5	5.70	1	Yes
XYLEN	0.0036	NA	NA	0.00087	NAV	NAv	NA
ZN	75	59	1	18.1	14.50	1	Yes

NA = Not Applicable NAv = Not Available NOAEL = no observable adverse effect level

^ Exposure Estimates = product of maximum soil concentration, food intake rate (0.00306 kg/d, where chemical concentration in food is assumed to be the same as soil; food intake rate = [0.398 x (body wt. in grams)^0.850]/1000, Eq. 3-4, EPA 1993), exposure area/period (100%), bioavailability (100%), and life stage used (i.e., 1 if non-adult or during reproduction, 2 if adult); all divided by body wt. (0.017 kg, arith. mean of 46 individuals collected at DPG 1995).

^^ Screening-level Values (mg/kg-body wt./day) = NOAELw = NOAELt (where w = wildlife, t = test animal, Eq. 6, Sample et al. 1996).

All body weights used in risk calculations were borrowed from study summaries in Sample et al. 1996.

~ Hazard Quotient = exposure estimate divided by the screening-level ecotoxicity value.

* Although it is acknowledged that mercury may bioaccumulate in some ecosystems, an avian BA value greater than 1 was not readily available in the literature.

Bold = metal concentrations above background on the basis of the ANOVA

negligible risk to the target receptor for that chemical; HQs that are greater than 10 but less than 100 are considered to possibly indicate some potential risk. Because none of the calculated HQs for potential total risk to the horned lark exceed 100, and considering site conditions and the natural levels of trace metals in soil at DPG, these potential total risks are moderate or negligible concern to avian receptors at DPG.

The deer mouse and horned lark HQs shown in the SWMU-specific risk computation tables represent potential total risk and thus include the background concentrations of inorganic chemicals as a major contributor to the computed risk levels of the inorganic chemicals. There were no organic chemicals at any SWMUs that produced HQs greater than 1. Generally, the background concentrations of those chemicals that led to a prediction of potential total risk contributed a large proportion of the total potential risk because the background concentrations so nearly approximated the maximum detected concentration. In fact, in many cases the background concentrations exceeded the maximum detected concentration in the most obviously contaminated area of a SWMU. (The HQ value resulting from the subtraction of the background concentration from the maximum detected concentration). When a statistically sound background soil concentration data set is available, as it is for DPG, the incremental risk HQs are a reasonable measures of the level of adverse impacts to biological receptors. Generally, incremental risk did not exceed an HQ of 10, and therefore can be considered negligible. The incremental risk estimates did exceed an HQ of 10 for barium (HQ = 21) at SWMU 20 and for lead (HQ = 14) at SWMU 164; however, the history of use and distribution of sampling results at these two SWMUs suggest that these metals concentrations are naturally occurring.

The predicted HQs should also be evaluated with the available site-specific ecological information. For example, good quality habitat is present at only one of the four Module 2 SWMUs, SWMU 20. For the type and extent of incremental risk predicted for SWMU 20, any remedial actions undertaken will likely be more detrimental to vegetation and wildlife than the potential impacts from leaving the suspected contamination, if any, in place. The remaining SWMUs (164, 166, and 170) are located in heavily disturbed areas of DPG associated with high human activity. Good quality animal habitat is absent at these SWMUs; thus, it is unlikely that SWMUs 164, 166, and 170 are utilized by DPG wildlife. A discussion of these site conditions

is presented with each SWMU evaluation (Sections 3.0 – 6.0). A conservative estimate of potential total risk, an evaluation of incremental risk, and consideration of site conditions support a recommendation of no corrective action at the Module 2 SWMUs.

For a preliminary screening-level risk assessment, it is customary (EPA 1994) to choose several ecological receptors that are part of significant complete exposure pathways representative of a taxonomic category, and that are fairly ubiquitous. Although several species that are were formerly listed or recommended for either state or federal protection (spotted bat, kit fox, and Skull Valley pocket gopher) may be present at DPG, only the kit fox has been documented to occur at DPG and none of these species have been sighted in the vicinity of the Module 2 SWMUs. Therefore, they were not considered to be at risk at the Module 2 SWMUs. For example, pocket gophers may be present at various locations at DPG; however, they are not likely to be present at SWMUs 164, 166, or and 170 because these sites are highly disturbed and covered with gravel and asphalt. The listed two predator species (i.e., kit fox and spotted bat) are unlikely to be at risk at these SWMUs based on the large foraging ranges of these two species when compared to the very small area of these SWMUs and, with the exception of DDT at SWMU 170, the COCs are not likely to biomagnify in the food chain. The suspected contamination, if any, at SWMU 20 is not considered sufficient to cause risk to any wildlife species.

3.0 CLOSURE PLAN FOR SWMU 20—CAMELS BACK RIDGE LANDFILL

3.1 UNIT CHARACTERISTICS

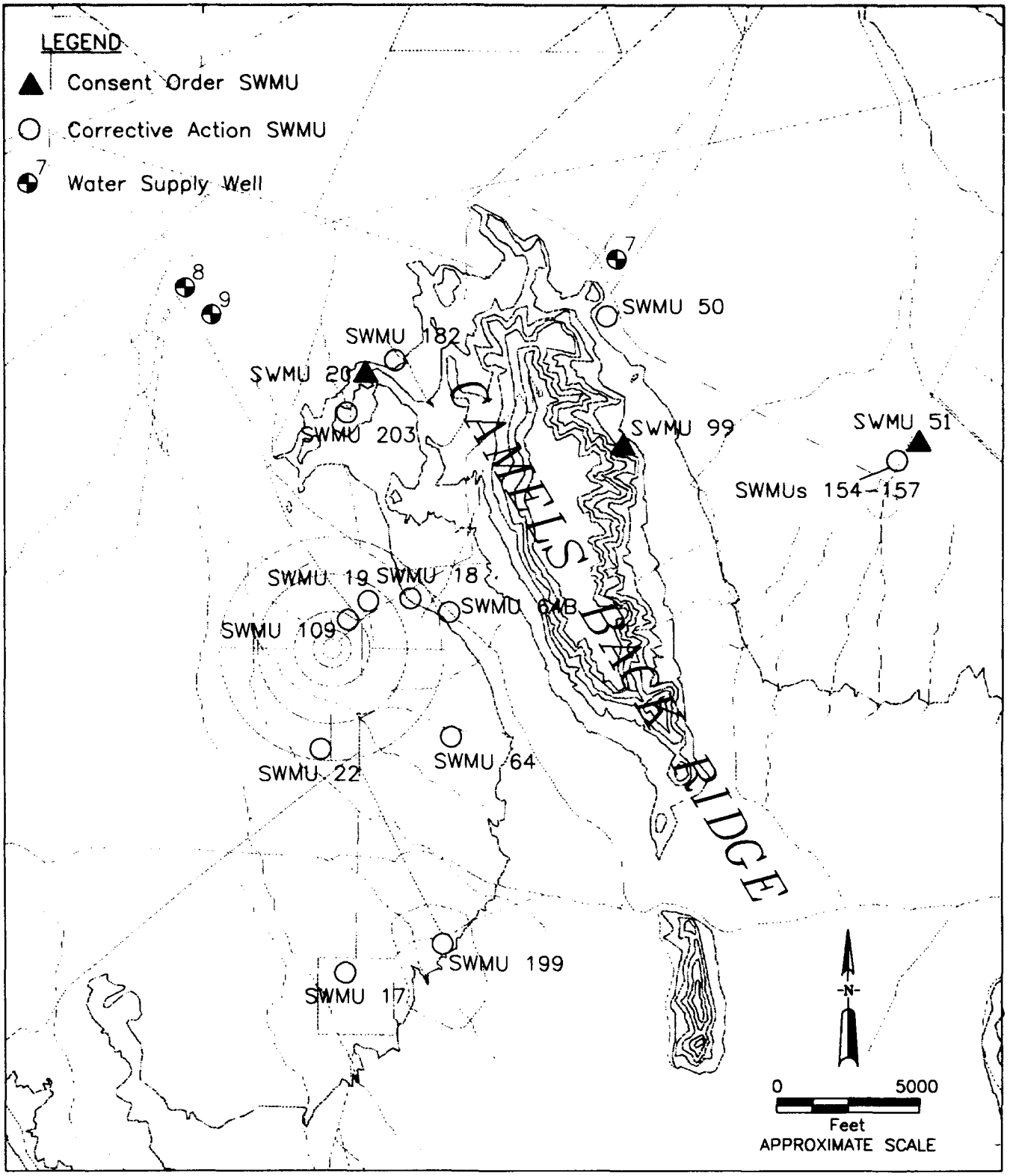
3.1.1 SWMU Description and History

SWMU 20, known as the Camels Back Ridge Landfill, is located on the northwest side of Camels Back Ridge, the prominent topographic feature southwest of Carr Facility (Figure 3.1-1). Camels Back Ridge is located on the divide between Dugway Valley and Government Creek Valley. SWMU 20 lies near the base of the steep, northwestern escarpment of Camels Back Ridge, at an approximate elevation of 4,350 ft msl on ground that slopes westward into the Dugway Valley (EPIC 1986).

SWMU 20 includes two areas of possible disposal activities, referred to as the west and east areas. These areas are identified as SWMUs 20-1 and 20-2, respectively (Figure 3.1-2). SWMU 20-1 is located approximately 120 ft southwest of the Open Detonation Unit (Corrective Action SWMU 182), also known as the Explosive Test Shield Facility, a building constructed of steel beams and thick steel plating that was used for explosives testing (Figure 3.1-3). A road lies between SWMU 20-1 and the Explosive Test Shield Facility. SWMU 20-1 is also located 400 ft south of the Suppressive Shield Facility, a large sheet-metal building at which tests for the suppression of chemical agent dispersion were conducted using simulants such as bis (2-ethylhexyl) hydrogen phosphite (BIS) and polyethylene glycol (Keetch 1995c). SWMU 20-2 is located approximately 1,300 ft southeast of the Explosive Test Shield Facility (Figure 3.1-2).

SWMU 20-1 (west area) covers an approximate area of 480 ft by 100 ft (Figure 3.1-3). Surface features in the west area consist of two low ridges of soil and two shallow depressions (EBASCO 1991). The ridges are 15 ft to 80 ft apart, converging west to east toward the Explosive Test Facility (Figure 3.1-4). They are 300 ft long, 3 ft wide, and 1 ft to 1.5 ft high. The shallow depressions run along the entire length of the ridges. They are 1 ft deep and are currently revegetating. A hummocky surface and a small earthen mound (approximately 3 ft high) are also found at the western end of the ridges, near a small rock outcrop. A shallow channel approximately 2 ft wide and 1 ft deep is located southeast of the ridges and depressions.

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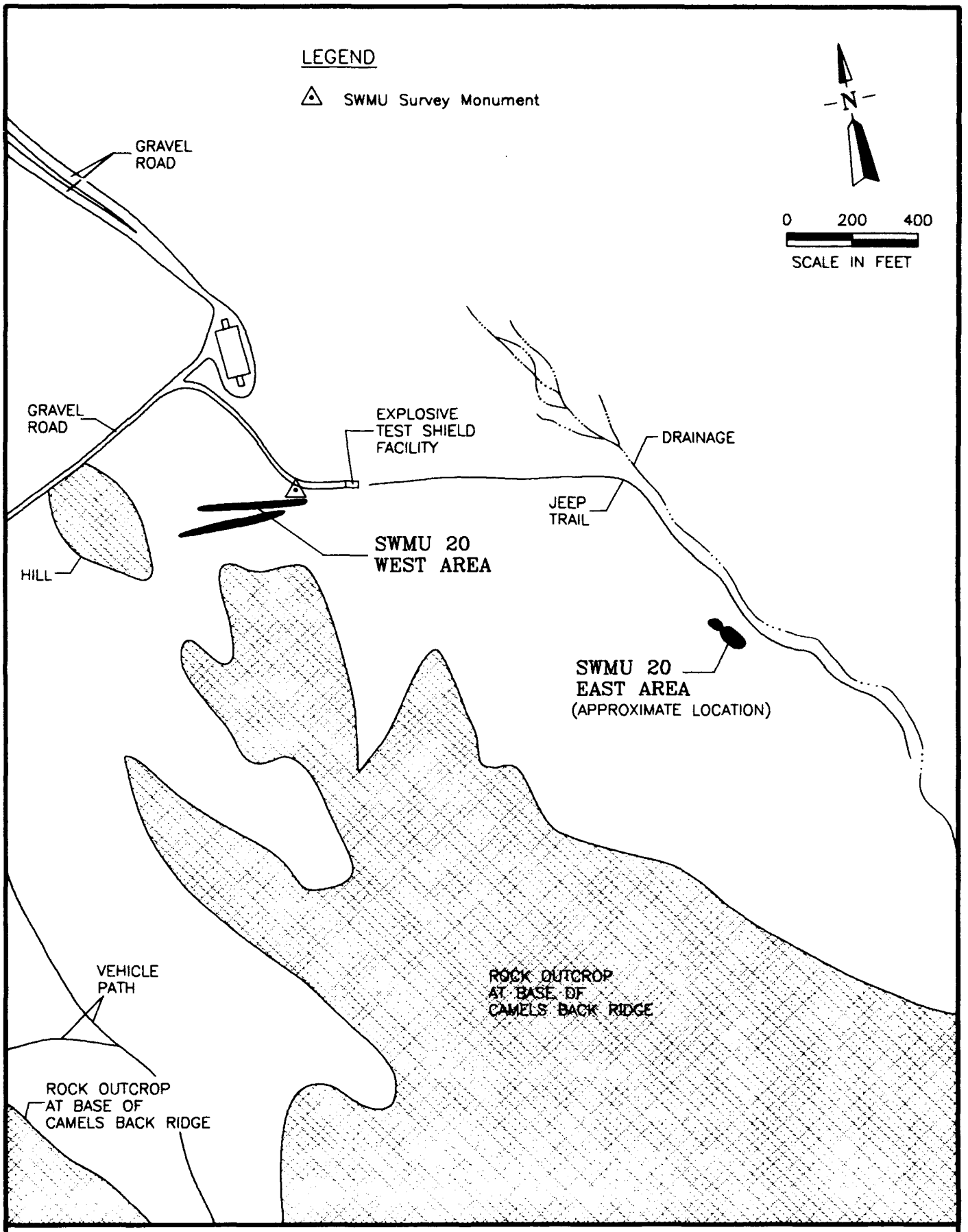


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Figure 3.1-1 SWMU 20 Location Map

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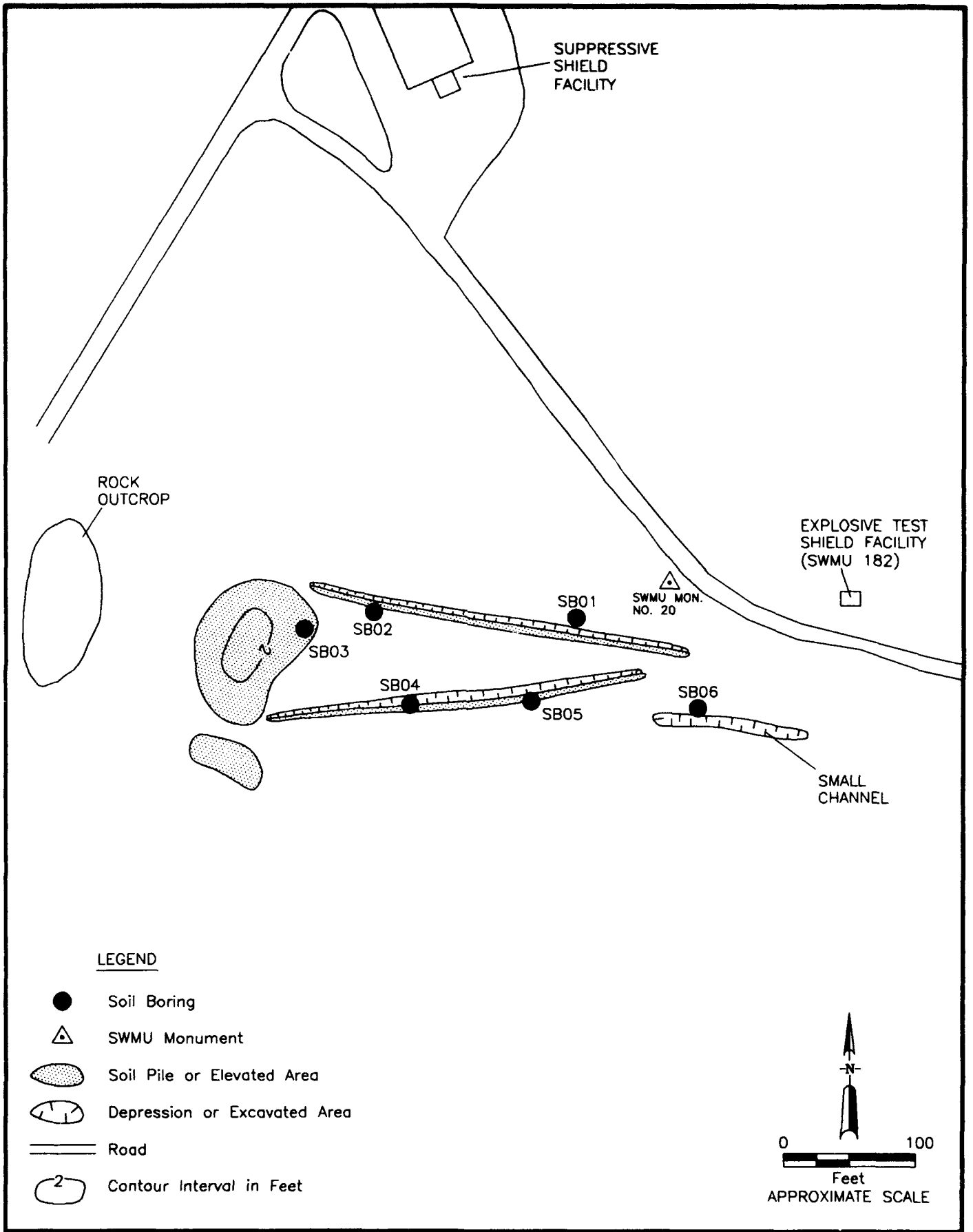


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Figure 3.1-2 SWMU 20 Detailed Location Map (Camels Back Ridge)

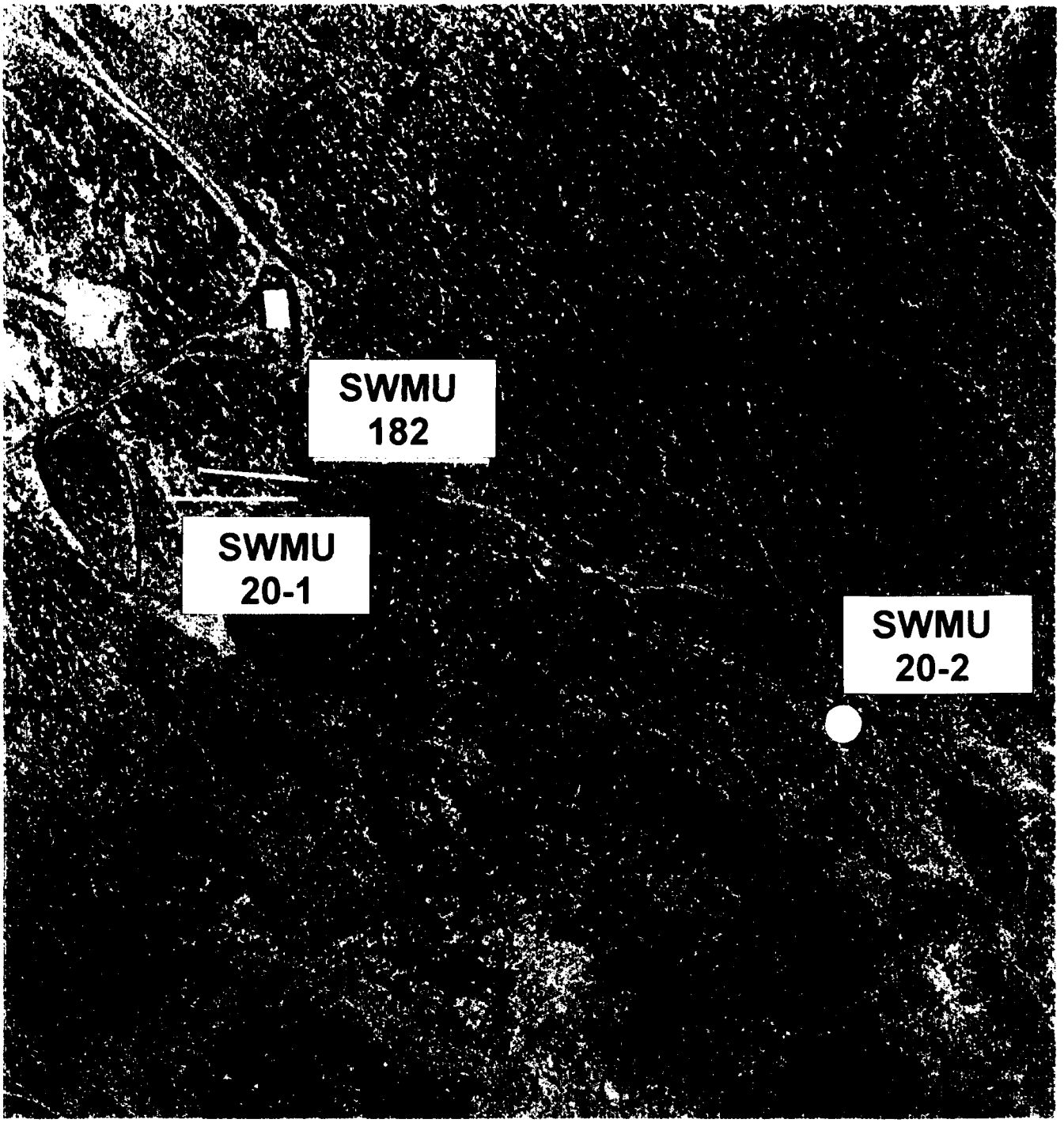


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**Figure 3.1-3 SWMU 20-1 (West Area)
Sample Location Map**

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Figure 3.1-4 SWMU 20
Aerial Photograph of SWMU 20 Area

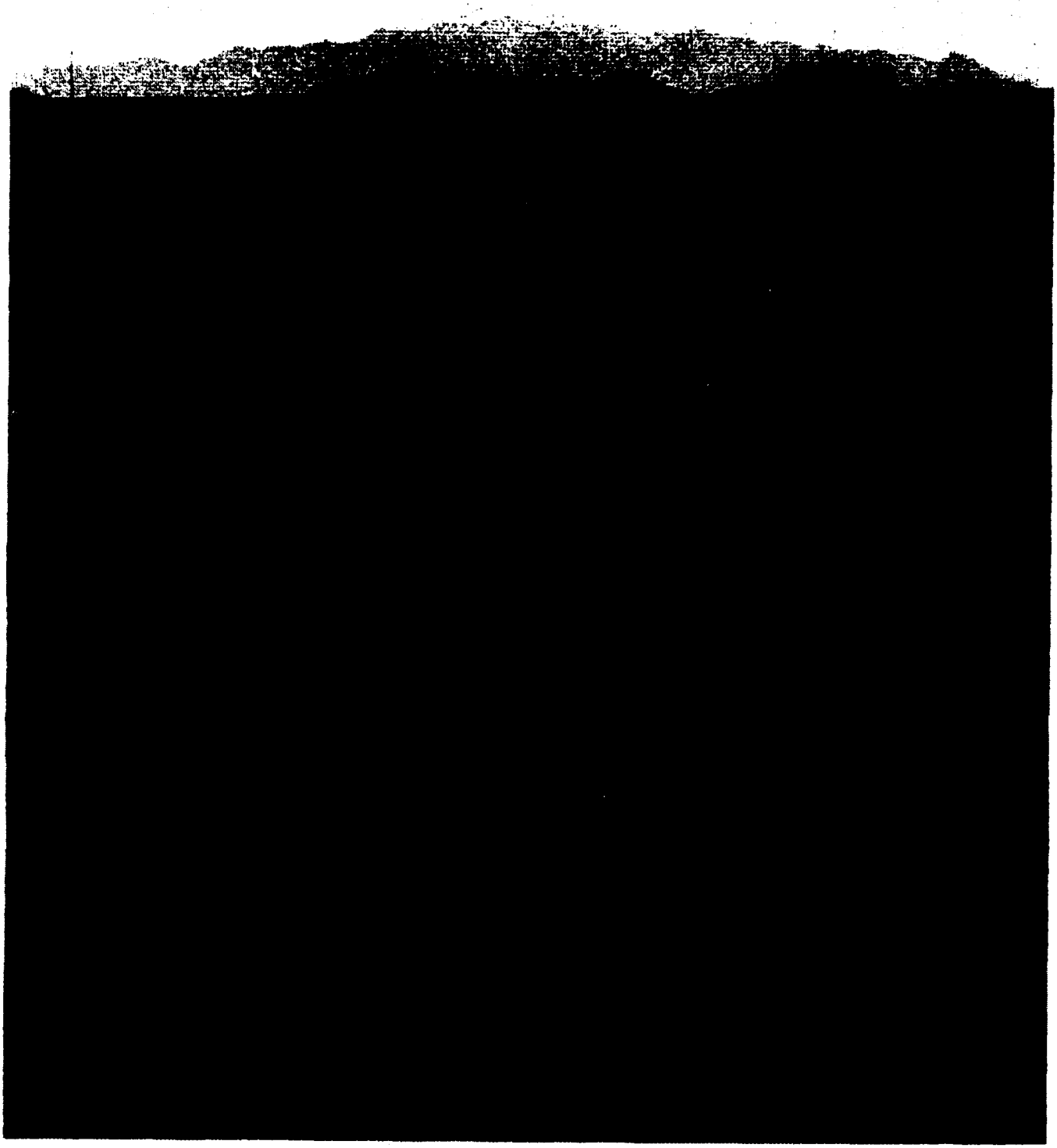
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Each depression was originally thought to be a partially backfilled disposal trench. According to the Installation Assessment Update (USATHAMA 1988b), miscellaneous refuse was disposed of in the unit between 1970 and 1980. An Army Corps of Engineers report (Llopis and Zawila 1992) states that the depressions at SWMU 20-1 were used to divert runoff from the Explosive Test Shield Facility. However, former DPG employees currently working as contractors state that the trenches were used to hold instrumentation cables that were used to measure test parameters at the Explosive Test Shield Facility. The instrumentation cables, which were contained in plastic conduits, ran from a control panel on the south side of the road at the Explosive Test Shield Facility to the project command post that was located behind the small rock outcrop west of the trenches (Keetch and Mattinson 1995) (Figure 3.1-5). The cables were removed from the trenches in the mid-1970s (Keetch 1995b).

The absence of buried wastes is supported by a previous geophysical investigation. A nonintrusive geophysical survey using magnetometer and electromagnetic induction instrumentation was conducted at SWMU 20-1 by the Army Corps of Engineers Geotechnical Laboratory. The survey did not indicate any major anomalies (Llopis and Zawaila 1992).

SWMU 20-2 consists of a 2-ft-deep excavation adjacent to a 2-ft-high earthen mound covering a combined area measuring approximately 100 ft by 25 ft (Figure 3.1-6). The area is located near a jeep trail that parallels a large drainage ditch southeast of the Explosive Test Shield Facility (Figure 3.1-7) (EBASCO 1993). According to James Keetch, Sr., a former DPG employee, soil was excavated from this pit to evaluate its use as a borrow soil. However, there was insufficient gravel content in the soil to justify further development (Keetch 1995a).

In conclusion, current information indicates that neither SWMU 20-1 nor 20-2 was used to manage hazardous or solid waste. SWMU 20-1 was used in the operation of the Explosive Test Shield Facility (SWMU 182), and SWMU 20-2 was an exploratory pit for borrow gravel. There are no plans to use either SWMU 20-1 or 20-2 in the future.



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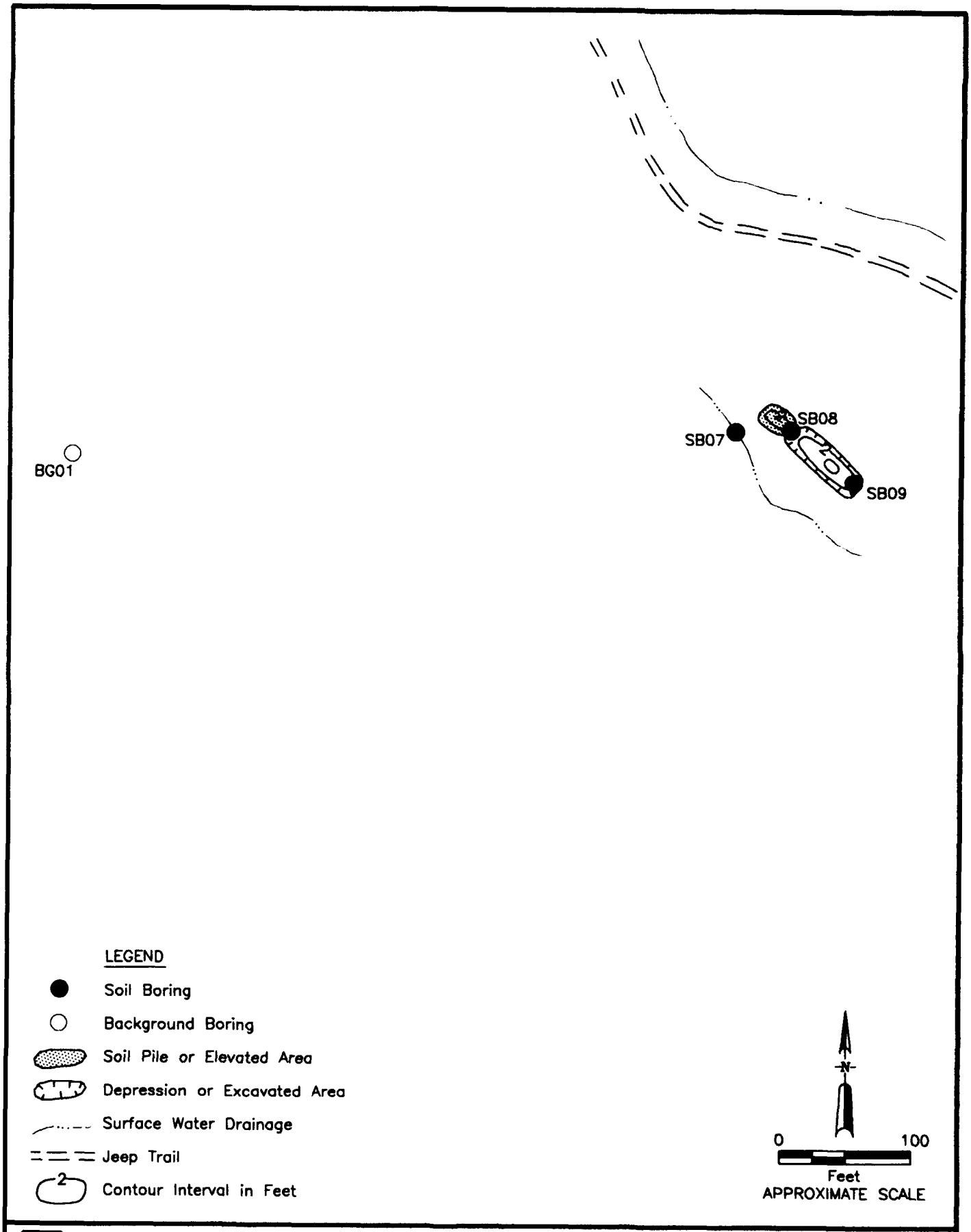
Figure 3.1-5 SWMU 20-1
Camels Back Ridge Landfill, West Area
(Looking West)



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Figure 3-16 SWMU 20.2
Camels Back Ridge Landfill - East Area
(Looking Southeast)

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Figure 3.1-7 SWMU 20-2 (East Area) Sample Location Map

3.1.2 Surface Water and Groundwater

SWMU 20 receives runoff from Camels Back Ridge, with most precipitation infiltrating the soil. Any runoff from SWMU 20 would continue to flow westward toward Dugway Valley.

Because there are no groundwater monitoring wells at SWMU 20, hydrogeologic conditions at the SWMU are inferred from data from nearby water supply wells 7, 8, and 9. Of these, only well 7, drilled on the eastern and opposite side of Camels Back Ridge, yielded fresh water. In this well, fresh water was encountered in a sand unit lying at a depth between 64 ft and 105 ft and in the upper portion of the bedrock at a depth between 105 ft and 120 ft. Wells 8 and 9, located several hundred feet west of SWMU 20, yielded nonpotable brackish water. In well 8, only saline groundwater was reported at a depth of 230 ft; in well 9, brackish water occurred at or near the bedrock-overburden interface at a depth of 35 ft (EBASCO 1993).

Within SWMU 20, shallow bedrock is expected to be overlain by relatively coarse-grained alluvial material, and at shallower depths, by finer-grained lacustrine deposits. Groundwater beneath the unit, if present, would likely be in the alluvial materials and upper portion of the bedrock. Groundwater beneath SWMU 20 probably flows north to northwest into the basin.

3.1.3 Maximum Extent of Operations

Neither hazardous waste nor other solid waste has been handled or disposed of at SWMU 20. Therefore, a survey plat of SWMU 20 is not required.

3.1.4 Nature and Extent of Contamination

Surficial and subsurface soil samples were collected from 10 soil borings, including 1 background location, to characterize the nature and extent of soil constituents at SWMU 20. Six shallow borings (SB01 through SB06) were drilled in native soil consisting of clayey silt at SWMU 20-1, and three borings (SB07 through SB09) were drilled in native soil consisting of silty sand with gravel at SWMU 20-2, consistent with colluvium in the area. Fill material was not present at

sample locations. The samples were analyzed for total metals, VOCs, SVOCs, explosives, agent breakdown products, and cyanide.

Data validation resulted in a small number of rejected data. Some detections of selenium, and a single detection of both isopropylmethyl phosphonic acid (IMPA), and cyclonite (RDX) were rejected (R-qualified); and some detections of acetone and methylene chloride were rejected because of contamination in the field of laboratory blank (B-qualified). Tables 3.1-1 and 3.1-2 summarize the constituents detected in SWMU 20 surficial and subsurface soil samples, which are discussed below. Further discussion of the sampling results can be found in Section 7.2.1 of the final Interim Report (EBASCO 1995a).

SWMU 20-1 (West Area)

Antimony, arsenic, barium, calcium, and sodium were detected at concentrations above background in five of six borings at SWMU 20-1 (Figure 3.1-8). The results of a nonparametric ANOVA of the soil samples showed that the distribution of only three of these metals (antimony, barium, and calcium) are significantly different from background at SWMU 20-1. Only the subsurface barium detection (1,500 µg/g) at a depth of 3 ft at SB06 was significantly above the background UTL (400 µg/g), and it was still well below the RBSL of 5,200 µg/g. The slightly elevated calcium concentrations at SWMU 20-1 may be due to erosion of the carbonate rocks that comprise Camels Back Ridge. These inorganic constituents will persist in the soil, generally adsorbing to the soil particles.

A single low-level detection (2.6 µg/g) of IMPA occurred in a subsurface sample (SB04, 3-ft to 4-ft interval) from the ridge adjacent to the southern depression. However, the sample was rejected because it exceeded the holding time. No other organic compounds were detected in the six borings at SWMU 20-1.

Table 3.1-1 Summary of Constituents Detected in SWMU 20 Surficial Soil*

Analyte	Percent Detections	Total Samples	Total Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Background Value (µg/g)	Detections> Background Value
AL	100	9	9	2,700	7,800	020SB01	19,000	0
AS	100	9	9	2.7	17	020SB09	13	3
BA	100	9	9	170	650	020SB03	400	4
BE	11	9	1	0.66	0.66	020SB06	1.0	0
CA	100	9	9	75,000	210,000	020SB01	190,000	1
CO	67	9	6	2.1	3.6	020SB01	7.9	0
CR	100	9	9	2.5	7.1	020SB06	17	0
CU	100	9	9	3.4	11	020SB01	25	0
FE	100	9	9	2,600	7,800	020SB06	19,000	0
K	100	9	9	880	3,900	020SB01	7,400	0
MEC6H5	100	2	2	0.50	0.51	020SB08	NA	NA
MG	100	9	9	14,000	31,000	020SB07	29,000	1
MN	100	9	9	150	260	020SB02	520	0
NA	100	9	9	500	7,600	020SB06	11,000	0
NI	100	9	9	2.2	7.7	020SB02	17	0
PB	100	9	9	4.1	6.8	020SB03	14	0
SB	67	9	6	5.8	11	020SB01	9.1	1
TL	22	9	2	17	20	020SB09	35	0
V	100	9	9	5.6	22	020SB06	29	0
ZN	100	9	9	10	26	020SB01	59	0

µg/g Micrograms per gram.
NA Not applicable. Background values cannot be determined for these analytes.
* Surficial soil samples collected from ground surface to depth of 1 ft in soil borings, and from ground surface to depth of 6 inches (0.5 ft) at surface soil (SS) locations. Analyte acronyms are defined in the Acronym List.

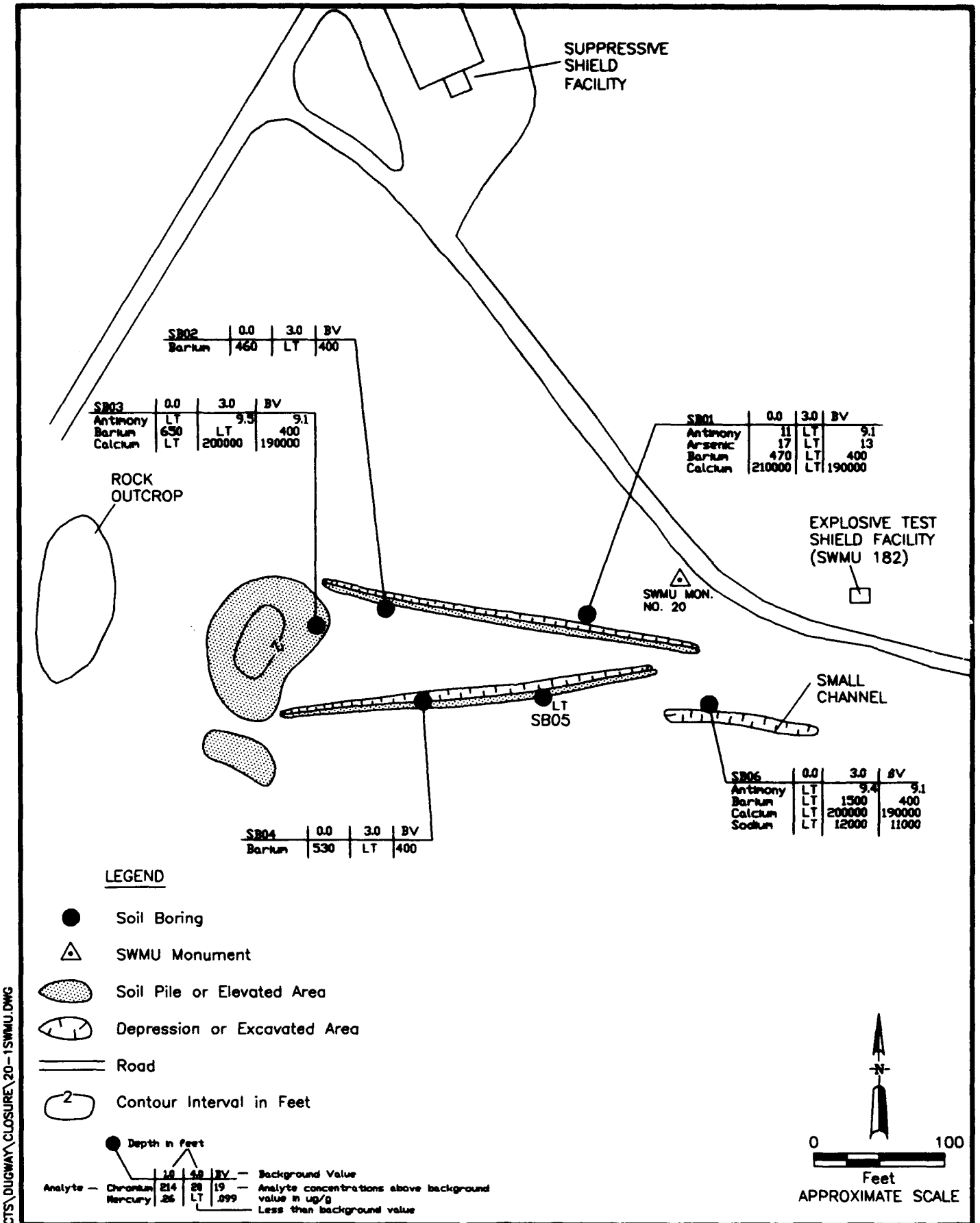
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Table 3.1-2 Summary of Constituents Detected in SWMU 20 Subsurface Soil*

Analyte	Percent Detections	Total Samples	Total Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Background Value (µg/g)	Detections> Background Value
AL	100	10	10	2,900	8,100	020SB04	19,000	0
AS	100	10	10	4.4	24	020SB09	13	2
BA	100	10	10	150	1,500	020SB06	400	1
BE	33	10	3	0.69	0.93	020SB01	1.0	0
CA	100	10	10	100,000	240,000	020SB09	190,000	3
CH2CL2	30	6	2	0.0054	0.025	020SB09	NA	NA
CO	60	10	6	2.6	4.9	020SB02	7.9	0
CR	100	10	10	3.1	9.7	020SB05	17	0
CU	100	10	10	3.1	10	020SB02	25	0
FE	100	10	10	3,200	9,400	020SB02	19,000	0
K	100	10	10	1,000	3,300	020SB01	7,400	0
MEC6H5	10	10	1	0.0087	0.0087	020BG01	NA	NA
MG	100	10	10	13,000	32,000	020SB07	29,000	1
MN	100	10	10	160	350	020SB02	520	0
NA	100	10	10	530	12,000	020SB06	11,000	1
NI	100	10	10	2.6	8.5	020SB04	17	0
PB	100	10	10	3.6	10	020SB01	14	0
SB	50	10	5	6.5	9.8	020SB07	9.1	3
TL	30	10	3	16	24	020SB09	35	0
V	100	10	10	6.4	24	020SB05	29	0
ZN	100	10	10	10	28	020SB02	59	0

µg/g Micrograms per gram.
NA Not applicable. Background values cannot be determined for these analytes.
* Subsurface soil samples collected from depths of 3 ft to 4 ft except in 20SB07, where samples collected from depths of 2 ft to 3 ft.
Analyte acronyms are defined in the Acronym List.

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Figure 3.1-6 SWMU 20-1 (West Area) Distribution of Inorganics in Soil

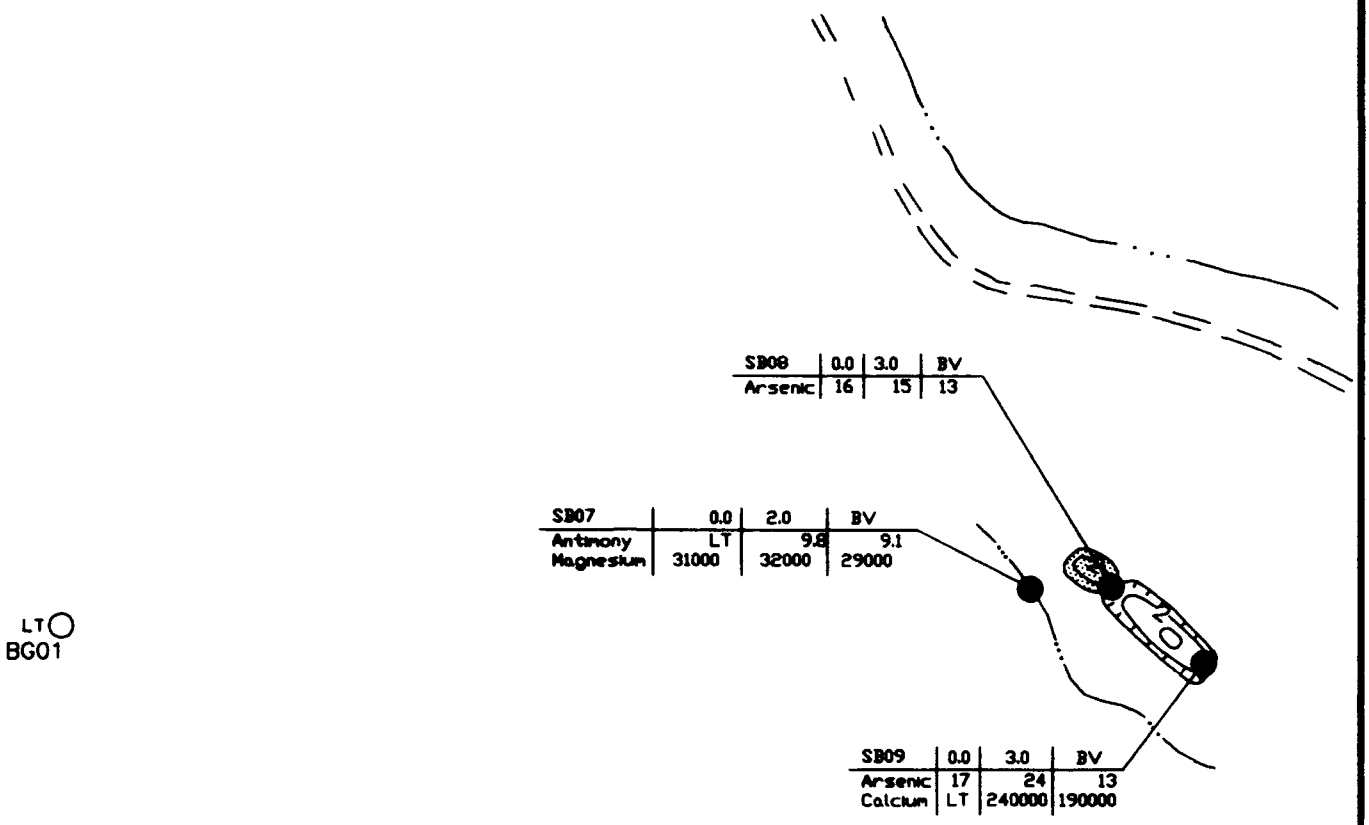
SWMU 20-2 (East Area)

Four inorganic constituents—antimony, arsenic, calcium, and magnesium—were detected at concentrations slightly above background in surficial and subsurface soils at SWMU 20-2 (Figure 3.1-9). Arsenic was also detected at concentrations above the RBSL (3.5 μg/g) since the RBSL for this metal is below its background UTL (13 μg/g). ANOVA results showed that the distributions of only three of these metals (antimony, calcium, and magnesium) are significantly different from background at SWMU 20-2 (Table 2.3-2). The elevated concentrations of calcium and magnesium at SWMU 20-2 may be due to erosion of the carbonate rocks that comprise Camels Back Ridge.

Only two organic compounds, methylene chloride and toluene, were detected in low concentrations in soil samples at SWMU 20-2 (Figure 3.1-10). Methylene chloride and toluene were also detected in the subsurface at the background location at SMWU 20. These organic compounds are not associated with the operations at SWMU 20-2 and were detected at low parts per billion (ppb) concentrations. No explosives or agent breakdown products were detected at SWMU 20-2. The organic constituents detected at SWMU 20-2 will not persist in the soil due to aerobic biodegradation and short degradation half-lives. Additional information on contaminant fate and transport is found in Appendix M, Table M.1-1 of the final Interim Report (EBASCO 1995a).

3.1.5 Maximum Waste Inventory

According to the most current information, the depressions at SWMU 20-1 were formerly shallow trenches used to convey instrumentation cables for the Explosive Test Shield Facility; this cable was later removed. The disturbed soil in SWMU 20-2 resulted from soil excavation to evaluate potential borrow soil. Therefore, there is no potential for hazardous wastes, including residues, at SWMU 20 because the site is not known to have been used to treat, store, or dispose of hazardous wastes and because there is no plan to handle hazardous waste at the site in the future.



LEGEND

- Soil Boring
- Background Boring
- Soil Pile or Elevated Area
- Depression or Excavated Area
- Surface Water Drainage
- Jeep Trail
- Contour Interval in Feet

Depth in Feet

Analyte		1.0	4.0	BV	
	Chromium	214	20	19	Background Value
	Mercury	.26	LT	.099	Analyte concentrations above background value in ug/g
					Less than background value

N

0 100

Feet

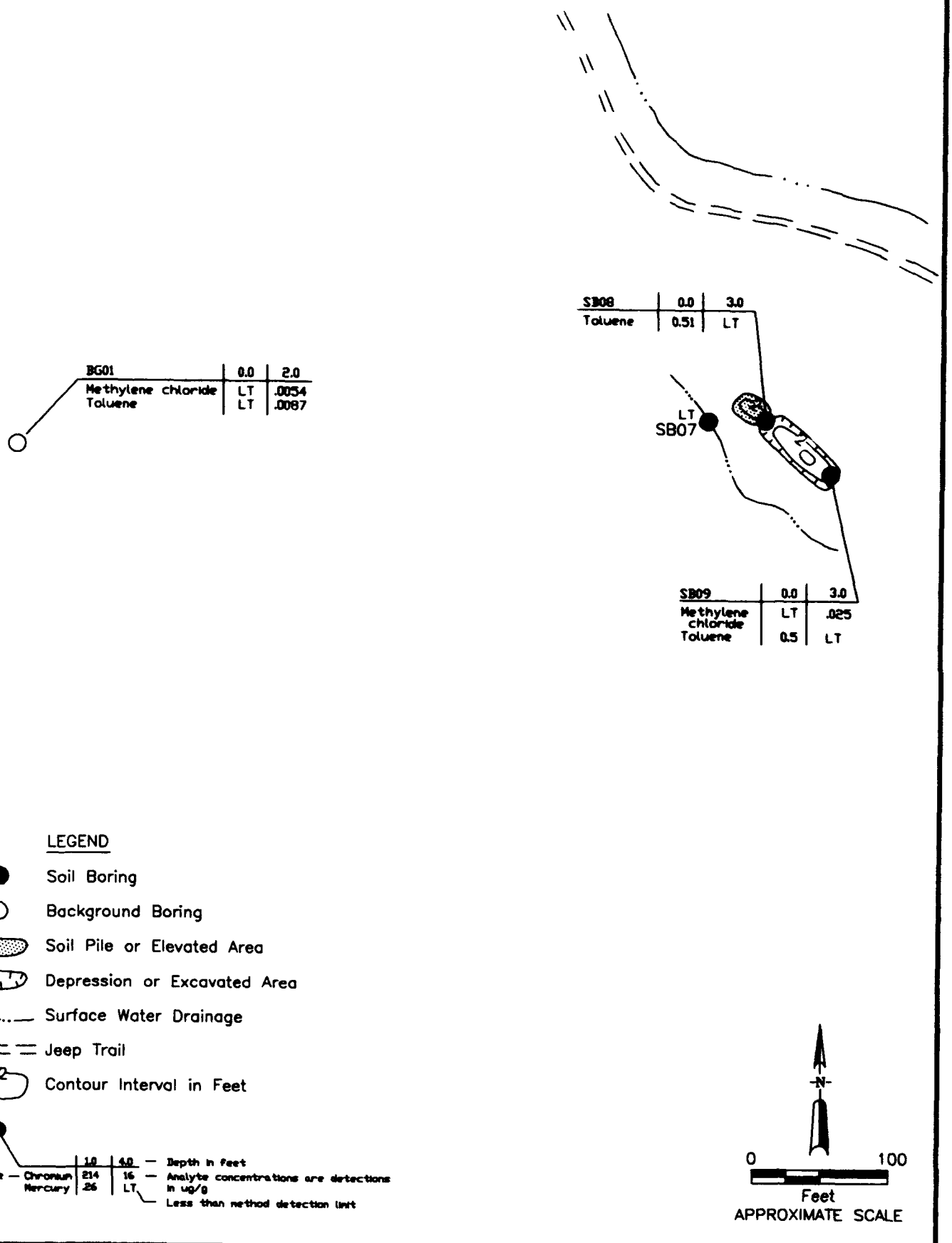
APPROXIMATE SCALE

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Figure 3.1-9 SWMU 20-2 (East Area) Distribution of Inorganics in Soil



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Figure 3.1-10 SWMU 20-2 (East Area) Distribution of Organics in Soil

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Surface and subsurface soil samples were collected from SWMUs 20-1 and 20-2 where disposal activities were once suspected. The samples were analyzed for metals, VOCs, SVOCs, explosives, agent breakdown products, and cyanide. As summarized in Section 3.1.4, there is no contaminated soil or residue related to operations at SWMU 20.

3.1.6 Baseline Human Health Risk Assessment

The baseline human health risk assessment for SWMU 20 was based on the soil analytical results presented in Section 3.1.4 and was developed using the risk assessment approach described in Section 2.5. It presents a conservative evaluation of potential health risks associated with a hypothetical residential use of SWMU 20.

3.1.6.1 Identification of COPCs

Using the conservative selection criteria defined in Section 2.5 and Figure 2.5-2 the following two constituents were selected as soil COPCs at SWMU 20 (Table 3.1-3):

- Methylene chloride
- Toluene

Toxicity profiles for these COPCs are presented in Appendix B.3.

As discussed in the preceding sections, the results of soil sampling do not indicate any significant contamination resulting from previous activities at the site. Concentrations of inorganic constituents are generally comparable to background and thus likely reflect natural variability. The ANOVA comparison identified four inorganic constituents that had concentrations greater than background. Calcium and magnesium were eliminated from consideration for COPCs because they are considered essential nutrients. Barium and antimony were also eliminated from further evaluation in the risk assessment because there is no evidence of historical use at SWMU 20. Four out of 19 barium detections slightly exceeded the UTL (400 μg/g), while the residential soil RBSL for barium (5,400 μg/g) was not exceeded in any samples. Four antimony detections slightly exceeded the UTL of 9.1 μg/g (less than 1.2 times background), while the residential soil

Table 3.1-3 Summary of Constituents Detected in SWMU 20 Soil Samples and Identification of COPCs

Analyte	Detection Frequency No. of Hits	Detection Frequency Percent	Maximum (µg/g)	EPC 95% UCL (µg/g)	UTL (µg/g)	ANOVA COPC[1]	Essential Nutrient?	Few Detections > UTL	EPC Less Than RBSL	Evidence of Historical Use	Selected as COPC?[2]
Aluminum	19/19	100%	8,100	6,400	19,000	No	No	--	--	--	No
Antimony	11/19	58%	11	7.2	9.1	Yes	No	Yes	Yes	No	No
Arsenic	19/19	100%	24	12	13	No	No	--	--	--	No
Barium	**19/19**	**100%**	**1,500**	**480**	**400**	**Yes**	**No**	**Yes**	**Yes**	**No**	**No**
Beryllium	4/18	21%	0.93	0.45	1.0	No	No	--	--	--	No
Calcium	19/19	100%	240,000	180,000	190,000	Yes	Yes	--	--	--	No
Chromium	19/19	100%	9.7	6.6	17	No	No	--	--	--	No
Cobalt	12/19	63%	4.9	2.9	7.9	No	No	--	--	--	No
Copper	19/19	100%	11	8.1	25	No	Yes	--	--	--	No
Iron	19/19	100%	9,400	7,000	19,000	No	Yes	--	--	--	No
Lead	19/19	100%	10	6.3	14	No	No	--	--	--	No
Magnesium	19/19	100%	32,000	24,000	29,000	Yes	Yes	--	--	--	No
Manganese	19/19	100%	350	250	520	No	Yes	--	--	--	No
Methylene chloride	**2/6**	**30%**	**0.025**	**0.015**	--	--	**No**	--	--	--	**Yes**
Nickel	19/19	100%	8.5	6.5	17	No	No	--	--	--	No
Potassium	19/19	100%	3,900	2,800	7,400	No	Yes	--	--	--	No
Sodium	19/19	100%	12,000	6,300	11,000	No	Yes	--	--	--	No
Thallium	5/19	26%	24	11	35	No	No	--	--	--	No
Toluene	**3/12**	**25%**	**0.51**	**0.19**	--	--	**No**	--	--	--	**Yes**
Vanadium	19/19	100%	24	15	29	No	No	--	--	--	No
Zinc	19/19	100%	28	22	59	No	Yes	--	--	--	No

[1] SWMU 20 soil analytical results were compared with site-specific background data using the Wilcoxon rank-sum analysis of variance (ANOVA); this approach is documented in Section 2.3 and Appendix A.

[2] Soil COPCs are highlighted above (listed in bold) and were identified using the selection criteria shown in Figure 2.5-2.

RBSL for antimony (31 μg/g) was not exceeded in any samples. The two organic compounds detected in SWMU 20 soil samples—methylene chloride and toluene—occur at low concentrations and are also common laboratory contaminants. Consequently, their presence is probably not site related. These observations are not unexpected as neither hazardous waste nor solid waste is known to have been handled or disposed of at SWMU 20.

One additional organic constituent, IMPA, was detected at SWMU 20-1. This detection was rejected because after extraction, the analysis missed by 15 days the prescribed holding time of 30 days. Consequently, IMPA was not identified as a COPC and the datum could not be used in the risk calculations. If the data were valid, the HQ for the detected IMPA concentration of 2.6 μg/g would be 0.00055. Therefore, the HQ for this single IMPA detection would have no significant effect on the SWMU 20 HI of 0.33, which is well below the target HI criterion of 1.0. Consequently, human health risks associated with potential exposure to the single rejected detection of IMPA at SWMU 20 are expected to be negligible.

As indicated in Section 3.1.4, its presence is not likely to be attributable to SWMU 20 activities, but rather to contamination or releases from other source areas. At corrective action SWMU 203, Camels Back Cave, IMPA was detected in one of five surface soil samples at 75 μg/g. SWMU 203 is 2,000 ft to 3,000 ft away from SWMU 20. It was used to test agent munitions on cave fortifications.

3.1.6.2 Analysis

As required by UAC R315-101 and as described in Section 2.5, the risk assessments for the Module 2 SWMUs evaluated two land-use scenarios. First, RBSLs were derived for a hypothetical residential land-use scenario using the current reasonable maximum exposure (RME) and toxicological parameters required by UAC R315-101 and recommended by EPA guidance (Table 2.5-1) and these RBSLs were used to calculate risks associated with site-specific COPC concentrations. This scenario assumes unrestricted land use and represents the most conservative analysis of potential soil exposures for the Module 2 SWMUs. Consequently, if

cancer risks and HIs calculated using the residential land-use RBSLs are below target risk criteria (e.g., a cancer risk of 10^{-6} and an HI of 1.0), then the Utah rules allow risk-based closure of the unit, and further risk evaluation is not warranted (Figure 2.5-1).

SWMU 20 is located in a remote area of DPG, between Dugway Valley and Government Creek Valley near the base of Camels Back Ridge. Under current site conditions, the most likely exposure scenario would involve occasional visits by a site maintenance worker or personnel involved in testing at the Explosive Test Shield Facility. The latter scenario also applies to potential future uses of SWMU 20. Residential use of this area is highly unlikely, as is a commercial or industrial setting involving chronic (long-term) exposures. Consequently, the residential land-use analysis used in the SWMU 20 baseline risk assessment is considered very conservative.

3.1.6.3 Risk Characterization

Table 3.1-4 summarizes the results of the risk assessment developed for future exposures to SWMU 20 surface and subsurface soil under a hypothetical residential land-use scenario. Cancer risks and noncancer HIs presented in this table were calculated using the RBSLs listed in Table 2.5-2 in accordance with the methods, equations, and assumptions outlined in Section 2.4 and Appendix B.1. The total cancer risk and HI calculated for the conservative SWMU 20 risk evaluation are 2.5×10^{-9} and 2.5×10^{-5}, respectively. Both values are well within the state of Utah criteria for risk-based closure (cancer risk of 10^{-6} and HI of 1.0).

3.1.6.4 Summary

The results of the human health risk assessment indicate that the risks posed to potential human receptors at SWMU 20 are negligible for hypothetical residential land uses. Risks associated with current site conditions (corresponding to minimal human exposure) or a potential future industrial-use scenario would be lower than those estimated for residential uses (Table 3.1-4), and therefore were not calculated. Based on UAC R315-101 and the decision framework illustrated in Figure 2.5-1, these results qualify the SWMU for risk-based closure.

Table 3.1-4 Results of SWMU 20 Cancer Risk and Hazard Index Calculations for the Hypothetical Residential Land-Use Scenario

Chemical of Potential Concern	Carcinogenic RBSL (µg/g)	EPC (µg/g)	Associated Cancer Risk[1]	Noncarcinogenic RBSL (µg/g)	EPC (µg/g)	Associated HQ[2]
Methylene Chloride	6	0.015	2.5E-09	7	0.015	2.0E-05
Toluene	--	0.19	--	490	0.19	5.3E-06
	Total Cancer Risk:		**2.5E-09**	**Total Hazard Index (HI):**		**2.5E-05**

[1] Cancer risks were calculated using the following equation: Risk = (EPC/RBSL)*TCR, where TCR equals a target cancer risk level of 10^{-6} (or 0.000001). Table 2.5-1 summarizes the underlying exposure assumptions; Appendix B.1 documents the RBSL equations and calculations.

[2] Chemical-specific HQs were calculated by dividing the EPC by the noncarcinogenic RBSL (Section 2.5.5, Appendix B.1).

Analyte acronyms are defined in the Acronym List.

3.1.7 Ecological Risk Assessment

3.1.7.1 Ecological Conditions

SWMU 20-1 is located in the middle of a well-established greasewood community that extends for some distance in all directions. SWMU 20-2 is topographically higher than most of the Consent Order units and, as a result, is located in a sagebrush community that is typical of the mountain flanks at DPG. This sagebrush community is particularly diverse, with rabbitbrush, shadscale saltbush, greasewood, cheatgrass, and kochia dispersed throughout the area. The greasewood and sagebrush communities at DPG are utilized by many wildlife species. A list of observed species and the habitats they were found to occur in during several site visits is presented in Table 2.1-1.

Except for two buildings and nearby associated areas, the region surrounding this SWMU is relatively undisturbed and is suitable habitat for much of DPG's wildlife. Disturbed areas are restricted to access roads and land immediately surrounding the large building and idle Explosive Test Shield Facility. Located on the west side of Camel's Back Ridge, this area is subjected to little human activity.

3.1.7.2 Evaluation of Detected Chemicals

Twenty-one chemicals, 19 inorganics and 2 organics, were detected in surficial soil samples at SWMU 20 (Table 2.6-1). Antimony, barium, calcium, and magnesium were the only chemicals whose concentration data distributions were considered above background concentrations according to the ANOVA comparison. HQs were computed for the entire list of detected chemicals when toxicological data were available and HQs representing total and incremental risk were evaluated. Screening-level mammalian toxicological data were not available for calcium, cobalt, iron, magnesium, potassium, and sodium. Screening-level avian toxicological data were not available for antimony, beryllium, calcium, cobalt, iron, magnesium, potassium, sodium, thallium, and toluene. However, almost all of these chemicals are considered to be essential nutrients and are toxic only at very high concentrations.

3.1.7.3 Evaluation of Site-Specific Information and Remedial Recommendation

The preliminary assessment for SWMU 20 showed that potential total risks were only from naturally occurring trace metals. Based on the ANOVA, only antimony, barium, calcium, and magnesium concentrations were above background concentrations. As previously stated, calcium and magnesium were not evaluated as they are considered essential nutrients and toxic only at very high concentrations. The ANOVA showed that the other metals were detected at concentrations consistent with background concentrations, and many of the maximum detected concentrations were less than the background UTLs for those metals. Thus, a relatively small percentage of the total potential risk may be due to anthropogenic sources. This comparison demonstrates that judgments on adverse impacts to receptors, and the remedial actions necessary to mitigate these perceived impacts, should be based on the increment of risk associated with concentrations above background. Additionally, because the computation of HQs in the preliminary ecological risk assessment uses many conservative assumptions (Section 2.6.2.3) to prevent the underestimation of potential risks, the true incremental risk estimates may actually be less than unity, indicating that there is negligible risk at this SWMU to the deer mouse and the horned lark and, by proxy through their conservative use, other receptors. The habitat at this SWMU is of a high quality and minimal ground surface disturbance has left much of the cryptogamic soil intact in the area.

The SWMU is currently inoperative and has not recently experienced significant human activity that would interfere with animal use of the local habitat. Intrusive remediation, such as soil removal or capping, would probably cause a more adverse impact to local wildlife populations and the vegetation than allowing the suspected contamination, if any, to remain and possibly attenuate naturally over time. Therefore, it is recommended that no corrective action related to suspected chemical contamination be performed at SWMU 20 based on risk results computed for the deer mouse and horned lark.

3.1.7.4 Ecological Risk Results for the Deer Mouse

Based only on the interpretation of HQ values previously described (Section 2.6.2.3), several detected metals would be predicted to cause risk at some level (Table 2.6-1). The computed HQs representing total potential risk from aluminum (631) and thallium (452) would indicate likely risk. Several other chemicals (antimony, 14; arsenic, 21; barium, 55) would indicate some potential total risk based on their predicted HQs, and negligible total risk was estimated for vanadium (HQ = 10).

If the background concentration is subtracted from the maximum concentration and that value used to compute an HQ, then the HQ represents the incremental risk to the receptor from levels of suspected inorganic contamination above naturally occurring levels. An examination follows of those metals with concentration data sets above the background concentration data set according to the ANOVA comparison.

An HQ of 14 was calculated from the maximum detected antimony concentration. The antimony background concentration (9.1 micrograms per gram [μg/g]) is equal to 83 percent of the maximum detected soil concentration (11 μg/g). Thus, incremental risk represents an HQ of 2 for the deer mouse from antimony (i.e., 14 minus 83 percent equals 2).

An HQ of 55 was calculated from the maximum detected barium concentration. The barium background soil concentration (400 milligrams per kilogram is equal to 62 percent of the maximum detected soil concentration (650 μg/g). Incremental risk, thus, represents an HQ of 21 to the deer mouse from barium.

Because the background concentration for these chemicals so nearly approximates the maximum detected concentration, the predicted incremental risk HQs are much smaller than the total risk HQs. The incremental risk estimates are likely to better approximate the actual adverse impacts, if any, to be experienced by the deer mouse population.

3.1.7.5 Ecological Risk Results for the Horned Lark

Based only on the interpretation of HQ values previously described (Section 2.6.2.3), several detected metals would be predicted to cause risk at some level (Table 2.6-2). The computed HQ representing potential total risk from aluminum (17) would indicate some potential risk. Several other metals (arsenic, 2; barium, 8; chromium, 2; lead, 1) would represent a negligible level of potential total risk. The maximum soil concentrations detected for aluminum, chromium, and lead were considerably lower than the background soil concentrations and the ANOVA comparison showed that these metals were detected at concentrations that are consistent with background concentrations. Thus, potential total risk is attributable entirely to background concentrations for these chemicals. Additionally, background concentrations of arsenic and barium nearly approximate the maximum detected concentrations and, therefore, the background concentrations of these metals contribute almost entirely to the potential total risk estimate. Thus, a relatively small percentage of the potential total risk may be due to anthropogenic sources.

If the background concentration is subtracted from the maximum concentration and that value used to compute an HQ, then the HQ represents the incremental risk to the receptor from levels of suspected metals contamination above naturally occurring levels. An examination follows of those metals with concentration data sets above the background concentration data set according to the ANOVA comparison.

An HQ of 8 was calculated from the maximum detected barium concentration. The barium background soil concentration (400 μg/g) is equal to 62 percent of the maximum detected soil concentration (650 μg/g). Incremental risk represents an HQ of 3 to the horned lark from barium.

Because the background concentrations of these chemicals nearly approximates the maximum detected concentration, the predicted incremental HQs are much smaller than the total risk HQs. The incremental risk estimates are more likely to better approximate the actual adverse impacts, if any, to be experienced by the horned lark population.

3.2 CLOSURE PERFORMANCE STANDARD

This closure plan is designed to provide for closure of SWMU 20 in a manner that will (1) protect human health and the quality of the environment; (2) control, minimize, or eliminate the escape of hazardous constituents to soil, surface water, groundwater, or the atmosphere during and after closure; and (3) minimize the need for further maintenance at the SWMU. These objectives are consistent with the requirements of UAC R315-101 that contain the following corrective action decision-making criteria to be used at DPG:

- Clean closure is allowed if the noncancer HI is less than 1.0 and the excess cancer risk is less than 1×10^{-6} for residential use.
- Site controls (or optional corrective action) are required if the excess cancer risk is greater than 1×10^{-6} for residential use and less than 1×10^{-4} for actual use and the noncancer HI is less than 1.0 for both residential and actual uses.
- Corrective action is required if the cancer risk is greater than 1×10^{-4} or the noncarcinogenic HI is greater than 1.0 for actual use.

There are no administrative rules requiring corrective action based on the results of the ecological risk assessment. Therefore, these risk results are evaluated subjectively.

In addition to the risk assessment results, the contamination assessment of the SWMU was evaluated with respect to the principle of nondegradation of the environment as required in UAC R315-101-3. This rule states that the unit is to be managed and closed in a way that the levels of contamination in groundwater, surface water, soil, and air will not increase after site management begins (during closure and post-closure care of the unit).

3.3 CLOSURE ACTIVITIES

Since current information indicates that there were no hazardous or other solid wastes disposed of at SWMU 20, no corrective action is necessary at either SWMU 20-1 or SWMU 20-2. Table 3.3-1 summarizes the risk assessment results and corrective action recommendations for closure of SWMU 20. There are no human health risks in excess of regulatory criteria under the hypothetical residential land-use scenario and no ecological risks present at this SWMU.

Table 3.3-1 • Corrective Action Recommendations for SWMU 20

Human Health Criteria

Receptors	Cancer Risk	Noncancer Hazard Index	Major COPCs in Risk Exceedance	Concentrations > Background or Risk-Based Cleanup Level	Recommendation
Hypothetical Residents	2.5×10^{-9}	2.5×10^{-5}	None	NA	No Action

Ecological Criteria

Biological Receptors	Major COPCs and Incremental Hazard Quotient	Concentration > Background or Cleanup Level	Recommendation
Deer Mouse	Antimony, 2; Barium, 21	Yes	No Action
Horned Lark	Barium, 3	Yes	No Action

Explosive Risk

Explosive Risk Level	Items Expected	Recommendation
None Expected	NA	No Action

Compliance with Nondegradation Principle

Potential Degradation Mechanism and Medium	Location	Recommendation
None	NA	No Action

NA - Not applicable

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Although SWMU 20 is closely associated with the Explosive Test Shield Facility, there is no explosive risk anticipated at SWMU 20 because no unexploded ordnance (UXO) were observed during sample location screening or are known to have been disposed of at the unit.

No releases of contamination to the environment are expected because no land disposal of hazardous wastes occurred at this unit, and no contaminated soil or residue is present at the unit that poses a risk to human health or the environment. Since no releases are expected, groundwater monitoring at SWMU 20 and a survey plat of the unit are not required. There is no requirement for decontamination, final cover, or site controls for closure of SWMU 20.

There is no planned future use of the unit and there are no closure activities at SWMU 20, so no schedule of closure activities or certification of closure is required. This unit will be considered clean-closed upon approval of this closure plan by the Utah Department of Environmental Quality (UDEQ), Division of Solid and Hazardous Waste.

3.4 POST-CLOSURE PLAN

Because SWMU 20 contains no hazardous waste and no hazardous waste will remain when the unit is closed, submission of a post-closure plan is not required.

3.5 PERMIT MODIFICATION

After the public comment period ends and the Closure Plan for SWMU 20 is approved, this SWMU will be deleted from Part A and from Tables 1 and 2 in Module IV of the DPG permit. Deletion of this unit from the permit is considered a Class 3 permit modification and may be deferred until the Phase II RFI is complete.

4.0 CLOSURE PLAN FOR SWMU 164—AVERY WASH RACK NO. 1

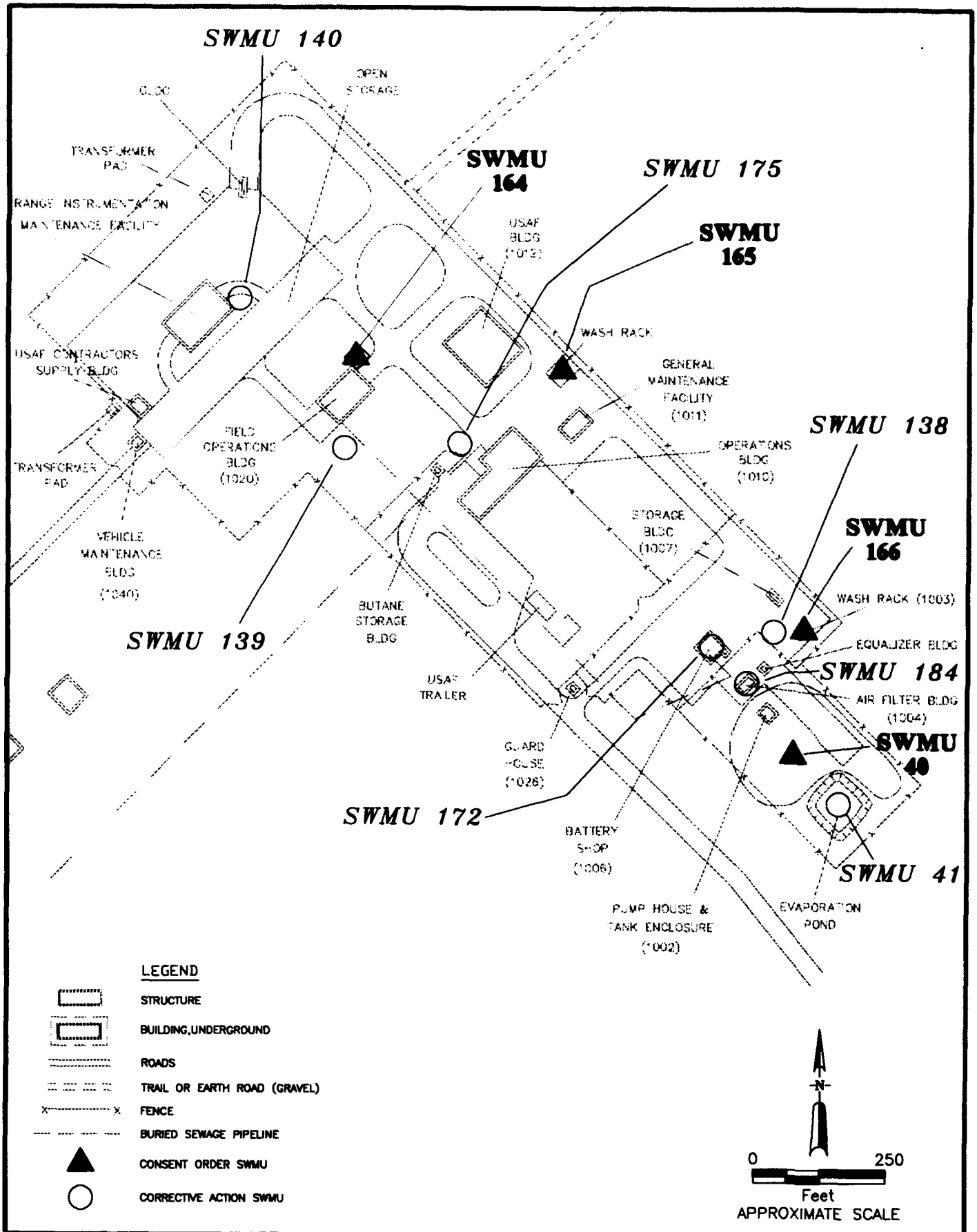
4.1 UNIT CHARACTERISTICS

4.1.1 SWMU Description and History

SWMU 164, known as the Avery Wash Rack No. 1, is located near the north side of Building 1020 in the north central section of Avery Technical Center (Figures 4.1-1 and 4.1-2). This unit is also located approximately 300 ft northwest of the Acid Neutralization Tank (Corrective Action SWMU 175), and 180 ft and 225 ft from two 90-day Hazardous Waste Holding Areas (Corrective Action SWMUs 139 and 140). SWMU 164 is located on level ground at an approximate elevation of 4,350 ft msl (EPIC 1986; DPG 1961b).

According to former and current DPG employees, the SWMU 164 wash rack was used by the Army between approximately 1980 and 1989 as a platform on which mud was cleaned from uncontaminated vehicles using water (Green 1991; Gourley 1991). Lubrication oils, solvents, fuel, and antifreeze are likely to have been removed from vehicles during washing, and are potential contaminants at the site (EBASCO 1992). SWMU 164 has not been used for vehicle cleaning since 1989, and there is no plan to use the unit in the future. The adjacent building is currently being used by the U.S. Air Force and is connected to the Avery sanitary sewer system on the southeast side of the building, away from the wash rack (DPG 1984a).

Site inspections were conducted by EBASCO personnel in October 1991 and February and July 1995. The wash rack, approximately 33 ft long, 20 ft wide, and 1 ft high, is built on native soil of a double layer of perforated Marston metal landing mats that are supported by 10 railroad rails (Figure 4.1-3). Minor vegetation was observed growing through and around the rack, and miscellaneous wooden debris was observed in 1991 on the ground to the east side of the mats (EBASCO/AGEISS 1993a). In July 1995, the entire area surrounding SWMU 164 was covered with gravel. An asphalt road enters Building 1020 near the SWMU. No stains were noted on the matting in 1991; the latter was still in place in 1995. However, the eastern section of the matting had been placed on top of the rack, and oil stains were noted on the gravel where DPG-EPO reported that oil was being drained from an Air Force vehicle in July 1995.

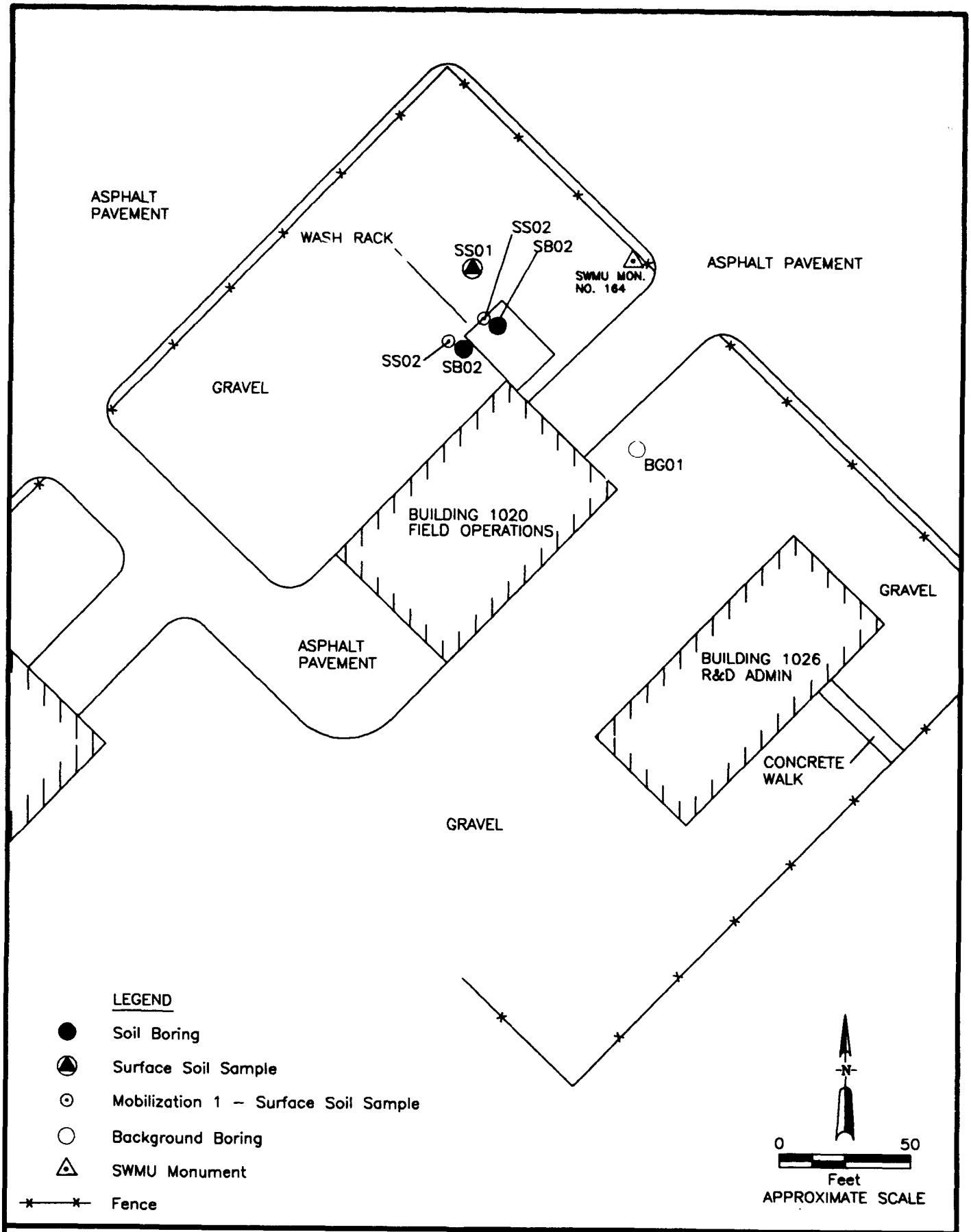


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Figure 4.1-1 SWMU 164 Location Map (Avery Technical Center)

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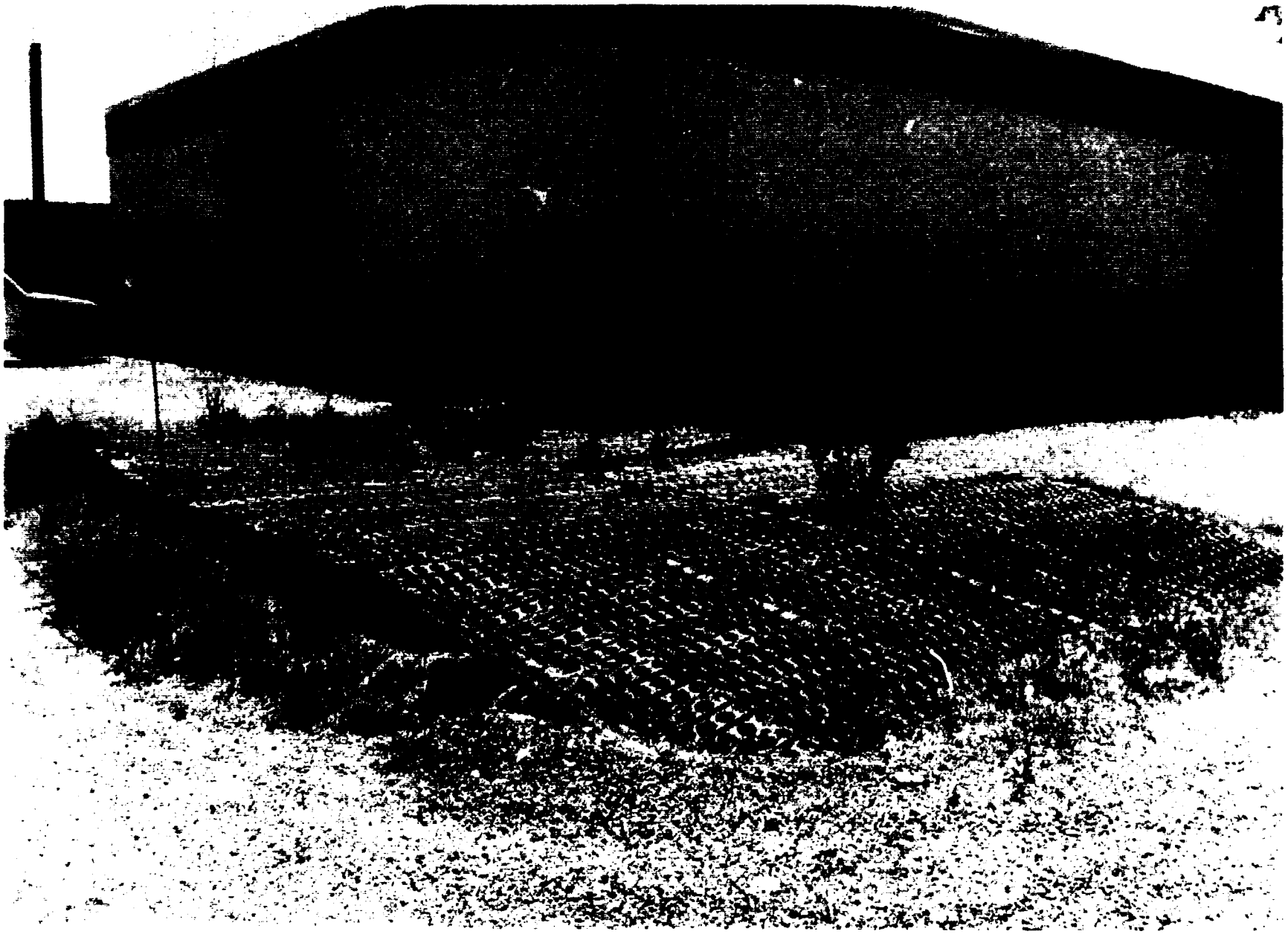


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Figure 4.1-2 SWMU 164 Sample Location Map



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Figure 4.1.3 SWMU 164
Avery Wash Rack No. 1
(Looking South)

4.1.2 Surface Water and Groundwater

There are no surface water features in the vicinity of SWMU 164. In general surface water flow in the Avery area is to the southwest toward Government Creek and Ditto (DPG 1984c).

Although there are no groundwater monitoring wells at SWMU 164, wells installed at the Evaporation Pond (Corrective Action SWMU 41) at the south end of Avery encountered unconfined groundwater at a depth of 20 ft (Parsons ES 1995). The closest water supply wells (wells 1, 2, 3, and 25) are located in Ditto. The principal water-bearing zones for fresh water in the deeper, confined aquifer in the general area of SWMU 164 are at depths ranging from 235 ft to 290 ft (USAEHA 1987). These zones are overlain by lacustrine clay with a low hydraulic conductivity. The groundwater in the upper portion of the clay forms the unconfined brackish groundwater aquifer. Although the screened intervals of the five water supply wells in the Ditto-Avery area are at depths between 168 ft and 290 ft, the static water levels in these wells are at depths between 3 ft and 12 ft, indicating a large pressure head in the confined freshwater aquifer. This upward gradient may cause the shallower brackish aquifer to be recharged from below. The deeper freshwater aquifer is primarily recharged by surface water that flows from the mountains into Government Creek Valley, where it infiltrates sand deposits on the edges of the valley.

4.1.3 Maximum Extent of Operations

No hazardous waste has been handled or disposed of at SWMU 164. Therefore, a survey plat of SWMU 164 is not required.

4.1.4 Nature and Extent of Contamination

Surficial and subsurface soil samples were collected from three surface soil locations and three soil borings, including one background location, to characterize the nature and extent of soil constituents at SWMU 164. The soil consisted of gravel near the surface mixed with silt to a depth of 2 ft. The soil is possibly fill or a mixture of fill and native soil. The samples were analyzed for metals, VOCs, SVOCs, TPHC, and cyanide.

Data validation resulted in some rejected and some blank qualified results. Results for several phenols and selenium were rejected; one cyanide result was missing; and some results for methylene chloride and di-n-butyl phthalate were blank qualified because of contamination in the field or laboratory blank. The constituents detected in SWMU 164 surficial and subsurface soil samples are summarized in Tables 4.1-1 and 4.1-2, respectively. Further discussion of the sampling results can be found in Section 32.2.1 of the final Interim Report (EBASCO 1995a).

Nine metals—antimony, arsenic, cadmium, chromium, copper, lead, mercury, thallium, and zinc—were detected at concentrations greater than the background UTL at SWMU 164 (Figure 4.1-4). Cadmium was detected in two Mobilization 1 samples at 2.4 µg/g (SS01, SS02); however, cadmium was not detected in two Mobilization 2 borings drilled adjacent to the previous detections. Four of these metals—chromium, lead, thallium, and zinc—were detected at concentrations slightly above the calculated background UTL at the background location (BG01). The nonparametric ANOVA results showed that the distributions of chromium, copper, lead, thallium, and zinc are significantly different from background at SWMU 164. Chromium detections at Mobilization 1 locations SS01 and SS02 (23 µg/g and 19 µg/g, respectively) were only slightly above background (17 µg/g), but well below the residential-use RBSL (140 µg/g). Copper was detected only slightly above the background UTL in one surface soil sample. Lead detections in surficial soil at Mobilization 1 locations SS01 and SS02 (61 µg/g and 81 µg/g, respectively) were both significantly above background (14 µg/g), but still well below the RBSL of 400 µg/g. Although the zinc detections (both 110 µg/g) at these locations were nearly twice background (59 µg/g), they were well below the RBSL for residential use (23,000 µg/g). Only arsenic and thallium exceeded the RBSLs for the hypothetical residential land-use scenario (0.35 µg/g and 29 µg/g, respectively). The background values for arsenic and thallium at DPG (13 µg/g and 35 µg/g, respectively) also exceed these RBSLs. Arsenic was detected at 14 µg/g (SS01) (only 1 µg/g above background) at one Mobilization 1 location, and thallium was detected at 50 µg/g (SB01, 3-ft to 4-ft interval). Neither metal exceeded the RBSLs (28 µg/g and 150 µg/g, respectively) for the industrial land-use scenario.

Five different organic compounds—acetone, bis(2-ethylhexyl)phthalate, methylene chloride, toluene, and total xylenes—and TPHC were detected in SWMU 164 soil samples, generally at low concentrations (Figure 4.1-5). Four VOCs (acetone, methylene chloride, toluene, and total xylenes) were detected at low ppb concentrations at five locations, including the background

Table 4.1-1 Summary of Constituents Detected in SWMU 164 Surficial Soil*

Analyte	Percent Detections	Total Samples	Total Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Background Value (µg/g)	Detections> Background Value
ACET	100	2	2	0.018	0.043	164SS02	NA	NA
AL	100	4	4	4,600	6,800	164SS02	19,000	0
AS	100	4	4	5.9	14	164SS01	13	1
B2EHP	25	4	1	0.69	0.69	164SS01	NA	NA
BA	100	4	4	130	270	164SS02	400	0
BE	25	4	1	0.84	0.84	164SS02	1	0
CA	100	4	4	86,000	150,000	164SS01	190,000	0
CD	50	4	2	2.4	2.4	164SS01	0.46	2
CO	100	4	4	2.3	3.3	164SS01	7.9	0
CR	100	4	4	11	23	164SS01	17	3
CU	100	4	4	12	27	164SS02	25	1
FE	100	4	4	7,500	9,500	164SS02	19,000	0
HG	25	4	1	0.07	0.07	164SS02	0.05	1
K	100	4	4	1,900	2,800	164SS02	7,400	0
MEC6H5	100	2	2	0.0017	0.0034	164SS01	NA	NA
MG	100	4	4	15,000	20,000	164SS01	29,000	0
MN	100	4	4	220	300	164SS02	520	0
NA	100	4	4	890	1,600	164SS02	11,000	0
NI	100	4	4	4.8	14	164SS01	17	0
PB	100	4	4	12	81	164SS02	14	3
SB	25	4	1	9.2	9.2	164SS01	9.1	1

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Table 4.1-1 Summary of Constituents Detected in SWMU 164 Surficial Soil*

Analyte	Percent Detections	Total Samples	Total Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Background Value (µg/g)	Detections> Background Value
TL	100	4	4	7.2	31	164SS01	35	0
TPHC	100	2	2	140	160	164SS01	NA	NA
V	100	4	4	16	19	164SS01	29	0
XYLEN	50	2	1	0.0016	0.0016	164SS01	NA	NA
ZN	100	4	4	39	110	164SS01	59	3

µg/g Micrograms per gram.
NA Not applicable. Background values cannot be determined for these analytes.
* Surficial soil samples collected from ground surface to depths of 1 ft in soil borings, and from ground surface to depths of 6 inches (0.5 ft) at surface soil (SS) locations. Analyte acronyms are defined in the Acronym List.

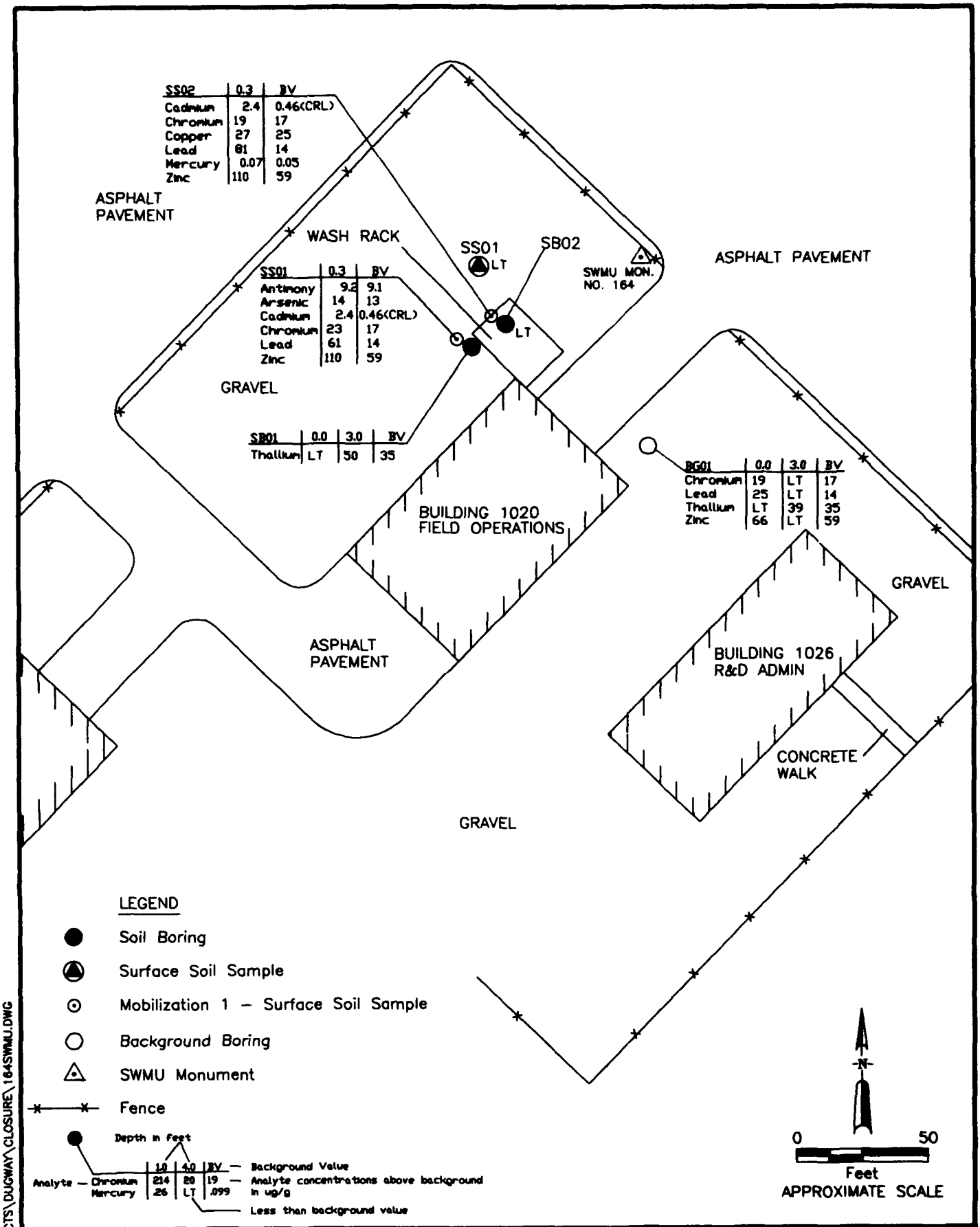
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Table 4.1-2 Summary of Constituents Detected in SWMU 164 Subsurface Soil*

Analyte	Percent Detections	Total Samples	Total Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Background Value (µg/g)	Detections> Background Value
ACET	67	3	2	0.015	0.018	164SB01	NA	NA
AL	100	3	3	5,800	16,000	164SB01	19,000	0
AS	100	3	3	3.7	11	164SB01	13	0
BA	100	3	3	160	280	164SB01	400	0
BE	67	3	2	0.67	0.76	164SB01	1	0
CA	100	3	3	32,000	130,000	164SB01	190,000	0
CH2CL2	100	1	1	0.027	0.027	164SB02	NA	NA
CO	100	3	3	2.7	5.9	164SB01	7.9	0
CR	100	3	3	6.3	14	164SB01	17	0
CU	100	3	3	6.2	16	164SB01	25	0
FE	100	3	3	6,600	13,000	164SB01	19,000	0
K	100	3	3	2,100	5,100	164SB01	7,400	0
MG	100	3	3	6,700	23,000	164SB01	29,000	0
MN	100	3	3	130	330	164SB01	520	0
NA	100	3	3	990	2,600	164SB01	11,000	0
NI	100	3	3	4.4	14	164SB01	17	0
PB	100	3	3	5.5	9	164SB02	14	0
TL	100	3	3	14	50	164SB01	35	2
V	100	3	3	18	29	164SB01	29	0
ZN	100	3	3	20	40	164SB01	59	0

µg/g Micrograms per gram.
NA Not applicable. Background values cannot be determined for these analytes.
* Subsurface soil samples collected from depths of 3 ft to 4 ft.
Analyte acronyms are defined in the Acronym List.

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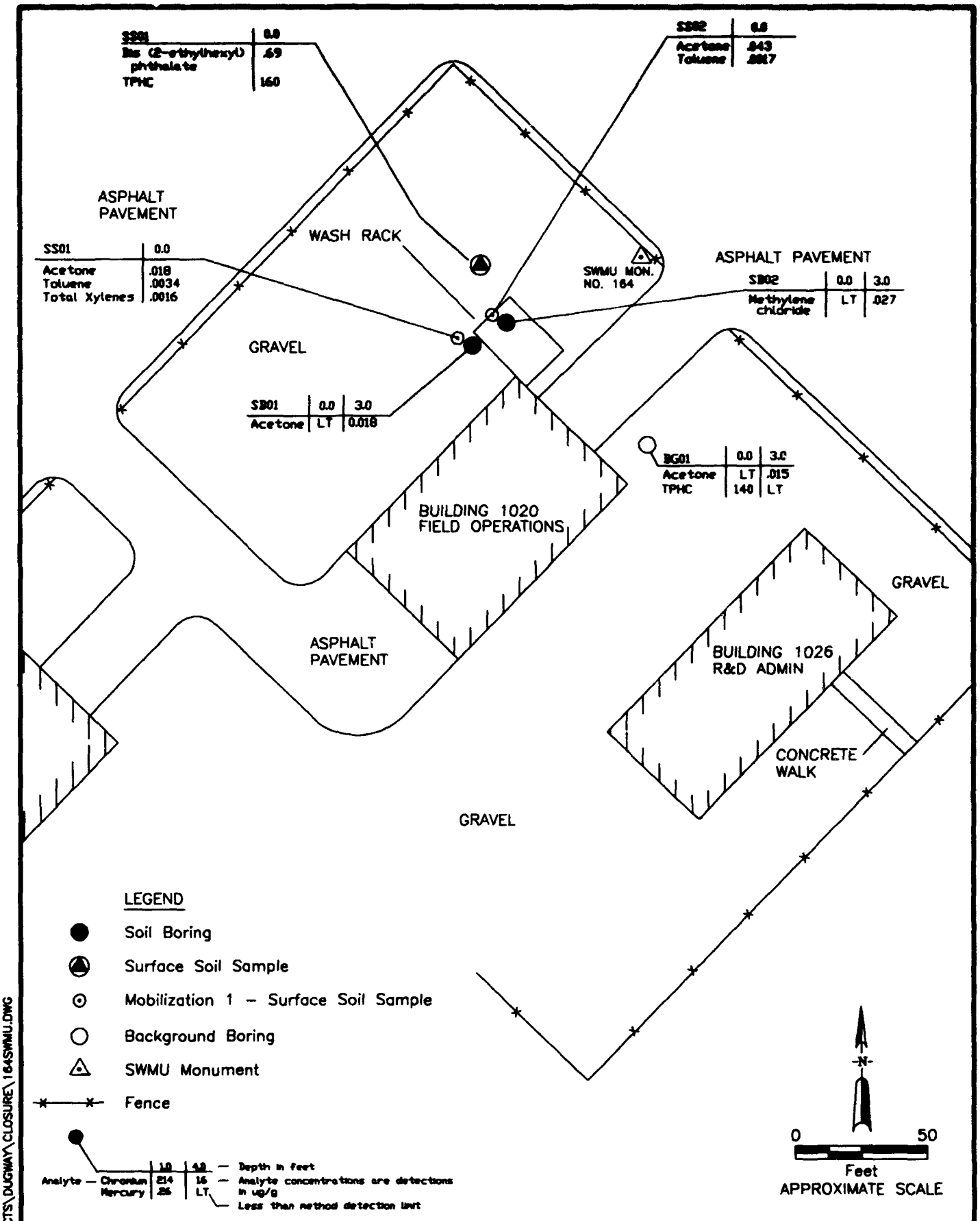


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Figure 4.1-4 SWMU 164
Distribution of Inorganics in Soil



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Figure 4.1-5 SWMU 164
Distribution of Organics in Soil

location. Methylene chloride may be a result of using chlorinated water at the wash rack, as it is formed during water chlorination (Micromedex 1996). The occurrence of toluene and xylenes could be related to petroleum products that may have been washed off dirty vehicles at the wash rack. Bis(2-ethylhexyl) phthalate, a plasticizer and common laboratory contaminant, was detected at only one location (SS01). The presence of TPHC in surficial samples from two locations, including the background boring, may be related either to petroleum products washed from the vehicles, to asphalt or asphalt-covered gravel in the sample, or to paved areas around the SWMU. The organic constituents detected at SWMU 164 will not persist in the soil due to aerobic biodegradation and short degradation half-lives. Additional information on contaminant fate and transport is found in Appendix M, Table M.1-1 of the final Interim Report (EBASCO 1995a).

4.1.5 Maximum Waste Inventory

The SWMU includes a wash rack that was used to wash uncontaminated vehicles with water. There is no potential for additional hazardous wastes, including residues, at SWMU 164, because the unit has not been used since 1989 and because there is no plan to manage hazardous waste at the unit in the future. Organic contamination detected in soil samples near the SWMU may be related to nearby maintenance activities at Building 1020 and on the graveled area around the wash rack or to asphalt paving rather than wastewater releases from the wash rack.

Since SWMU 164 was used to wash vehicles, there was a concern whether lubrication oils, solvents, fuel, or antifreeze could have been released from the vehicles during washing at the unit. Therefore, several surficial and subsurface soil samples were collected and analyzed for VOCs, SVOCs, metals, cyanide, and TPHC. As indicated in Section 4.1.4, five metals slightly above their background UTLs and six organic analytes at very low concentrations were detected in the soil samples collected at SWMU 164. The detected values were well below the RBSLs for four of the metals. The remaining metal, thallium is not believed to have been used or disposed of at this SWMU. Consequently, there are no significantly contaminated soil or residues resulting from vehicle washing operations at the SWMU.

4.1.6 Baseline Human Health Risk Assessment

This section presents the results of the baseline human health risk assessment developed for SWMU 164. The analysis is based on the soil analytical results presented in Section 4.1.4 and the risk assessment approach described in Section 2.5. As outlined by UAC R315-101, this approach provides a conservative evaluation of potential health risks associated with a hypothetical residential and optional evaluation of the actual use of SWMU 164.

4.1.6.1 Identification of COPCs

Using the selection criteria defined in Section 2.5 and Figure 2.5-2, the following five constituents were selected as soil COPCs for SWMU 164 (Table 4.1-3):

- Acetone
- Bis(2-ethylhexyl)phthalate
- Methylene chloride
- Toluene
- Xylenes

Toxicity profiles for these COPCs are provided in Appendix B.3. As discussed in the preceding sections, the results of soil sampling do not indicate any significant contaminant releases from previous use of the site. Concentrations of metals are generally comparable to background and probably reflect the natural variability of these constituents in soil. The ANOVA comparison identified lead as the only metal whose concentrations exceeded background concentrations. The EPA has established an interim soil lead level of 400 μg/g (EPA 1994b). The maximum detection of lead was 81 μg/g. Therefore, lead was eliminated from further evaluation in the risk assessment because it is well below the interim soil lead level set by the EPA. Two of the VOCs detected, acetone and methylene chloride, are possible laboratory contaminants and may not be site related. Toluene is a common fuel constituent.

Table 4.1-3 Summary of Constituents Detected in SWMU 164 Soil Samples and Identification of COPCs

Analyte	Detection Frequency No. of Hits	Detection Frequency Percent	Maximum (µg/g)	EPC 95% UCL (µg/g)	UTL (µg/g)	ANOVA COPC[1]	Essential Nutrient?	Few Detections > UTL	EPC Less Than RBSL	Evidence of Historical Use	Selected as COPC?[2]
Acetone	**4/5**	**80%**	**0.043**	**0.043**	--	--	**No**	--	--	--	**Yes**
Aluminum	7/7	100%	16,000	12,000	19,000	No	No	--	--	--	No
Antimony	1/7	14%	9.2	5.4	9.1	No	No	--	--	--	No
Arsenic	7/7	100%	14	11	13	No	No	--	--	--	No
Barium	7/7	100%	280	250	400	No	No	--	--	--	No
Beryllium	3/7	43%	0.84	0.67	1.0	No	No	--	--	--	No
Bis(2-ethylhexyl) phthlate	**1/7**	**14%**	**0.69**	**0.41**	--	--	**No**	--	--	--	**Yes**
Calcium	7/7	100%	150,000	140,000	190,000	No	Yes	--	--	--	No
Cadmium	2/7	29%	2.4	1.6	0.46	No	No	--	--	--	No
Chromium	7/7	100%	23	19	17	Yes	No	No	Yes	No	No
Cobalt	7/7	100%	5.9	4.5	7.9	No	No	--	--	--	No
Copper	7/7	100%	27	21	25	Yes	Yes	Yes	Yes	No	No
Iron	7/7	100%	13,000	11,000	19,000	No	Yes	--	--	--	No
Lead	7/7	100%	81	51	14	Yes	No	Yes	Yes	No	No
Magnesium	7/7	100%	23,000	21,000	29,000	No	Yes	--	--	--	No
Manganese	7/7	100%	330	300	520	No	No	--	--	--	No
Mercury	1/7	14%	0.07	0.07	0.05	No	No	--	--	--	No
Methylene Chloride	**1/3**	**33%**	**0.027**	**0.027**	--	--	**No**	--	--	--	**Yes**
Nickel	7/7	100%	14	13	17	No	No	--	--	--	No
Potassium	7/7	100%	5,100	3,900	7,400	No	Yes	--	--	--	No
Sodium	7/7	100%	2,600	1,800	11,000	No	Yes	--	--	--	No
Thallium	7/7	100%	50	37	35	Yes	No	Yes	No	No	No
Toluene	**2/5**	**40%**	**0.0034**	**0.0034**	--	--	**No**	--	--	--	**Yes**
TPHC	2/5	40%	160	140	--	--	No	--	--	--	No
Vanadium	7/7	100%	29	25	29	No	No	--	--	--	No
Xylene	**1/5**	**20%**	**0.0016**	**0.0016**	--	--	**No**	--	--	--	**Yes**
Zinc	7/7	100%	110	87	59	No	Yes	No	Yes	No	No

[1] SWMU 164 soil analytical results were compared with site-specific background data using the Wilcoxon rank-sum analysis of variance (ANOVA); this approach is documented in Section 2.3 and Appendix A.

[2] Soil COPCs are highlighted above (listed in bold) and were identified using the selection criteria shown in Figure 2.5-2.

-- Not applicable to organic analytes

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4.1.6.2 Analysis

As described in Section 2.5, the risk assessments for the Module 2 SWMUs were developed to evaluate both hypothetical residential and actual industrial land-use scenarios (Figure 2.5-1). The first step involved deriving RBSLs for a hypothetical residential land-use scenario using the current RME and toxicological parameters required by UAC 315-101 or recommended by EPA guidance (Table 2.5-1). This scenario assumes unrestricted land use and represents the most conservative analysis of potential soil exposures for the Module 2 SWMUs. The second step involved deriving RBSLs for an industrial land-use scenario (Table 2.5-1), which serves as a conservative approximation of actual use of this part of DPG. However, since the risk calculated for the residential use scenario qualifies the unit for clean closure, it is not necessary to calculate the lower risk that would apply for the actual site use.

SWMU 164 located in the north-central section of Avery. For current site conditions, the most likely exposure scenario would involve occasional access by a site maintenance worker. The industrial land-use scenario also applies to foreseeable future uses of SWMU 164, given the unit's proximity to Avery. However, the industrial-use evaluation is also conservative since long-term exposures are not expected, and the SWMU's small size probably does not constitute a reasonable area over which to average human exposures.

4.1.6.3 Risk Characterization

Hypothetical Residential Use Scenario

Table 4.1-4 summarizes the results of the risk assessment developed for exposures to SWMU 164 surface and subsurface soil under the hypothetical residential land-use scenario. Cancer risks and noncancer HIs presented in this table were calculated using the RBSLs listed in Table 2.5-2 in accordance with the methods, equations, and assumptions outlined in Section 2.5 and Appendix B.1. The total cancer risk and HI calculated for residential use of SWMU 164 are 2.4×10^{-8} and 4.5×10^{-4}, respectively. The cancer risk estimate is well below 10^{-6}, qualifying the SWMU for risk-based closure (UAC R315-101). The HI is below the target HI criterion of 1.0 for noncancer endpoints.

Table 4.1-4 Results of SWMU 164 Cancer Risk And Hazard Index Calculations for the Hypothetical Residential Land-Use Scenario

Chemical of Potential Concern	Carcinogenic RBSL* (µg/g)	EPC (µg/g)	Associated Cancer Risk[1]	Noncarcinogenic RBSL* (µg/g)	EPC (µg/g)	Associated HQ[2]
Acetone	--	0.043	--	4,700	0.043	9.3E-06
Bis(2-ethylhexyl) phthalate	22	0.041	1.9E-08	940	0.41	4.3E-06
Methylene Chloride	6	0.027	4.5E-09	7	0.027	9.5E-06
Toluene	--	0.0034	--	490	0.0034	3.4E-06
Xylenes	--	0.0016	--	320	0.0016	1.7E-08
	Total Cancer Risk:		**2.4E-08**	**Total Hazard Index (HI):**		**4.5E-04**

1 Cancer risks were calculated using the following equation: Risk = (EPC/RBSL)*TCR, where TCR equals a target cancer risk level of 10^{-6} (or 0.000001). Table 2.5-1 summarizes the underlying exposure assumptions; Appendix B.1 documents the RBSL equations and calculations.

2 Chemical-specific HQs were calculated by dividing the EPC by the noncarcinogenic RBSL (Section 2.5.5 and Appendix B.1).

-- Endpoint not applicable

* RBSL for residential land-use scenario (Table 2.5-2 and Appendix B.1)

4.1.6.4 Summary

The results of the human health risk assessment indicate that the risks posed to potential human receptors at SWMU 164 are negligible for the hypothetical residential land use, and that risks associated with current site conditions (corresponding to minimal human exposure) would actually be lower than those estimated for the residential land-use scenario. Based on state of Utah guidelines and the decision framework illustrated in Figure 2.5-1, these risk results support a no further action designation for this SWMU.

4.1.7 Ecological Risk Assessment

4.1.7.1 Ecological Conditions

The habitat surrounding SWMU 164 and the entire Avery compound is primarily greasewood. The SWMU proper is bare of vegetation and is considered unsuitable for wildlife use due to human activity and the gravel cover. This very small SWMU is surrounded by other development such as buildings, pavement, and gravel. These areas within the Avery compound are also considered poor habitat for DPG wildlife. A list of observed species and the habitats they were found to occur in during several site visits is presented in Table 2.1-1.

4.1.7.2 Evaluation of Detected Chemicals

Twenty-seven chemicals, 21 inorganics and 6 organics, were detected in surficial soil samples at SWMU 164 (Table 2.6-3). Of the metals, only lead was significantly above DPG background levels according to ANOVA results. Chromium, copper, thallium, and zinc were also identified by the ANOVA to be above background; however, only one copper and no detections of thallium were above the background UTL. Furthermore, there is no historical evidence that any of these metals was used or disposed of at the SWMU. However, HQs were computed for the entire list of detected analytes and HQs representing total and incremental risk were evaluated. In addition, a conservative BAF derived from mink liver (Wren *et al.*, 1987) was applied to the preliminary HQ (0.004) for mercury for deer mouse to account for its potentially bioaccumulative properties, even though the maximum detected concentration of mercury (0.07 μg/g) was essentially the same as the background

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concentration (0.073 µg/g). The final mercury HQ of 0.1 is based on the total risk from mercury to the deer mouse (Table 2.6-3). Although some forms of mercury may bioaccumulate under certain conditions, an avian BAF was not found in the literature that could be applied in a conservative fashion. Screening-level mammalian toxicological data were not available for calcium, chrysene, cobalt, iron, magnesium, potassium, and sodium. Screening-level avian toxicological data were not available for acetone, antimony, benzo(a)pyrene, beryllium, calcium, chrysene, cobalt, iron, magnesium, potassium, sodium, thallium, toluene, and xylene. However, most of the inorganic chemicals are considered to be essential nutrients and are only toxic at very high concentrations.

4.1.7.3 Evaluation of Site-Specific Information and Remedial Recommendation

The preliminary assessment for SWMU 164 showed that potential total risks were only from naturally occurring trace metals. In many instances, the ANOVA comparison showed that these metals were detected at concentrations consistent with background concentrations, and the maximum detected concentrations were less than the background UTLs for those metals. This comparison demonstrates that judgments on adverse impacts to receptors, and the remedial actions necessary to mitigate these perceived impacts, should be based on the increment of risk associated with concentrations above background. Additionally, because the computation of HQs in the preliminary ecological risk assessment uses many conservative assumptions (Section 2.6.2.3) to prevent the underestimation of potential risks, the true incremental risk estimates may actually be less than or near unity, indicating that there is negligible risk at this SWMU to the deer mouse and the horned lark and, by proxy through their conservative use, other receptors.

This SWMU is not likely to provide any suitable habitat for DPG wildlife because it is covered by gravel and pavement and located within a heavily used technical center. Due to the poor habitat and unlikelihood of exposure to DPG wildlife from site contamination, if actually present at toxic levels, it is recommended that no corrective action related to suspected chemical contamination be performed at SWMU 164 based on risk results computed for the deer mouse and horned lark.

4.1.7.4 Ecological Risk Results for the Deer Mouse

Based only on the interpretation of HQ values previously described (Section 2.6.2.3), several detected trace metals would be predicted to cause risk at some level (Table 2.6-3). The computed HQs representing total potential risk from aluminum (550) and thallium (633) are considered to indicate likely risk. Several other chemicals (arsenic, 17; antimony, 11; barium, 23;) are considered to possibly indicate some potential total risk based on their predicted HQs, and negligible total risk was estimated for vanadium (HQ = 8). For aluminum, barium, thallium, and vanadium, the maximum soil concentration detected was considerably lower than the background UTL soil concentrations. Although the HQs are very high for aluminum and thallium, the total potential risk for these chemicals is attributable entirely to background concentrations. Additionally, both the ANOVA and UTL comparisons showed that the background concentrations of antimony and arsenic contributed the total potential risk estimate for these metals. Thus, a relatively small percentage of the total potential risk may be due to anthropogenic sources. Of the metals above background, only lead (HQ = 0.9) had an HQ that approached a value of 1.

4.1.7.5 Ecological Risk Results for the Horned Lark

Based only on the interpretation of HQ values previously described (Section 2.6.2.3), several detected inorganic chemicals are predicted to cause risk at some level (Table 2.6-4). The computed HQs representing potential total risk from aluminum (15) and lead (17) are considered to possibly indicate some potential risk. Several other chemicals (arsenic, 1; barium, 3; chromium, 6; mercury, 3; and zinc, 2) are considered to represent a negligible level of potential total risk. The maximum soil concentrations detected for aluminum and barium were considerably lower than the background soil concentrations. For these chemicals, potential total risk is likely attributable entirely to background concentrations. Additionally, background concentrations of arsenic, chromium, and mercury nearly approximate the maximum detected concentrations and, therefore, the background concentrations of these metals contribute almost entirely to the potential total risk estimate. Thus, a relatively small percentage of the potential total risk may be due to anthropogenic sources.

If the background concentration is subtracted from the maximum concentration and that value used to compute an HQ, then the HQ represents the incremental risk to the receptor from levels of suspected inorganic contamination above naturally occurring levels. An examination follows of the metals with HQs greater than 1 and concentration datasets above the background concentration dataset according to the ANOVA comparison.

An HQ of 6 was calculated from the maximum detected chromium concentration. The chromium background soil concentration (17 µg/g) is equal to 74 percent of the maximum detected soil concentration (23 µg/g). Incremental risk represents an HQ of 2 to the horned lark from chromium (i.e., 6 minus 74 percent equals 2).

An HQ of 17 was calculated from the maximum detected lead concentration. The lead background soil concentration (14 µg/g) is equal to 17 percent of the maximum detected soil concentration (81 µg/g). Incremental risk represents an HQ of 14 to the horned lark from lead (i.e., 17 minus 17 percent equals 14).

An HQ of 2 was calculated from the maximum detected zinc concentration. The zinc background soil concentration (59 µg/g) is equal to 54 percent of the maximum detected soil concentration (110 µg/g). Incremental risk represents an HQ of 1 to the horned lark from zinc (i.e., 2 minus 54 percent equals 1).

Because the background concentration for some of these chemicals so nearly approximates the maximum detected concentration, the predicted incremental risk HQs are much smaller than the total risk HQs. The incremental risk estimates are more likely to better approximate the actual adverse impacts, if any, to be experienced by the horned lark population.

4.2 CLOSURE PERFORMANCE STANDARD

This closure plan is designed to provide for closure of SWMU 164 in a manner that will (1) protect human health and the quality of the environment; (2) control, minimize, or eliminate the escape of

hazardous constituents to soil, surface water, groundwater, or the atmosphere during and after closure; and (3) minimize the need for further maintenance at the SWMU. These objectives are consistent with the requirements of UAC (R315-101) that contain the following corrective action decision-making criteria to be used at DPG:

- Clean closure is allowed if the noncancer HI is less than 1.0 and the excess cancer risk is less than 1×10^{-6} for residential use.

- Site controls (or optional corrective action) are required if the excess cancer risk is greater than 1×10^{-6} for residential use and less than 1×10^{-4} for actual use and the noncancer HI is less than 1.0 for both residential and actual uses.

- Corrective action is required if the cancer risk is greater than 1×10^{-4} or the noncarcinogenic HI is greater than 1.0 for actual use.

There are no administrative rules requiring corrective action based on the results of the ERA. Therefore, these risk results are evaluated subjectively.

In addition to the risk assessment results, the contamination assessment of the SWMU was evaluated with respect to the principle of nondegradation of the environment as required in UAC R315-101-3. This rule states that the unit is to be managed and closed in a way that the levels of contamination in groundwater, surface water, soil, and air will not increase after site management begins (during closure and post-closure care of the unit).

4.3 CLOSURE ACTIVITIES

Table 4.3-1 summarizes the risk assessment results and corrective action recommendations for closure of SWMU 164. The human health risk assessment results indicate that under a hypothetical residential land-use scenario, the total cancer risk is 2.4×10^{-8} and the HI is 4.5×10^{-4}. The total cancer risk does not exceed the upper bound of the carcinogenic risk range for which risk-based closure is permitted.

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Table 4.3-1 • Corrective Action Recommendations for SWMU 164

Human Health Criteria

Receptors	Cancer Risk	Noncancer Hazard Index	Major COPCs in Risk Exceedance	Concentrations > Background or Risk-based Cleanup Level	Recommendation
Hypothetical Residents	2.4×10^{-8}	4.5×10^{-4}	None	NA	No Action

Ecological Criteria

Biological Receptors	Major COPCs and Incremental Hazard Quotient	Concentrations > Background or Cleanup Level	Recommendation
Deer Mouse	None	No	No Action
Horned Lark	Chromium, 2; Lead, 14; Zinc, 1	Yes	No Action

Explosive Risk

Explosive Risk Level	Items Expected	Recommendation
None Expected	NA	No Action

Compliance with Nondegradation Principle

Potential Degradation Mechanism and Medium	Location	Recommendation
None	NA	No Action

NA - Not applicable

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The results of the ecological risk assessment showed there are low potential risks to the horned lark from exposure to four metals above background in surface soil at SWMU 164, and because the SWMU is in a developed industrial area, wildlife exposure is unlikely to occur. The incremental HQs for chromium, lead, and zinc are 2, 14, and 1. On this basis, no corrective action is recommended at SWMU 164.

There is no explosive risk anticipated because no UXO have been observed at the SWMU or are known to have been handled or disposed of at the SWMU. Corrective action including decontamination and final cover of soil is not necessary during closure of SWMU 164, and there is no hazardous waste present at the unit. TPHC was detected at concentrations far below the waste oil cleanup guideline of 500 parts per million (ppm) established by UDEQ, so further degradation of the environment is not expected. Consequently, groundwater monitoring at SWMU 164 and a survey plat of the unit are not required.

There is no planned future use of the unit to manage solid or hazardous wastes and there are no closure activities at SWMU 164, so a schedule of closure activities or certification of closure is not required. This unit will be considered clean-closed upon approval of this closure plan by UDEQ, Division of Solid and Hazardous Waste.

4.4 POST-CLOSURE PLAN

Because SWMU 164 contains no hazardous waste and no hazardous waste will remain in the unit when it is closed, submission of a post-closure plan is not required.

4.5 PERMIT MODIFICATION

After the public comment period ends and the Closure Plan for SWMU 164 is approved, this SWMU will be deleted from Part A and from Tables 1 and 2 in Module IV of the DPG permit. Deletion of this unit from the permit is considered a Class 3 permit modification and may be deferred until the Phase II RFI is complete.

5.0 CLOSURE PLAN FOR SWMU 166—AVERY WASH RACK NO. 3

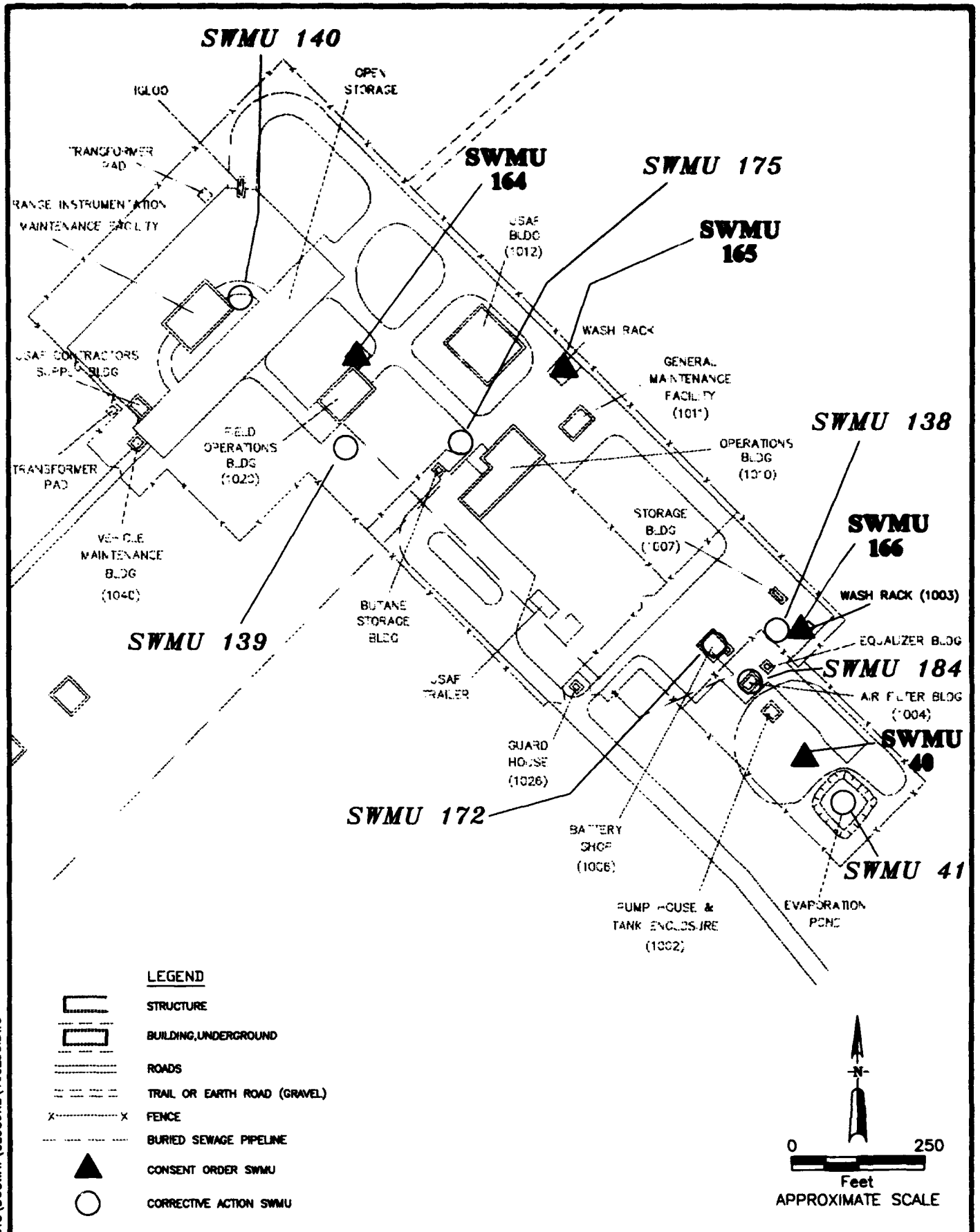
5.1 UNIT CHARACTERISTICS

5.1.1 SWMU Description and History

SWMU 166, known as the Avery Wash Rack No. 3, is located approximately 60 ft northeast of the Avery Battery, PCB, and 3X Metal Storage Area (SWMU 40) and 150 ft east of Building 1006 (Corrective Action SWMU 172), along the eastern perimeter fence of Avery (Figure 5.1-1). The unit is also approximately 300 ft north of the Evaporation Pond (Corrective Action SWMU 41) and 50 ft southeast of a 90-Day Hazardous Waste Holding Area (Corrective Action SWMU 138). SWMU 166 is located on an asphalt pad at approximately 4,350 ft msl (DPG 1961b; EPIC 1986).

The wash rack was operated from the 1950s through 1961 (EBASCO 1992). According to former DPG employees, it was used to wash dolly-type railcars that had held items during irradiation with radioactive cobalt (Hanson 1995a; Green 1991; EBASCO/AGEISS 1993a). The cobalt source (Co^{60}) was used to kill bacteria with high-energy gamma radiation in food-preservation experiments conducted in Building 1010, and would not have radioactively contaminated the railcars or dust or other material on them. The railcars were washed with soapy water to remove dirt and dust both prior to and after going into Building 1010 for reasons of cleanliness and to minimize the risk of food contamination. Food items to be irradiated were placed in No. 10 cans for the experiments. After the experiments were completed, the containers were removed from the railcars in Building 1010 and eventually shipped to one of four universities (Syracuse University, University of Utah, University of Oregon, or University of Washington) for analysis (Hanson 1995a). The wash rack was used approximately four times each year while the experiments were being conducted (Hanson 1995b).

The concrete wash rack measures approximately 30 ft by 24 ft (Figure 5.1-2). A single standard-gauge railroad track formerly ran from Building 1010, through Building 1007, and terminated at the wash rack. The track between Building 1007 and the wash rack is still in place. The track straddles a single-chamber sump, which is 26 inches square and centrally located within the wash

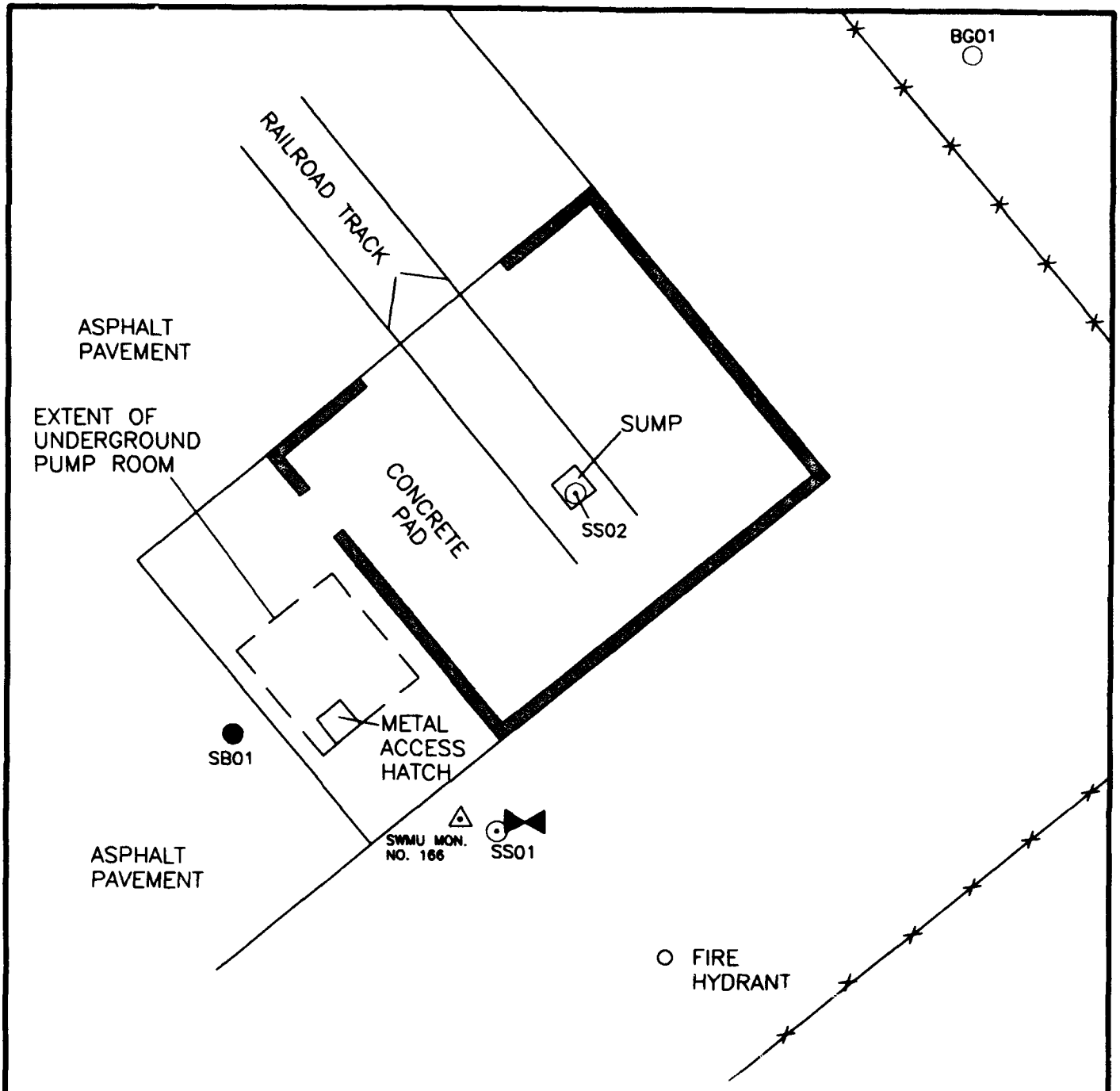


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Figure 5.1-1 SWMU 166
Location Map (Avery Technical Center)



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Figure 5.1-2 SWMU 166 Sample Location Map

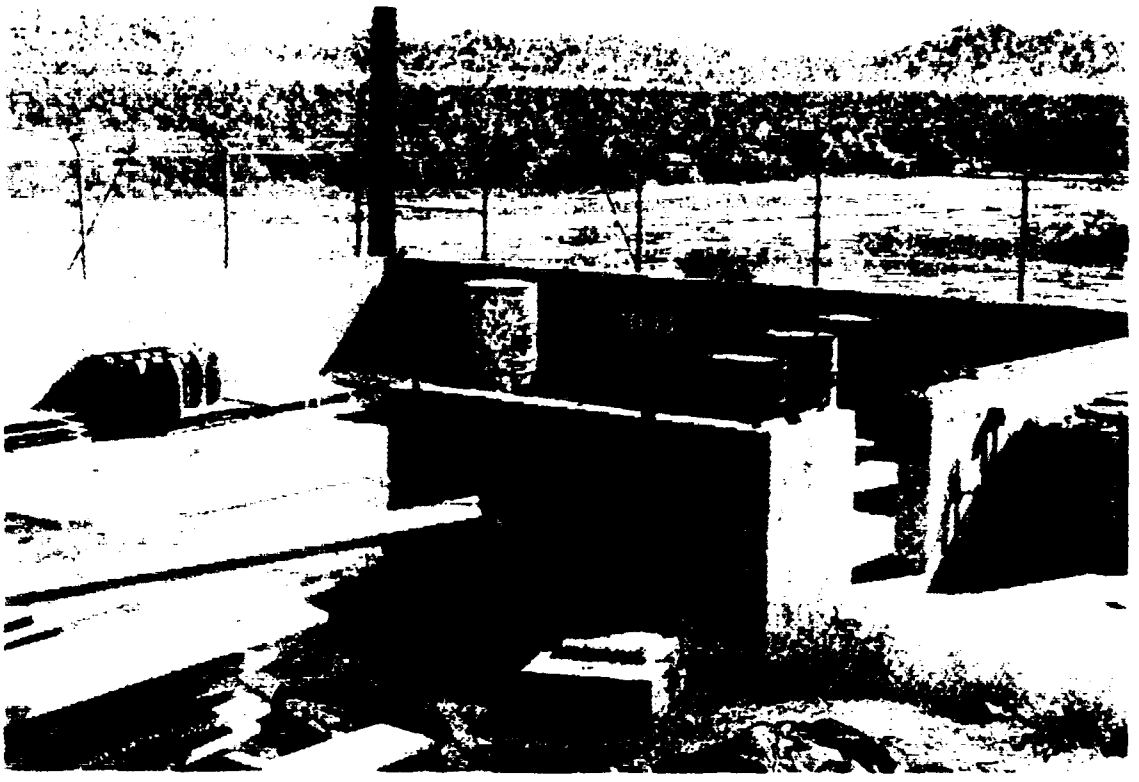
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rack. The concrete pad slopes toward the sump, which collected solid and liquid waste from all parts of the pad. A concrete wall, approximately 4 ft high and 8 inches thick, surrounds the perimeter of the rack. According to a former DPG employee, the wash rack was not enclosed by higher walls, but was once covered by a sheet-metal roof 8 ft above the concrete pad (Hanson 1995a). A shallow, ditch-like depression approximately 6 inches deep runs southward from the south corner of the rack for approximately 10 ft before it ends at a gate valve. The gate valve is connected to the water supply line that runs around the perimeter of Avery Technical Center and was used to supply water to the wash rack when it was in use (Jorgensen 1994). Washdown water was provided to the wash rack by hoses attached to 1-inch pipes that run up the outside of the concrete walls (Hanson 1995c). Adjacent to the southwest side of the concrete wall is a 10-ft by 22-ft concrete pad overlying an underground pump room that is 7.5 ft square and 8 ft deep. The metal hatch to the pump room vault is approximately 6 inches above grade and allowed access to a network of piping and control valves when the unit was in operation. The piping received wastewater from the sump at the wash rack. The pump room was used to send wastewater from the wash rack to the 1,000-gallon wastewater collection tanks located in the underground vault in Building 1002 within the boundaries of SWMU 40 (Hanson 1995a). The SWMU was never connected to the Avery Sanitary Sewer System (DPG 1968, DPG 1984a). A shallow depression marks the location of the line between the pump room and Building 1002.

Because SWMU 166 was thought to be associated with radioactive materials, a radiation survey was conducted during Mobilization 1 at SWMU 166 to determine if such contamination was present. The radiation survey was conducted prior to collecting soil samples and evaluated alpha, beta, and gamma radiation. Two 4-ft-wide pathways were screened across the SWMU and at the opening into the underground pump room. No radiation was detected above background levels, and the levels measured in soil samples were at or below the background levels. In addition, exit-radiation screening levels for personnel were below the prescreening background levels (EBASCO/AGEISS 1993a).

An initial visual site inspection was conducted at SWMU 166 in October 1991. At that time, the wash rack appeared as described above, with vegetation growing in and immediately around the sump, at the edge of the concrete wall, and in the vicinity of the ditch and surrounding area. Construction materials and possible hazardous materials were being stored in the unit at that time. Several 55-gallon drums and a number of 5-gallon cans were stored on wooden pallets within the wash rack area. Miscellaneous scrap metal, lumber, wire, tarps, and several unmarked 55-gallon drums were also present along the outer perimeter of the wash rack wall (Figure 5.1-3a). According to Victor Warr, a former DPG employee who worked at Avery, these materials belonged to Grid Operations, who used SWMU 166 as a storage area for petroleum, oil, and lubricants (POL). The 5-gallon cans contained fuel, while blue drums contained hydraulic fluid or oil. Yellow cans contained antifreeze (Warr 1996). According to Mr. Warr and Paul Gourley, (1996) who also worked for Grid Operations, the POL was used for heavy equipment utilized by Grid Operations. During subsequent site inspections in September 1993 and August 1994, it was determined that liquid from the sump was directed to the underground piping system in the pump room vault for discharge routing. In addition, it was noted that materials were no longer stored within the enclosed portion of the unit. An inspection in June 1995 indicated that the piping in the underground vault had been plugged and that no materials were being stored in the unit (Figure 5.1-3b). Two concrete footings are present on the floor of the underground vault, and an 18-inch-diameter sump is located in the west corner of the vault. In June 1995, there was no pump in the vault and only plugged piping along the walls of the vault. There are no plans to use SWMU 166 as a wash rack or hazardous materials storage area in the future.

According to a discussion during the March 1996 site inspection, UDEQ considers the underground piping from the sump to the vault and from the vault to the underground treatment tanks at Building 1002 to be part of Corrective Action SWMU 41. No visible staining of the concrete, except for rust, was observed at SWMU 166 during this inspection.



(A) 1991 Photograph



(B) 1995 U.S. Army Photograph

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Figure 5.1-3 SWMU 166
Avery Wash Rack No 3
(Looking Southeast)

5.1.2 Surface Water and Groundwater

In general, surface water at Avery Technical Center drains to the southwest toward Ditto (Figure 2.1-5). However, in the immediate vicinity of SWMU 166 surface water is directed to the southeast (DPG 1984c). A linear, 6-inch-deep depression extends about 5 ft from the southern corner of the wash rack and ends at a gate valve. It is possible that runoff could pond in this depression. Otherwise, runoff can be expected to flow away from the unit toward the Avery perimeter security fence. Water collecting inside the walls at the SWMU is directed to the sump, which currently has no outlet.

No monitoring wells were installed at SWMU 166; however, shallow groundwater was encountered at a depth of 20 ft in wells to the southwest at Corrective Action SWMU 41 (Parsons ES 1995). General hydrogeologic information in this area is available from the Ditto and Avery area supply wells. The principal water-bearing zones for fresh water in the deeper confined aquifer in the general area of SWMU 166 are between 235 and 290 ft below ground surface. This aquifer is overlain by lacustrine clays of low hydraulic conductivity. The groundwater in the upper portion of the clays forms the unconfined brackish aquifer that occurs between 20 and 40 ft below ground surface in this area. Although the screened intervals of the five water supply wells in the Ditto area are at depths between 168 and 290 ft, the static water levels in these wells are at depths between 3 and 12 ft, indicating a large pressure head in the confined freshwater aquifer. This upward gradient may cause the shallower brackish aquifer to be recharged from below. The deeper freshwater aquifer is primarily recharged by surface water that flows from the mountains to Government Creek Valley, where it infiltrates the sandy deposits near the mountain fronts.

5.1.3 Maximum Extent of Operations

Although POL have been stored there, no hazardous waste has been handled or disposed of at SWMU 166. Therefore a survey plat of SWMU 166 is not required.

5.1.4 Nature and Extent of Contamination

Surficial and subsurface soil samples were collected from four locations, including two borings, to characterize the nature and extent of soil constituents at SWMU 166. All samples at SB01 were collected beneath a 0.3-ft-thick layer of asphalt. The shallow sample was taken in road base material consisting of crushed aggregate and sand, with silty sand beneath the aggregate. The soil is probably fill or a mixture of fill and native soil. One of the surficial samples, SS02, was collected from sediment in the sump at the wash rack. This sample consisted of yellowish-brown silty sand with minor organic debris. One of the soil borings was the background location for the SWMU, and the other boring (SB01) was located in the asphalt pad southwest of the SWMU. Samples were analyzed for metals, VOCs, SVOCs, TPHC, and cyanide. Data validation indicated that all reported values are acceptable and 100 percent complete.

The constituents detected in SWMU 166 surficial and subsurface soil samples are summarized in Tables 5.1-1 and 5.1-2, respectively. No analytes were detected above both the background value and the RBSL. Further discussion of the sampling results can be found in Section 34.2.1 of the final Interim Report (EBASCO 1995a).

Eight metals—barium, cadmium, calcium, lead, magnesium, manganese, nickel, and zinc—were detected in SWMU 166 soil samples at concentrations slightly above the background values (Figure 5.1-4). The results of a nonparametric ANOVA showed that the distributions of four metals (antimony, arsenic, barium, and calcium) are different from background. Although the distribution of antimony differed from background, all detections of antimony were below the background UTL. Since the other three metals are not suspected contaminants at this SWMU, these results may simply reflect natural variability of these metals in the soil. Arsenic is not a suspected contaminant at this SWMU and the calculated ANOVA statistic only slightly exceeds the critical statistic. Additionally, arsenic concentrations are below the background value of 13 µg/g in all of the SWMU 166 samples (Section 2.3). Cadmium was not shown by the ANOVA to be significantly above background. However, one cadmium detection was approximately five times the background UTL. This concentration is still well below the RBSL for this metal.

Table 5.1-1 Summary of Constituents Detected in SWMU 166 Surficial Soil*

Analyte	Percent Detections	Total Samples	Total Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Background Value (µg/g)	Detections> Background Value
ACET	50	2	1	0.045	0.045	166SS01	NA	NA
AL	100	3	3	3,100	7,400	166SS02	19,000	0
AS	100	3	3	7	10	166SB01	13	0
BA	100	3	3	210	300	166SS02	400	0
BE	67	3	2	0.74	0.74	166SS01	1	0
CA	100	3	3	110,000	200,000	166SB01	190,000	1
CD	33	3	1	2.6	2.6	166SS01	0.46	1
CH2CL2	50	2	1	0.0045	0.0045	166SS01	NA	NA
CO	67	3	2	2.8	4.4	166SS02	7.9	0
CR	100	3	3	4.1	11	166SS01	17	0
CU	100	3	3	4.4	14	166SS01	25	0
FE	100	3	3	4,700	9,300	166SS02	19,000	0
K	100	3	3	1,300	2,900	166SS01	7,400	0
MEC6H5	50	2	1	0.013	0.013	166SS01	NA	NA
MG	100	3	3	15,000	42,000	166SB01	29,000	1
MN	100	3	3	140	310	166SS02	520	0
NA	100	3	3	760	910	166SS02	11,000	0
NI	100	3	3	3.6	9.5	166SS02	17	0
PB	100	3	3	3.7	36	166SS01	14	1
SB	67	3	2	8.1	8.6	166SB01	9.1	0
TL	67	3	2	8.1	9.5	166SS01	35	0

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Table 5.1-1 Summary of Constituents Detected in SWMU 166 Surficial Soil*

Analyte	Percent Detections	Total Samples	Total Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Background Value (µg/g)	Detections> Background Value
TPHC	100	1	1	63	63	166SB01**	NA	NA
V	100	3	3	12	20	166SS02	29	0
XYLEN	50	2	1	0.0083	0.0083	166SS01	NA	NA
ZN	100	3	3	12	110	166SS01	59	1

µg/g Micrograms per gram.
NA Not applicable. Background values cannot be determined for these analytes.
* Surficial soil samples collected from depth of 0.3 ft to 1 ft in soil boring 166SB01 (beneath 0.3 ft of asphalt pavement), and from ground surface to 6 inches (0.5 ft) at surface soil (SS) locations
** Surficial soil sample 166SB01 consisted of road-base material (crushed aggregate and sand).
Analyte acronyms are defined in the Acronym List.

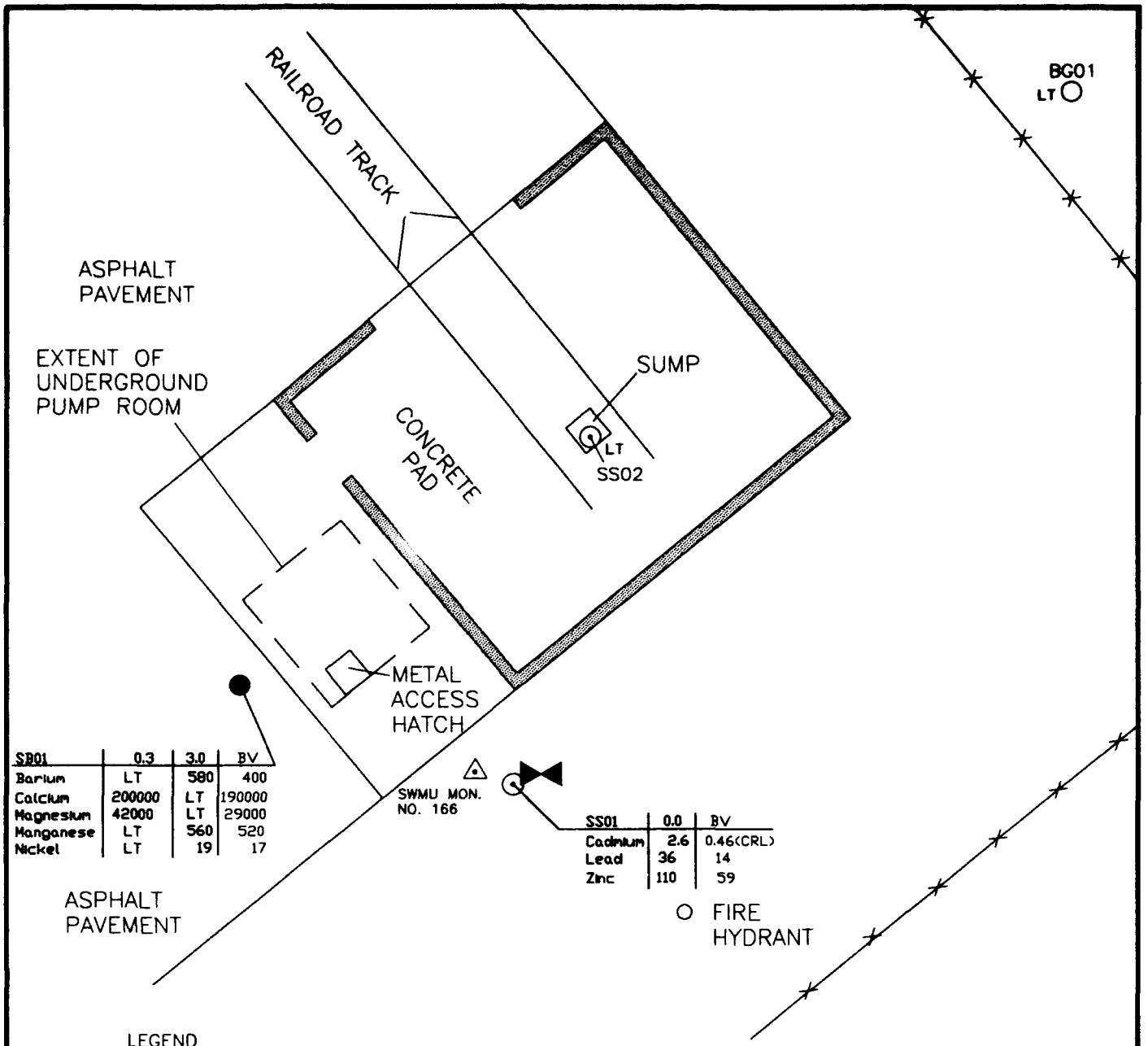
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Table 5.1-2 Summary of Constituents Detected in SWMU 166 Subsurface Soil*

Analyte	Percent Detections	Total Samples	Total Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Background Value (µg/g)	Detections> Background Value
AL	100	1	1	12,000	12,000	166SB01	19,000	0
AS	100	1	1	12	12	166SB01	13	0
BA	100	1	1	580	580	166SB01	400	1
BE	100	1	1	0.75	0.75	166SB01	1	0
CA	100	1	1	170,000	170,000	166SB01	190,000	0
CO	100	1	1	6.4	6.4	166SB01	7.9	0
CR	100	1	1	13	13	166SB01	17	0
CU	100	1	1	22	22	166SB01	25	0
FE	100	1	1	15,000	15,000	166SB01	19,000	0
K	100	1	1	3,900	3,900	166SB01	7,400	0
MG	100	1	1	18,000	18,000	166SB01	29,000	0
MN	100	1	1	560	560	166SB01	520	1
NA	100	1	1	930	930	166SB01	11,000	0
NI	100	1	1	19	19	166SB01	17	1
PB	100	1	1	6.4	6.4	166SB01	14	0
V	100	1	1	27	27	166SB01	29	0
ZN	100	1	1	52	52	166SB01	59	0

µg/g Micrograms per gram.
NA Not applicable. Background and/or RBSL values cannot be determined for these analytes.
* Subsurface soil samples collected from depths of 3 ft to 4 ft.
Analyte acronyms are defined in the Acronym List.

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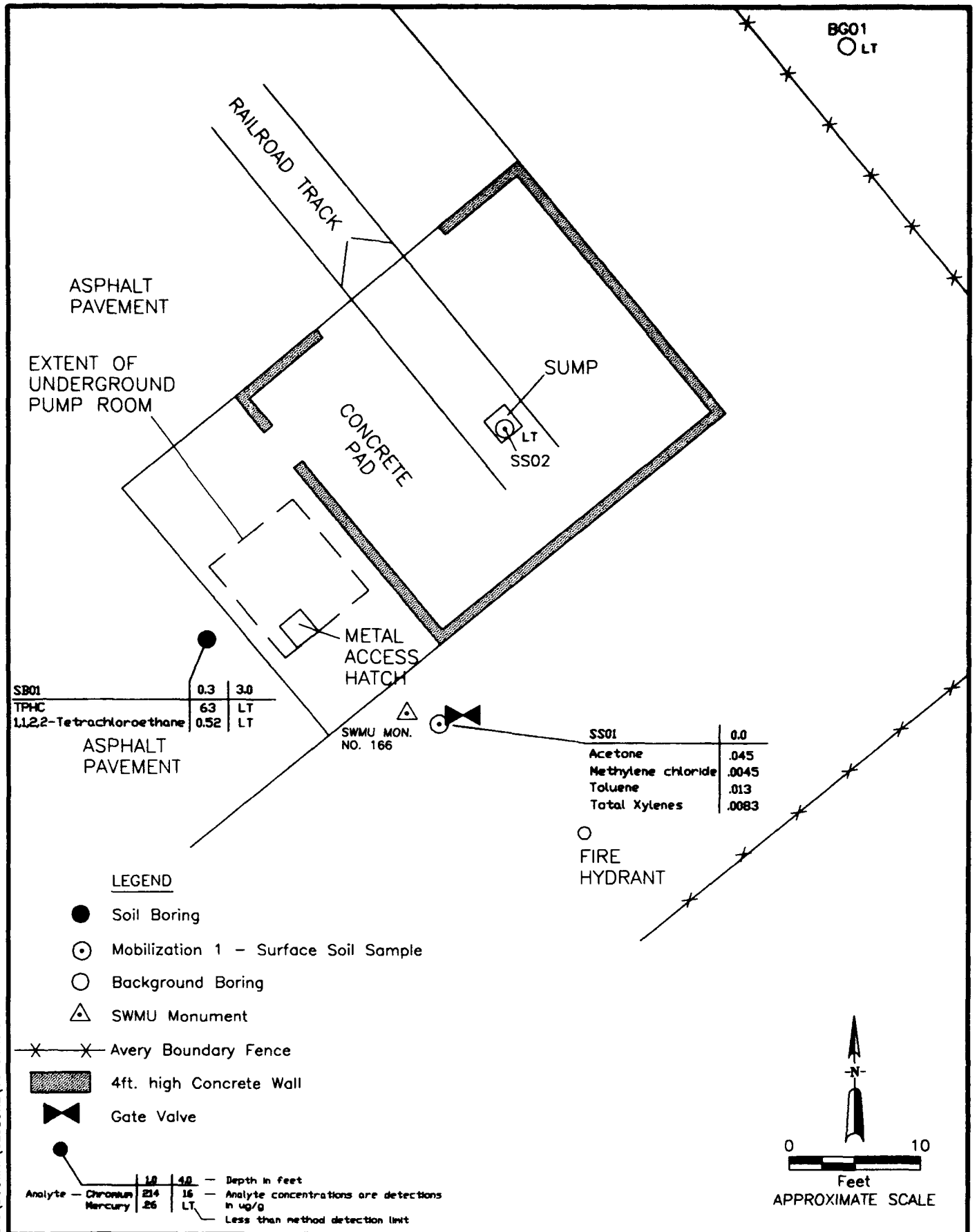
Figure 5.1–4 SWMU 166
Distribution of Inorganics in Soil

Four VOCs and TPHC were detected in soil samples from two locations at SWMU 166 (Figure 5.1-5). TPHC was detected in the surficial sample at SB01, which was collected directly beneath the asphalt pad. This detection is probably related to the overlying asphalt paving or the presence of asphalt-coated material in the sample. The four VOCs—acetone, methylene chloride, toluene, and total xylenes—were detected in ppb concentrations in surficial soil at SS01. Low concentrations of methylene chloride may have resulted from the chlorinated water in the water line adjacent to SS01, as it is formed during water chlorination. The low concentrations of acetone, toluene, and total xylenes may be due to vehicle emissions from traffic on the nearby Avery perimeter road (Figure 5.1-3a). A solvent (1,1,2,2-tetrachloroethane) was detected using the SVOC method in the sample underlying the asphalt pavement at SB01, however, it was not detected using the VOC method for which it is a target analyte. Therefore, this detection is not considered valid and was not evaluated further. The other organic compounds are not expected to persist in the soil due to aerobic biodegradation and short degradation half-lives. More information on degradation half-lives is found in Appendix M, Table M.1-1 of the Final Interim Report (EBASCO 1995a).

5.1.5 Maximum Waste Inventory

The SWMU includes a wash rack that was used to wash railcars with soapy water. SWMU 166 has also been used to store petroleum products for heavy equipment in the area, but is not known ever to have been used to treat, store, or dispose of hazardous wastes.

Since SWMU 166 was used to wash railcars carrying items that were irradiated with radioactive cobalt and later to store petroleum products, two radiation surveys were conducted and several surface and subsurface soil samples were collected and analyzed for VOCs, SVOCs, metals, cyanide, and TPHC. As indicated in Section 5.1.4, the results indicate there is no significantly contaminated soil or residue as a result of past railcar-washing operations at the unit.



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Figure 5.1-5 SWMU 166
Distribution of Organics in Soil

5.1.6 Baseline Human Health Risk Assessment

This section presents the results of the baseline human health risk assessment developed for SWMU 166. The analysis is based on the soil analytical results presented in Section 5.1.4 and was developed using the risk assessment approach described in Section 2.5. It presents a conservative evaluation of potential health risks associated with a hypothetical residential use of SWMU 166.

5.1.6.1 Identification of COPCs

Using the conservative selection criteria defined in Section 2.5 and Figure 2.5-2 the following four constituents were selected as soil COPCs for SWMU 166 (Table 5.1-3):

- Acetone
- Methylene chloride
- Toluene
- Xylenes

Toxicity profiles for these COPCs are provided in Appendix B.3.

These four VOCs were detected at very low concentrations; two of them, acetone and methylene chloride, are possible laboratory contaminants. Concentrations of inorganic constituents are generally comparable to background, but the ANOVA comparison identified three metals with concentrations that are significantly different from background concentrations. Antimony and barium were eliminated from further evaluation in the risk assessment because there is no evidence of historical use at SWMU 166. Calcium was eliminated because it is an essential nutrient. No antimony detections exceeded the UTL, and all were below the maximum and UCL95 concentrations, both equal to 8.6 µg/g, as well as the residential soil RBSL for antimony of 31 µg/g. In addition, the maximum and UCL95 concentrations of barium, 580 µg/g and 530 µg/g, respectively, are well below the residential soil RBSL for barium of 5,400 µg/g.

Table 5.1-3 Summary of Constituents Detected in SWMU 166 Soil Samples and Identification of COPCs

Analyte	Detection Frequency No. of Hits	Percent	Maximum (µg/g)	EPC 95% UCL (µg/g)	UTL (µg/g)	ANOVA COPC[1]	Essential Nutrient	Few Detections > UTL	EPC Less Than RBSL	Evidence of Historical Use	Selected as COPC[2]
Acetone	**1/3**	**33%**	**0.045**	**0.045**	--	--	**No**	--	--	--	**Yes**
Aluminum	4/4	100%	12,000	11,000	19,000	No	No	--	--	--	**No**
Antimony	2/4	50%	8.6	8.6	9.1	Yes	Yes	**No**	**Yes**	**No**	**No**
Arsenic	1	100%	12	12	13	Yes	No	**None**	**No**	**No**	**No**
Barium	4/4	100%	580	530	400	Yes	No	**Yes**	**Yes**	**No**	**No**
Beryllium	3/4	75%	0.75	0.75	1.0	No	No	--	--	--	**No**
Cadmium	1/4	25%	2.6	2.2	0.46	No	No	**Yes**	**Yes**	**No**	**No**
Calcium	4/4	100%	200,000	190,000	190,000	Yes	Yes	--	--	--	**No**
Chromium	4/4	100%	13	13	17	No	No	--	--	--	**No**
Cobalt	3/4	75%	6.4	6.4	7.9	No	No	--	--	--	**No**
Copper	4/4	100%	22	22	25	No	No	--	--	--	**No**
Iron	4/4	100%	15,000	14,000	19,000	No	Yes	--	--	--	**No**
Lead	4/4	100%	36	31	14	No	No	--	--	--	**No**
Magnesium	4/4	100%	42,000	38,000	29,000	No	Yes	--	--	--	**No**
Manganese	4/4	100%	560	520	520	No	No	--	--	--	**No**
Methylene Chloride	**1/3**	**33%**	**0.0045**	**0.0045**	--	--	**No**	--	--	--	**Yes**
Nickel	4/4	100%	19	18	17	No	Yes	--	--	--	**No**
Potassium	4/4	100%	3,900	3,900	7,400	No	Yes	--	--	--	**No**
Sodium	4/4	100%	930	930	11,000	No	Yes	--	--	--	**No**
Thallium	2/4	50%	9.5	9.5	35	No	No	--	--	--	**No**
Toluene	**1/3**	**33%**	**0.013**	**0.013**	--	--	**No**	--	--	--	**Yes**
TPHC	1/2	50%	63	63	--	--	No	--	--	--	**No**
Vanadium	4/4	100%	27	26	29	No	No	--	--	--	**No**
Xylene	**1/3**	**33%**	**0.0083**	**0.0083**	--	--	**No**	--	--	--	**Yes**
Zinc	4/4	100%	110	100	59	No	No	--	--	--	**No**

[1] SWMU 166 soil analytical results were compared with site-specific background data using the Wilcoxon rank-sum analysis of variance (ANOVA); this approach is documented in Section 2.3 and Appendix A.

[2] Soil COPCs are highlighted above (listed in bold) and were identified using the selection criteria shown in Figure 2.5-2.

-- Not applicable to organic analytes

5.1.6.2 Analysis

As required by state of Utah Administrative Rules and as described in Section 2.5, the risk assessments for the Module 2 SWMUs were developed first for hypothetical residential use (Figure 2.5-1). The first step involved deriving RBSLs for a conservative residential land-use scenario using the current RME and toxicological parameters required by UAC 315-101 or recommended by EPA guidance (Table 2.5-1). This scenario assumes unrestricted land use and represents the most conservative analysis of potential soil exposures for the Module 2 SWMUs. Consequently, if cancer risks and HIs calculated using the residential land-use RBSLs are below target risk criteria (e.g., a cancer risk of 10^{-6} and an HI of 1.0), then further risk evaluation is not warranted (Figure 2.5-1).

SWMU 166 is a 30 ft long by 25 ft wide wash rack located along the eastern perimeter of Avery. Under current site conditions, the most likely exposure scenario would involve occasional visits by a site maintenance worker. However, future industrial use of the site involving more frequent exposures is possible given its proximity to other operations at Avery. Given the industrial setting of the SWMU's location, residential use is unlikely.

5.1.6.3 Risk Characterization

Table 5.1-4 summarizes the results of the risk assessment developed for exposures to SWMU 166 surface and subsurface soil under a hypothetical residential land-use scenario. Cancer risks and noncancer HIs presented in this table were calculated using the RBSLs listed in Table 2.5-2 in accordance with the methods, equations, and assumptions outlined in Section 2.5 and Appendix B.1. The total cancer risk and HI calculated for the SWMU 166 risk evaluation are 7.4×10^{-10} and 2.4×10^{-5}, respectively. The cancer risk estimate is well below 10^{-6}, qualifying the SWMU for risk-based closure (UAC R315-101). The HI is below target HI criterion of 1.0 for noncancer endpoints.

Table 5.1-4 Results of SWMU 166 Cancer Risk and Hazard Index Calculations for the Hypothetical Residential Land-Use Scenario

Chemical of Potential Concern	Carcinogenic RBSL (µg/kg)	EPC (µg/g)	Associated Cancer Risk[1]	Noncarcinogenic RBSL (µg/g)	EPC (µg/g)	Associated HQ[2]
Acetone	--	0.045	--	4,700	0.045	9.5E-06
Methylene chloride	6	0.0045	7.4E-10	7	0.0045	1.6E-06
Toluene	--	0.013	--	490	0.013	1.3E-05
Xylenes	--	0.0083	--	320	0.0083	8.6E-08
	Total Cancer Risk:		**7.4E-10**	**Total Hazard Index (HI):**		**2.4E-05**

[1] Cancer risks were calculated using the following equation: Risk = (EPC/RBSL)*TCR, where TCR equals a target cancer risk level of 10^{-6} (or 0.000001). Table 2.5-1 summarizes the underlying exposure assumptions; Appendix B.1 documents the RBSL equations and calculations.

[2] Chemical-specific HQs were calculated by dividing the EPC by the noncarcinogenic RBSL (Section 2.5.5 and Appendix B.1).

-- Not applicable.

5.1.6.4 Summary

The results of the human health risk assessment indicate that negligible human health risks are associated with potential exposure to soil. Based on state of Utah guidelines and the decision framework illustrated in Figure 2.5-1, these findings qualify the unit for risk-based closure.

5.1.7 Ecological Risk Assessment

5.1.7.1 Ecological Conditions

The habitat surrounding SWMU 166 and the Avery compound is primarily greasewood. However, this wash rack is a very small concrete pad with four-foot concrete walls on three sides surrounded by pavement and gravel areas. There is no greasewood habitat present at this SWMU or within the Avery compound.

5.1.7.2 Evaluation of Detected Chemicals

Twenty-four chemicals, 20 inorganics and 4 organics, were detected in surficial soil samples at SWMU 166 (Table 2.6-5). Cadmium, calcium, lead, magnesium, and zinc were detected above background levels for surficial soils. However, according to ANOVA results, antimony, arsenic, barium, and calcium had concentrations data sets that were significantly different from the background concentration data sets. HQs were computed for the entire list of detected COPCs when toxicological data were available and HQs representing total and incremental risk were evaluated. Screening-level mammalian toxicological data were not available for calcium, cobalt, iron, magnesium, potassium, and sodium. Screening-level avian toxicological data were not available for acetone, beryllium, calcium, cobalt, iron, magnesium, methylene chloride, potassium, sodium, thallium, toluene, and xylene. However, most of the inorganic chemicals listed are considered to be essential nutrients and are only toxic at very high concentrations.

5.1.7.3 Evaluation of Site-Specific Information and Remedial Recommendation

The preliminary assessment for SWMU 166 showed that potential total risks were only from naturally occurring trace metals. In most instances, the ANOVA showed that these metals were detected at concentrations consistent with background concentrations, and the maximum detected

concentrations were less than the background UTL for the same metal. Additionally, because the computation of HQs in the preliminary ecological risk assessment uses many conservative assumptions (Section 2.6.2.3) to prevent the underestimation of potential risks, it is likely that only negligible risk may exist at this SWMU to the deer mouse and the horned lark and, by proxy through their conservative use, other appropriate receptors. This SWMU is not likely to provide any suitable habitat for DPG wildlife because it is covered by gravel and pavement and is located within a heavily used technical center. Due to the poor habitat and low probability of exposure of DPG wildlife to site contamination, if actually present at toxic levels, it is recommended that no corrective action related to suspected chemical contamination be performed at SWMU 166 based on risk results computed for the deer mouse and horned lark.

5.1.7.4 Ecological Risk Results for the Deer Mouse

Based only on the interpretation of HQ values previously described (Section 2.6.2.3), several detected metals would be predicted to cause risk at some level (Table 2.6-5). The computed HQs representing potential total risk from aluminum (599) and thallium (215) would indicate likely risk. Several other chemicals (antimony, 11; arsenic, 12; barium, 26) would indicate some potential total risk based on their predicted HQs, and negligible total risk was estimated for vanadium (HQ = 9). However, based on the ANOVA comparison, antimony, arsenic, barium, and calcium were above background concentrations. The total potential risk for aluminum and thallium, therefore, is likely attributable entirely to background concentrations. For the chemicals at this SWMU that had maximum concentrations greater than background, there were no HQs greater than 1 and so incremental risk was not evaluated.

5.1.7.5 Ecological Risk Results for the Horned Lark

Based only on the interpretation of HQ values previously described (Section 2.6.2.3), several detected metals would be predicted to cause risk at some level (Table 2.6-6). The computed HQs representing potential total risk from aluminum (16) and antimony (17) would indicate some potential risk. Several other metals (barium, 3; chromium, 3; lead, 8; and zinc, 2) would represent a negligible level of potential total risk. Thus, the potential total risk for aluminum and

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antimony is likely attributable entirely to background concentrations. Additionally, background concentrations of lead and zinc account for a significant portion of the maximum detected concentrations and, therefore, the background concentrations of these metals contribute substantially to the potential total risk estimate. Thus, a relatively small percentage of the potential total risk may be due to anthropogenic sources.

If the background concentration is subtracted from the maximum concentration and that value used to compute an HQ, then the HQ represents the incremental risk to the receptor from levels of suspected inorganic contamination above naturally occurring levels. Although these metals passed the ANOVA comparison, no incremental risk can be calculated because the background concentration was greater than the maximum detections.

Because the background concentrations of these metals so nearly approximate the maximum detected concentrations, the predicted incremental risk HQs are much smaller than the potential total risk HQs. The incremental risk estimates are more likely to better approximate the actual adverse impacts, if any, to be experienced by the horned lark population.

5.2 CLOSURE PERFORMANCE STANDARD

This closure plan is designed to provide for closure of SWMU 166 in a manner that will (1) protect human health and the quality of the environment; (2) control, minimize, or eliminate the escape of hazardous constituents to soil, surface water, groundwater, or the atmosphere during and after closure; and (3) minimize the need for further maintenance at the SWMU. These objectives are consistent with the requirements of UAC R315-101 that contain the following corrective action decision-making criteria to be used at DPG:

- Clean closure is allowed if the noncancer HI is less than 1.0 and the excess cancer risk is less than 1×10^{-6} for residential use.

- Site controls (or optional corrective action) are required if the excess cancer risk is greater than 1×10^{-6} for residential use and less than 1×10^{-4} for actual use and the noncancer HI is less than 1.0 for both residential and actual uses.

- Corrective action is required if the cancer risk is greater than 1×10^{-4} or the noncarcinogenic HI is greater than 1.0 for actual use.

There are no administrative rules requiring corrective action based on the results of the ecological risk assessment. Therefore, these risk results are evaluated subjectively.

In addition to the risk assessment results, the contamination assessment of the SWMU was evaluated with respect to the principle of nondegradation of the environment as required in UAC R315-101-3. This rule states that the unit is to be managed and closed in a way that the levels of contamination in groundwater, surface water, soil, and air will not increase after site management begins (during closure and post-closure care of the unit).

5.3 CLOSURE ACTIVITIES

Table 5.3-1 summarizes the risk assessment results and corrective action recommendations for closure of SWMU 166. The human health risk assessment results for the hypothetical residential land-use scenario (total cancer risk of 7.4×10^{-10} and HI of 2.4×10^{-5}) qualify this unit for risk-based closure.

The results of the preliminary ecological risk assessment showed that there are no incremental risks to representative wildlife receptors through exposure to contaminated soil. High potential total risks were computed from metal concentrations that were below the background concentrations. Therefore, corrective action based on ecological risk during closure of SWMU 166 is not necessary.

Table 5.3-1 • Corrective Action Recommendations for SWMU 166

Human Health Criteria					
Receptors	**Cancer Risk**	**Noncancer Hazard Index**	**Major COPCs in Risk Exceedance**	**Concentrations > Background or Risk-Based Cleanup Level**	**Recommendation**
Hypothetical Residents	7.4×10^{-10}	2.4×10^{-5}	None	NA	Site controls

Ecological Criteria			
Biological Receptors	**Major COPCs and Incremental Hazard Quotient**	**Maximum Conc. > Background or Cleanup Level**	**Recommendation**
Deer Mouse	None	No	No Action
Horned Lark	None	No	No Action

Explosive Risk		
Explosive Risk Level	**Items Expected**	**Recommendation**
None Expected	NA	No Action

Compliance with Nondegradation Principle		
Potential Degradation Mechanism and Medium	**Location**	**Recommendation**
None	NA	No Action

NA - Not applicable

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There is no hazardous waste present at the unit that could result in further degradation of environmental media and the detected concentration of TPHC beneath the asphalt pavement is far below the waste oil cleanup guideline of 500 ppm established by UDEQ, so no releases to the environment are expected. There is no explosive risk anticipated because no UXO are known to have been handled or disposed of at the SWMU.

5.3.1 Facility Decontamination

The potentially contaminated structures, equipment, and media areas associated with this SWMU are the concrete pad and walls, the sump, the rails, and soil. The details of decontamination activities associated with equipment and media at SWMU 166 are discussed in Sections 5.3.1.1 and 5.3.1.2, respectively.

5.3.1.1 Equipment Decontamination

The structures and equipment associated with SWMU 166 include the concrete pad and walls, the sump, and the railroad rails. Although no material samples of the concrete were analyzed, the lack of detections of organic or metal contamination in sample SS02 from the sump confirms that no hazardous waste releases occurred at the wash rack and that only non-hazardous materials were used at the unit. The lack of visible staining on the pad also confirms that releases have not occurred at SWMU 166. Because there is no planned use of the unit, no closure activities are recommended with respect to the concrete pad and associated equipment

5.3.1.2 Soil Decontamination

The risk assessment results indicate that negligible human health risks are associated with potential exposure to soil. Under a residential land-use scenario, the total cancer risk is 7.4 x 10^{-10}, and the HI is 2.4 x 10^{-5}. These results indicate that this SWMU qualifies for risk-based closure.

Because the results of the preliminary ecological risk assessment indicated that there are no incremental risks to the chosen representative wildlife receptors, remediation to reduce ecological risk is not recommended at SWMU 166.

5.4 POST-CLOSURE PLAN

Because no hazardous waste will remain at SWMU 166 when it is closed, submission of a post-closure plan is not required.

5.5 PERMIT MODIFICATION

After the public comment period ends and the Closure Plan for SWMU 166 is approved, this SWMU will be deleted from Part A and from Tables 1 and 2 in Module IV of the DPG permit.

6.0 CLOSURE PLAN FOR SWMU 170—ENGLISH VILLAGE STEAM CLEANING AREA

6.1 UNIT CHARACTERISTICS

6.1.1 SWMU Description and History

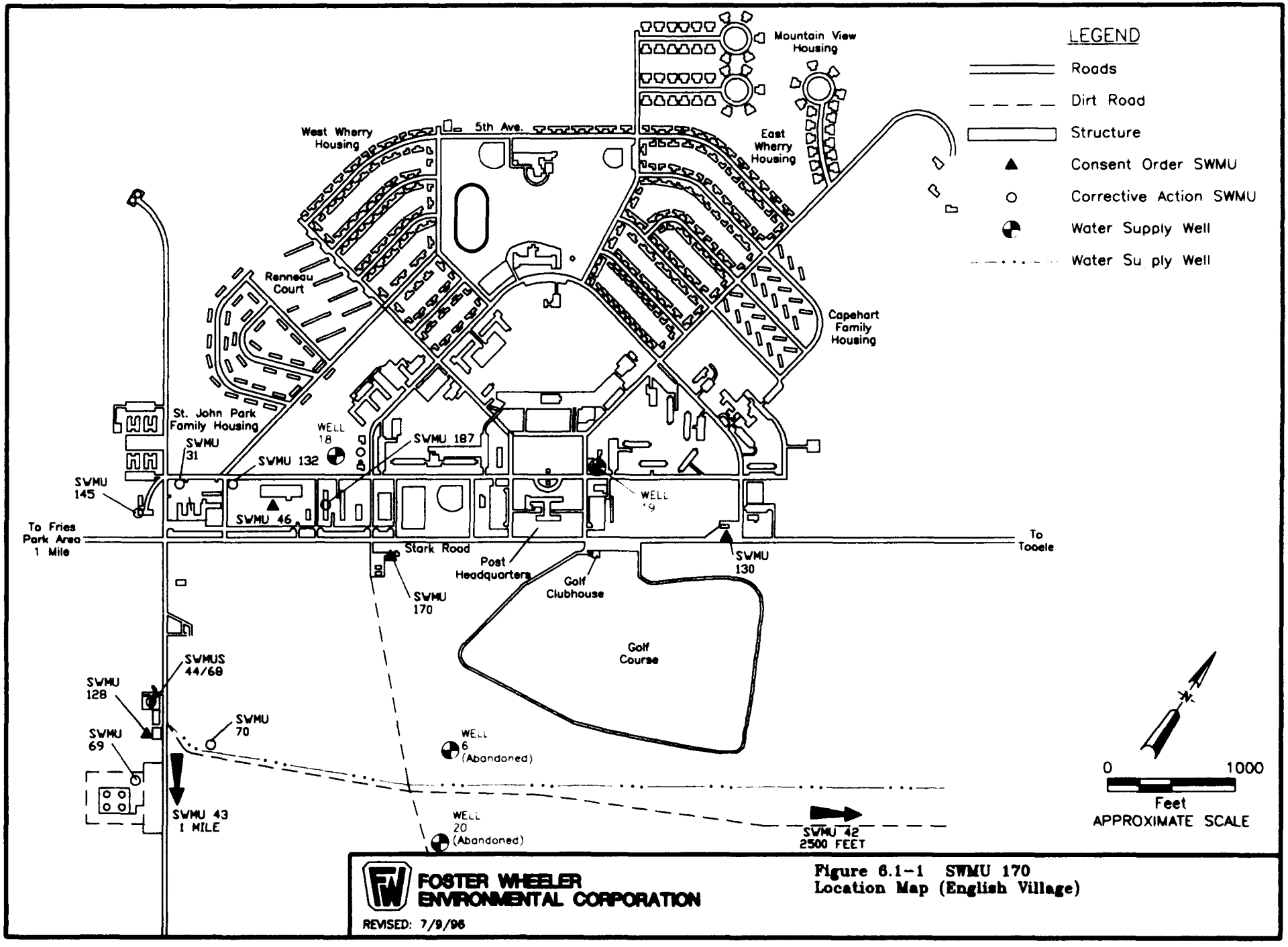
SWMU 170, known as the English Village Steam Cleaning Area, is located in the southwest portion of English Village on the south side of Stark Road between Buildings 5982 and 5983 (DPG 1984a). SWMU 170 is approximately 1,000 ft southeast of SWMU 46 and 2,000 ft northeast of SWMU 128. The unit is also located approximately 700 ft southeast of the Print Shop (Corrective Action SWMU 187) and 1,400 ft southeast of the 90-Day Hazardous Waste Holding Area (Corrective Action SWMU 132). SWMU 170 is located on a gravel pad at an approximate elevation of 4,835 ft msl (DPG 1961a, EPIC 1986) (Figure 6.1-1).

SWMU 170 is a concrete steam cleaning pad that is located between Buildings 5982 and 5983 (Figure 6.1-2). Originally, the pad was used to clean uncontaminated vehicles, but it has since been modified to include a steel frame, anchored into the concrete pad, that is used to hold dumpsters during cleaning operations (Figures 6.1-3a and 6.1-3b). Presently, SWMU 170 is used only to clean trash barrels and dumpsters from the English Village residential area.

The concrete pad is 35 ft long and 15 ft wide. A tapered 6-inch-wide curb encloses the pad on three sides. The pad slopes toward a sump at the eastern end that is connected to the sanitary sewer system at English Village (Anderson 1995). As noted during a March 1996 site inspection, the sump is actually a drain covered by a metal grate to keep large solids from entering the drain. The concrete underlying the grate slopes toward a vertical ceramic pipe that is connected to the sanitary sewer line observed in the nearby manhole. No solid or liquid wastes remain in the drain. The area west and south of the SWMU is covered with gravel. Adjacent Building 5983 is a combination boiler and pump house, and Building 5982 is a former sewage lift station located immediately to the southeast of the SWMU (EBASCO/AGEISS 1993b).

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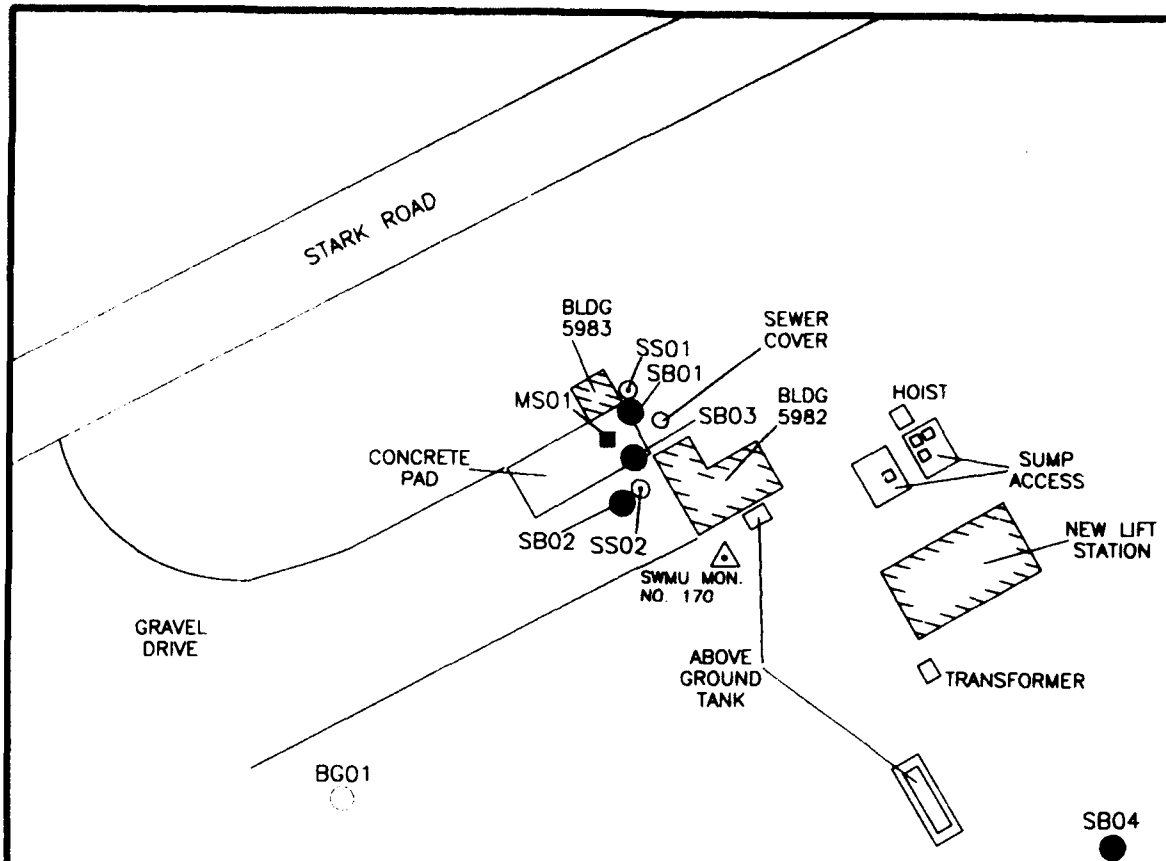
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Figure 6.1-1 SWMU 170 Location Map (English Village)



LEGEND

- Soil Boring
- Mobilization 1 – Surface Soil Sample
- Material Sample
- Background Boring
- SWMU Monument

N

0 50

Feet

APPROXIMATE SCALE

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Figure 6.1-2 SWMU 170 Sample Location Map



(A) 1991 Photograph



(B) 1995 Photograph

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Figure 6.1-3 SWMU 170
English Village Steam Cleaning Sump
(Looking North)

During a visual inspection conducted in July 1993 a ditch was noted about 4 ft east of Building 5982 that terminates in a low-lying area. A ceramic pipe ran from the lift station at Building 5982 to the ditch. This pipe is not associated with SWMU 170. Excavation in the area where the pipe entered the ditch was occurring during this inspection, and pieces of the pipe were scattered on the ground. The purpose of this excavation was apparently related to the preparation of a foundation for a new sewage lift station. It was also noted during the site visit that the concrete pad and the adjacent manhole associated with SWMU 170 were dry (EBASCO/AGEISS 1993b).

According to DPG employees, only residential trash barrels and dumpsters are currently cleaned at SWMU 170 (Anderson 1995). Wastewater generated from this activity is discharged directly to the sewer system and nothing hazardous has been stored, generated, or disposed of at this site. Consequently, it is unlikely that environmental contamination due to washing operations exists at the site (EBASCO 1992).

6.1.2 Surface Water and Groundwater

The general direction of surface drainage for the English Village area is to the northeast, toward Skull Creek, with northward drainage in Skull Valley toward the center of the Great Salt Lake Desert (Figure 2.1-5). Surface drainage in the area immediately surrounding SWMU 170 is likely influenced by manmade ditches that parallel Stark Road (DPG 1984b).

No groundwater monitoring wells have been installed at SWMU 170. Monitoring wells installed at Corrective Action SWMUs 44/68 and 69, located approximately 0.5 miles southwest of SWMU 170, encountered groundwater at depths between 80 ft and 85 ft (Parsons ES 1995). The five water supply wells in the English Village area (wells 6, 18, 19, 20, and 26) encountered fresh water in gravel deposits at depths of 80 ft to 200 ft. Groundwater beneath the English Village area occurs at depths of approximately 80 ft under unconfined conditions (USAEHA 1987). The groundwater is a part of the Skull Valley aquifer system and generally flows toward the axis of

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the valley. However, this flow pattern may be altered in the English Village area due to pumping of water supply wells (EBASCO 1990).

6.1.3 Maximum Extent of Operations

Neither hazardous nor other solid waste has been handled or disposed of at SWMU 170. Therefore, a survey plat of SWMU 170 is not required.

6.1.4 Nature and Extent of Contamination

6.1.4.1 Material Sampling Results

This section presents the results of material and soil sampling at SWMU 170. The sampling program was designed to meet the Army's objective of protecting human health and the environment during continued military use of the area.

Because of the design of the drain at SWMU 170, no samples of solid or liquid waste were collected and analyzed. However, a sample of ceramic tile from the drain system was analyzed for RCRA characteristics using toxicity characteristic leaching procedure methods. Data validation indicated all results are acceptable and 100 percent complete. Although arsenic, barium, and chloroform were detected in the sample, the concentrations were well below regulatory limits. The pipe is not a characteristic hazardous waste based on analysis for toxicity, ignitability, or corrosivity. Results of the material sample analysis are summarized in Table 6.1-1.

6.1.4.2 Soil Sampling Results

Surficial and subsurface soil samples were collected from two surface soil locations and five soil borings, including the background location, to characterize the nature and extent of soil constituents at SWMU 170. Gravels, sand, and stone fill materials were encountered to a depth of 2.5 ft south of the pad. The soil is probably fill or a mixture of fill and native soil here and east of the pad. Silty clay native soil was encountered in SB04 southeast of the pad. Samples were analyzed for metals, VOCs, SVOCs, PCBs, and pesticides.

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Table 6.1-1 Summary of SWMU 170 Material Sample Results

Material Sample	Location	Physical State	RCRA Characteristic	Analytical Results[1]	Regulatory Level[1]
MS01	Sump (Clay Pipe)	Solid Waste	Toxicity-As	130 µg/L	5,000 µg/L
			Toxicity-Ba	260 µg/L	100,000 µg/L
			Toxicity-CHCL3	2.50 µg/L	6,000 µg/L
			Ignitability	>60°C	<60°C
			Corrosivity-pH	8.9	≤2 or ≥12.5

Notes:[1] Hazardous waste designations are based on criteria specified in 40 CFR 261 Subpart C. These criteria define characteristics of ignitability (D001) as flashpoint < 60° C; corrosivity (D002) as an aqueous sample with pH ≤ 2 or ≥ 12.5, or a liquid which corrodes steel at a rate > 6.35 mm/yr; reactivity (D003) as cyanide > 250 mg/kg or sulfide > 500 mg/kg; and toxicity (D004-43), for which regulatory levels and hazardous waste numbers are listed in Table 1 of Subpart C (Part 261.24). Some analytical results reflected in this table are total concentrations and regulatory levels are based on results of toxicity characteristic leaching procedure. For solid materials, a comparison of analytical results to regulatory levels can be made by dividing the total result by a factor of 20 (40 CFR 261, Appendix II). Liquid results can be compared directly.

Data validation resulted in the rejection of selenium results for one sample. No SWMU 170 soil sample results were rejected because of contamination in the field or laboratory method blank. The constituents detected in SWMU 170 surficial and subsurface soil samples are summarized in Tables 6.1-2 and 6.1-3, respectively. Further discussion of the sampling results can be found in Section 37.2 of the final Interim Report (EBASCO 1995a).

Six metals—chromium, lead, mercury, sodium, vanadium, and zinc—were detected at concentrations slightly above background in SWMU 170 soil at two locations (Figure 6.1-4). The results of a nonparametric ANOVA of the soil samples showed that the distributions of only three of these metals (chromium, lead, and zinc) are significantly different from background at SWMU 170 (Table 2.3-2). These inorganic constituents will persist in the soil, generally adsorbing to the soil particles.

Five organic compounds—acetone, methylene chloride, PPDDT, toluene, and total xylenes—were detected in soil samples at three locations near the concrete pad at SWMU 170 (Figure 6.1-5). The detections of methylene chloride may be a result of using chlorinated water at the pad, as it is formed during water chlorination (Micromedex 1996). The low concentrations of toluene and total xylenes are most likely a result of vehicle emissions from the trucks transporting the dumpsters to the cleaning pad. The low concentrations of PPDDT may be due to application of pesticides around the SWMU, as it was used until the early 1970s throughout English Village. Two other compounds—endrin ketone and endosulfan sulfate—were tentatively identified using the SVOC method, but were not confirmed using EPA Method 8080 for detection of pesticides; therefore, these two detections were not considered valid or evaluated further. Except for PPDDT, the organic constituents will not persist in the soil due to aerobic biodegradation and short degradation half-lives. Further discussion of the fate and transport of these organic

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Table 6.1-2 Summary of Constituents Detected in SWMU 170 Surficial Soil*

Analyte	Percent Detections	Total Samples	Total Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Background Value (µg/g)	Detections> Background Value
ACET	100	2	2	0.0077	0.01	170SS01	NA	NA
AL	100	2	2	6,900	9,100	170SS01	19,000	0
AS	100	2	2	6.7	8.3	170SS01	13	0
BA	100	2	2	190	320	170SS01	400	0
BE	50	2	1	0.68	0.68	170SS02	1.0	0
CA	100	2	2	96,000	130,000	170SS01	190,000	0
CO	100	2	2	3.6	4.2	170SS01	7.9	0
CR	100	2	2	13	27	170SS01	17	1
CU	100	2	2	14	18	170SS02	25	0
FE	100	2	2	10,000	12,000	170SS01	19,000	0
HG	50	2	1	0.063	0.063	170SS02	0.050	1
K	100	2	2	3,200	4,200	170SS01	7,400	0
MEC6H5	50	2	1	0.003	0.003	170SS02	NA	NA
MG	100	2	2	12,000	16,000	170SS02	29,000	0
MN	100	2	2	260	360	170SS01	520	0
NA	100	2	2	2,100	12,000	170SS01	11,000	1
NI	100	2	2	9.3	10	170SS01	17	0
PB	100	2	2	11	29	170SS02	14	1
PPDDT	100	2	2	0.012	0.018	170SS02	NA	NA
V	100	2	2	19	35	170SS01	29	1
XYLEN	100	2	2	0.0017	0.0036	170SS02	NA	NA
ZN	100	2	2	50	75	170SS02	59	1

µg/g Micrograms per gram.
NA Not applicable. Background values cannot be determined for these analytes.
* Surficial soil samples collected from ground surface to depth of 1 ft in soil borings, and from ground surface to depth of 6 inches (0.5 ft) in surface soil (SS) locations. Analyte acronyms are defined in the Acronym List.

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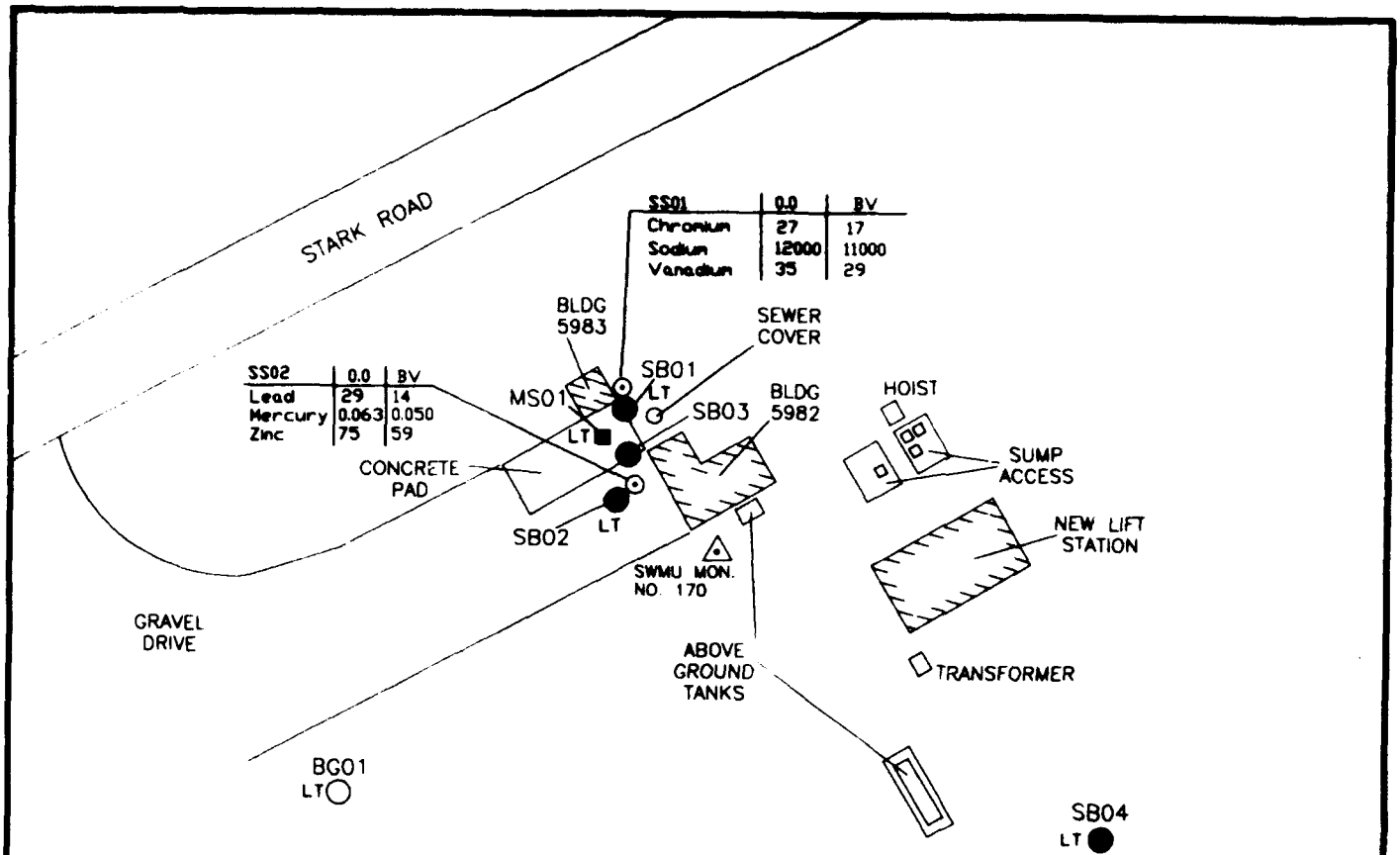
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Table 6.1-3 Summary of Constituents Detected in SWMU 170 Subsurface Soil*

Analyte	Percent Detections	Total Samples	Total Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Background Value (µg/g)	Detections> Background Value
AL	100	4	4	8,600	16,000	170SB04	19,000	0
AS	100	4	4	4.5	6.3	170SB04	13	0
BA	100	4	4	190	310	170SB01	400	0
BE	50	4	2	0.64	0.64	170SB03	1.0	0
CA	100	4	4	78,000	140,000	170SB01	190,000	0
CH2CL2	25	4	1	0.0069	0.0069	170SB03	NA	NA
CO	100	4	4	3.2	6.6	170SB04	7.9	0
CR	100	4	4	11	16	170SB04	17	0
CU	100	4	4	11	18	170SB04	25	0
FE	100	4	4	9,500	16,000	170SB04	19,000	0
K	100	4	4	3,700	6,500	170SB04	7,400	0
MG	100	4	4	9,600	12,000	170SB04	29,000	0
MN	100	4	4	230	370	170SB04	520	0
NA	100	4	4	870	2,600	170SB01	11,000	0
NI	100	4	4	8.4	16	170SB04	17	0
PB	100	4	4	7	12	170SB04	14	0
TL	25	4	1	21	21	170SB03	35	0
V	100	4	4	16	27	170SB04	29	0
ZN	100	4	4	33	55	170SB04	59	0

µg/g Micrograms per gram.
NA Not applicable. Background and/or RBSL values cannot be determined for these analytes.
* Subsurface soil samples collected from depth of 3 ft to 4 ft.
Analyte acronyms are defined in the Acronym List.
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LEGEND

- Soil Boring
- Mobilization 1 – Surface Soil Sample
- Material Sample
- Background Boring
- SWMU Monument

Depth in Feet

Analyte		1.0	4.0	BV	
	Chromium	214	20	19	— Background Value
	Mercury	.26	LT	.099	— Analyte concentrations above background value in ug/g

— Less than background value

N

0 50

Feet

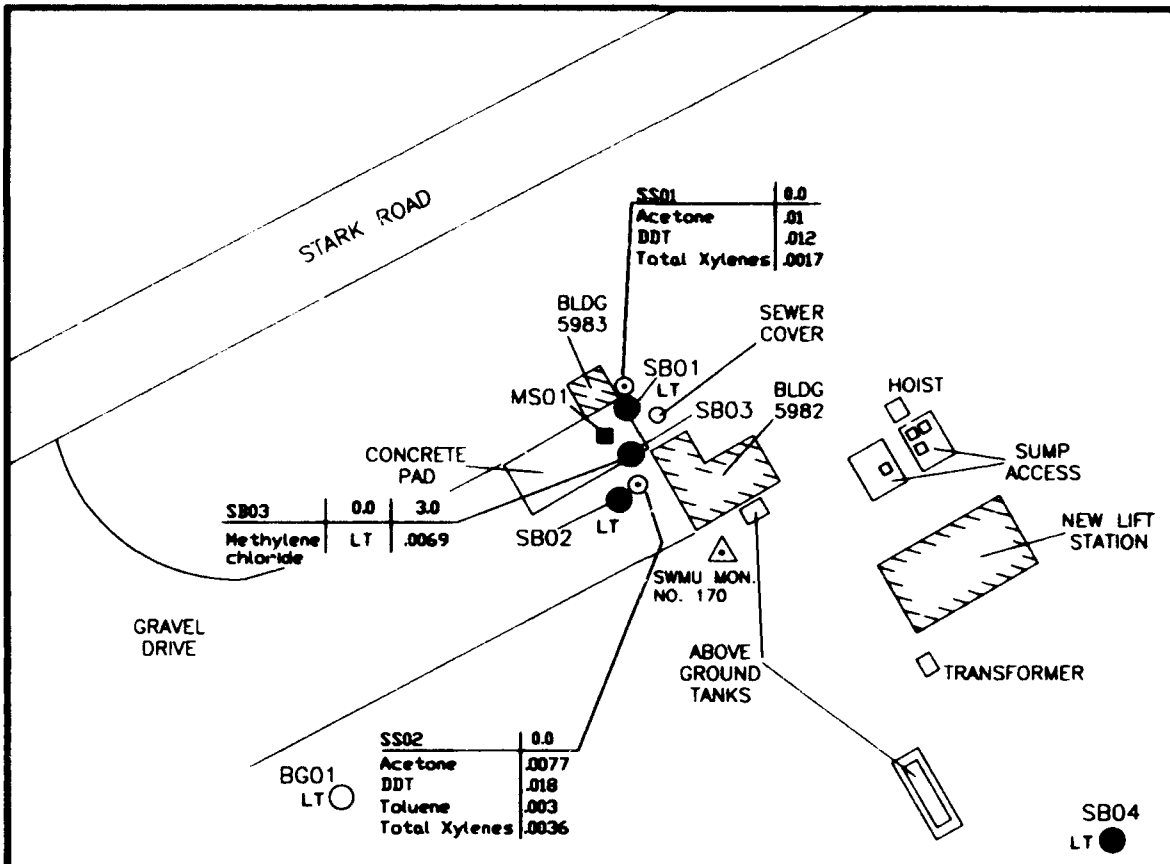
APPROXIMATE SCALE

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Figure 6.1-4 SWMU 170
Distribution of Inorganics in Soil

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LEGEND

- Soil Boring
- Mobilization 1 – Surface Soil Sample
- Material Sample
- Background Boring
- SWMU Monument

Analyte – Chromium | 1.0 | 4.0 — Depth in feet
Chromium | 214 | 16 — Analyte concentrations are detections
Mercury | .26 | LT — in ug/g
Less than method detection limit

N

0 50

Feet

APPROXIMATE SCALE

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Figure 6.1-5 SWMU 170
Distribution of Organics in Soil

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compounds and additional information on the degradation half-lives are provided in Section 37.3.1 and Appendix M, Table M.1-1, respectively, of the final Interim Report (EBASCO 1995a).

6.1.5 Maximum Waste Inventory

There is no potential for hazardous wastes, including residues, at SWMU 170 because the unit is not known to have ever been used to treat, store, or dispose of hazardous wastes. The wastes generated from the cleaning activities are discharged directly to the sewer system. The current cleaning operation at the unit, i.e., steam cleaning residential trash barrels and dumpsters is not considered a hazardous waste treatment, storage, or disposal activity. This operation is expected to continue into the foreseeable future.

A material sample and several soil samples were collected from the pipe and the areas around the concrete pad, respectively, to identify any contamination at the unit. The material sample was analyzed for RCRA waste characteristics and the soil samples for VOC, SVOC, pesticide, and metal analytes. As summarized in Section 6.1.4, no contaminated soil or residues are present that can be related to operations at the SWMU. The material sample collected from the ceramic pipe that drains the wastes generated from the steam cleaning operation at SWMU 170 did not show any hazardous characteristics.

6.1.6 Baseline Human Health Risk Assessment

This section presents the results of the baseline human health risk assessment developed for SWMU 170. The analysis is based on the soil analytical results presented in Section 6.1.4 and was developed using the risk assessment approach described in Section 2.5. It presents a conservative evaluation of potential human health risks associated with a hypothetical residential use of SWMU 170.

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6.1.6.1 Identification of COPCs

As discussed in the preceding sections, the results of soil sampling do not indicate any significant contamination resulting from washing operations conducted at the site. Concentrations of metals are generally comparable to background. The ANOVA comparison identified three metals, however, that have concentration data sets that are significantly different from the background concentration data set. Zinc was eliminated because it is considered an essential nutrient, there is no historical use of zinc at this SWMU, and all zinc detections were well below the residential use RBSL. Lead was not evaluated because the maximum detection of lead, 29 µg/g, is well below the interim soil lead level set by the EPA. Chromium was also excluded from further evaluation, because there is no expected historical use of chromium at this SWMU, and the maximum detected concentration (27 µg/g) is well below its residential-use RBSL (390 µg/g). Four VOCs were detected at very low concentrations. PPDDT was detected in two surficial soil samples collected near the concrete pad during Mobilization 1. Its presence in these samples could be attributable to previous application of pesticides throughout English Village.

Using the selection criteria defined in Section 2.5 and Figure 2.5-2, the following five constituents were selected as soil COPCs for SWMU 170 (Table 6.1-4):

- Acetone
- PPDDT
- Methylene chloride
- Toluene
- Xylenes

Toxicity profiles for these COPCs are provided in Appendix B.3.

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Table 6.1-4 Summary of Constituents Detected in SWMU 170 Soil Samples and Identification of COPCs

Analyte	Detection Frequency		Maximum	EPC 95%	UTL	ANOVA	Essential	Few Detections	EPC Less	Evidence of	Selected
	No. of Hits	Percent	(µg/g)	UCL (µg/g)	(µg/g)	COPC[1]	Nutrient?	> UTL	Than RBSL	Historical Use	as COPC?[2]
Acetone	**2/6**	**33%**	**0.01**	**0.01**	--	--	**No**	--	--	--	**Yes**
Aluminum	6/6	100%	16,000	12,000	19,000	No	No	--	--	--	No
Arsenic	6/6	100%	8.3	7.3	13.0	No	No	--	--	--	No
Barium	6/6	100%	320	300	400	No	No	--	--	--	No
Beryllium	3/6	50%	0.68	0.63	1.0	No	No	--	--	--	No
Calcium	6/6	100%	140,000	130,000	190,000	No	Yes	--	--	--	No
Chromium	6/6	100%	27	20	17	Yes	No	Yes	Yes	No	No
Cobalt	6/6	100%	6.6	5.4	7.9	No	No	--	--	--	No
Copper	6/6	100%	18	17	25	No	No	--	--	--	No
Iron	6/6	100%	16,000	13,000	19,000	No	Yes	--	--	--	No
Lead	6/6	100%	29	20	14	Yes	No	Yes	Yes	No	No
Magnesium	6/6	100%	16,000	14,000	29,000	No	Yes	--	--	--	No
Manganese	6/6	100%	370	340	520	No	No	--	--	--	No
Mercury	1/6	17%	0.063	0.076	0.05	No	No	--	--	--	No
Methylene Chloride	**1/6**	**17%**	**0.007**	**0.007**	--	--	**No**	--	--	--	**Yes**
Nickel	6/6	100%	16	13	17	No	No	--	--	--	No
Potassium	6/6	100%	6,500	5,200	7,400	No	Yes	--	--	--	No
PPDDT	**2/6**	**33%**	**0.018**	**0.012**	--	--	**No**	--	--	--	**Yes**
Sodium	6/6	100%	12,000	6,900	11,000	No	Yes	--	--	--	No
Thallium	1/6	17%	21	13	35	No	No	--	--	--	No
Toluene	**1/6**	**17%**	**0.003**	**0.002**	--	--	**No**	--	--	--	**Yes**
Vanadium	6/6	100%	35	29	29	No	No	--	--	--	No
Xylene	**2/6**	**33%**	**0.0036**	**0.003**	--	--	**No**	--	--	--	**Yes**
Zinc	6/6	100%	75	61	59	Yes	No	Yes	Yes	No	No

[1] SWMU 170 soil analytical results were compared with site-specific background data using the Wilcoxon rank-sum analysis of variance (ANOVA); this approach is documented in Section 2.3 and Appendix A.

[2] Soil COPCs are highlighted above (listed in bold) and were identified using the selection criteria shown in Figure 2.5-2.

-- Not applicable to organic analytes

6.1.6.2 Analysi

As described in Section 2.5, the risk assessments for the Module 2 SWMUs were developed using a streamlined approach (Figure 2.5-1). The initial assessment involved deriving RBSLs for a conservative residential land-use scenario using the current RME and toxicological parameters required by UAC 315-101 or recommended by EPA guidance (Table 2.5-1). This scenario assumes unrestricted land use and represents the most conservative analysis of potential soil exposures for the Module 2 SWMUs. Consequently, if cancer risks and HIs calculated using the residential land-use RBSLs are below target risk criteria (i.e., a cancer risk of 10^{-6} and an HI of 1.0), then further risk evaluation is not warranted (Figure 2.5-1).

For current site conditions, exposure (e.g., by a site worker) is expected to be negligible given that the SWMU is paved and most of the surrounding area is covered with gravel. Future residential use of the SWMU and surrounding area is possible. However, its small size (approximately 500 square feet) probably does not constitute a reasonable area over which to average human exposures. Consequently, the residential land-use analysis applied in the SWMU 170 baseline risk assessment is considered conservative.

6.1.6.3 Risk Characterization

Table 6.1-5 summarizes the results of the risk assessment developed for future exposures to SWMU 170 surface and subsurface soil under a hypothetical residential land-use scenario. Cancer risks and noncancer HIs presented in this table were calculated using the RBSLs listed in Table 2.5-2 in accordance with the methods, equations, and assumptions outlined in Section 2.5 and Appendix B.1. The total cancer risk and HI calculated for the conservative SWMU 170 risk evaluation are 1.5×10^{-8} and 5.2×10^{-4}, respectively. Both values are within state of Utah criteria for risk-based closure (cancer risk of 10^{-6} and HI of 1.0).

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Table 6.1-5 Results of SWMU 170 Cancer Risk And Hazard Index Calculations for the Hypothetical Residential Land-Use Scenario

Chemical of Potential Concern	Carcinogenic RBSL* (µg/g)	EPC (µg/g)	Associated Cancer Risk[1]	Noncarcinogenic RBSL* (µg/g)	EPC (µg/g)	Associated HQ[2]
ACET	--	0.01		4,700	0.01	2.1E-06
PPDDT	0.89	0.012	1.4E-08	24	0.012	5.1E-04
CH2CL2	6	0.007	1.1E-09	7	0.007	2.4E-06
MEC6H5	--	0.002		490	0.002	2.0E-06
XYLEN	--	0.003		320	0.003	3.1E-08
	Total Cancer Risk:		**1.5E-08**	**Total Hazard Index (HI):**		**5.2E-04**

[1] Cancer risks were calculated using the following equation: Risk = (EPC/RBSL)*TCR, where TCR equals a target cancer risk level of 10^{-6} (or 0.000001). Table 2.5-1 summarizes the underlying exposure assumptions; Appendix B.1 documents the RBSL equations and calculations.

[2] Chemical-specific HQs were calculated by dividing the EPC by the noncarcinogenic RBSL (Section 2.5.5 and Appendix B.1).

* RBSL for residential land-use scenario (Table 2.5-2 and Appendix B.1)

-- Endpoint not applicable

Analyte acronyms are defined in the Acronym List.

6.1.6.4 Summary

The results of the human health risk assessment indicate that the risks posed to human receptors at SWMU 170 are negligible for hypothetical residential land uses. Risks associated with current site conditions (corresponding to less frequent human exposures) or a future industrial land-use scenario would be lower than those estimated for residential uses (Table 6.1-5), and therefore were not calculated. Based on state of Utah guidelines and the decision framework illustrated in Figure 2.5-1, these findings qualify this unit for risk-based closure.

6.1.7 Ecological Risk Assessment

6.1.7.1 Ecological Conditions

The habitat surrounding SWMU 170 is characterized as a developed area composed mostly of ornamental trees and mowed lawns. The habitat alteration in the English Village area is extensive, non-native, and covers many acres. These changes include the planting of exotic grasses for lawns and the nearby golf course, as well as the planting of trees and ornamental shrubs in English Village. The area directly south consists of bare ground and thick sagebrush and annual grassland vegetation.

6.1.7.2 Evaluation of Detected Chemicals

Twenty-one chemicals, 18 inorganic and 3 organic, were detected in surficial soil samples at SWMU 170 (Table 2.6-7). Chromium, lead, and zinc were above background levels according to the ANOVA comparison. HQs were computed for the entire list of detected chemicals when toxicological data were available and HQs representing total and incremental risk were evaluated. Screening-level mammalian toxicological data were not available for calcium, cobalt, iron, magnesium, potassium, and sodium. Screening-level avian toxicological data were not available for acetone, beryllium, calcium, cobalt, iron, magnesium, potassium, sodium, toluene, and xylene. However, almost all of the inorganic chemicals are considered to be essential nutrients and are only toxic at very high concentrations. In addition, a conservative BAF derived from mink liver (Wren *et al.*, 1987) was applied to the preliminary HQ (0.004) for mercury for deer mouse to

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account for its potentially bioaccumulative properties, even though the maximum detected concentration of mercury (0.063 μg/g) was below the background concentration (0.073 μg/g). The final mercury HQ of 0.1 is based on the total risk from mercury to the deer mouse (Table 2.6-7). Although it is recognized that some forms of mercury may bioaccumulate under certain conditions, an avian BAF was not found in the literature that could be applied in a conservative fashion.

6.1.7.3 Evaluation of Site-Specific Information and Remedial Recommendation

The preliminary assessment for SWMU 170 showed that potential total risks were only from naturally occurring trace metals. In many instances, the ANOVA showed that these metals were detected at concentrations consistent with background concentrations, and the maximum detected concentrations were less than the background UTLs for those metals. This comparison demonstrates that judgments on adverse impacts to receptors, and the remedial actions necessary to mitigate these perceived impacts, should be based on the increment of risk associated with concentrations above background. Additionally, because the computation of HQs in the preliminary ecological risk assessment uses many conservative assumptions (Section 2.6.2.3) to prevent the underestimation of potential risks, the true incremental risk estimates may actually be less than unity, indicating that there is negligible risk at this SWMU to the deer mouse and the horned lark and, by proxy through their conservative use, other receptors.

This SWMU is located in English Village, which is mostly paved or developed with little native habitat remaining. Also, this area supports a high level of human activity. Due to the absence of suitable habitat within the SWMU itself, it is not likely the deer mouse or horned lark would be exposed to the suspected contamination. As long as this SWMU remains in use, exposure to soil contaminants is not likely to occur. Therefore, although HQs were calculated as a measure of potential risk if wildlife exposure were to occur, it is recommended that no corrective action related to suspected chemical contamination be performed at SWMU 170 based on these risk results.

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6.1.7.4 Ecological Risk Results for the Deer Mouse

Based only on interpretation of the HQ values previously described (Section 2.6.2.3), several detected metals would be predicted to cause risk at some level (Table 2.6-7). The computed HQ representing total potential risk from aluminum (736) would indicate likely risk. Several other chemicals (barium, 27; vanadium, 15) would indicate some potential total risk based on their predicted HQs, and negligible total risk was estimated for arsenic (HQ = 10). However, based on the ANOVA comparison, only chromium, lead, and zinc concentrations were above background concentrations and the HQs for these three metals were less than 1 so there are no incremental risks. Thus, a relatively small percentage of the total potential risk may be due to anthropogenic sources.

6.1.7.5 Ecological Risk Results for the Horned Lark

Based only on the interpretation of HQ values previously described (Section 2.6.2.3), several detected metals would be predicted to cause risk at some level (Table 2.6-8). The computed HQ representing potential total risk from aluminum (20) and mercury (53) would indicate some potential risk. Several other chemicals (barium, 4; chromium, 7; lead, 6; vanadium, 1; and zinc, 1) would represent a negligible level of potential total risk. However, the maximum soil concentrations detected for aluminum, barium, and mercury were lower than the background soil concentrations, and the ANOVA comparison showed these metals were detected at concentrations that are consistent with background concentrations. Thus, the potential total risk for these metals is attributable entirely to background concentrations. Additionally, background concentrations of chromium and zinc nearly approximate the maximum detected concentrations. Therefore, a relatively small percentage of the potential total risk may be due to anthropogenic sources.

If the background concentration is subtracted from the maximum concentration and that value used to compute an HQ, then the HQ represents the incremental risk to the receptor from levels of suspected inorganic contamination above naturally occurring levels. An examination follows

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of those metals with concentrationed data sets above the background concentration data set according to the ANOVA comparison.

An HQ of 7 was calculated from the maximum detected chromium concentration. The chromium background soil concentration (17 μg/g) is equal to 63 percent of the maximum detected soil concentration (27 μg/g). Incremental risk, thus, represents an HQ of 3 to the horned lark from chromium (i.e., 7 minus 63 percent equals 3).

An HQ of 6 was calculated from the maximum detected lead concentration. The lead background soil concentration (14 μg/g) is equal to 48 percent of the maximum detected soil concentration (29 μg/g). Incremental risk represents an HQ of 3 to the horned lark from lead.

An HQ of 1 was calculated from the maximum detected zinc concentration. The zinc background soil concentration (59 μg/g) is equal to 79 percent of the maximum detected soil concentration (75 μg/g). Incremental risk represents an HQ of less than 1 to the horned lark from zinc.

Because the maximum detected concentrations nearly approximate the background concentrations of these metals, the predicted incremental HQs are much smaller than the total risk HQs. The incremental risk estimates are more likely to better approximate the actual adverse impacts, if any, to be experienced by the horned lark population.

6.2 CLOSURE PERFORMANCE STANDARD

This closure plan is designed to provide for closure of SWMU 170 in a manner that will (1) protect human health and the quality of the environment; (2) control, minimize, or eliminate the escape of hazardous constituents to soil, surface water, groundwater, or the atmosphere during and after closure; and (3) minimize the need for further maintenance at the SWMU. These objectives are consistent with the requirements of UAC R315-101 that contain the following corrective action decision-making criteria to be used at DPG:

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- Clean closure is allowed if the noncancer HI is less than 1.0 and the excess cancer risk is less than 1×10^{-6} for residential use.

- Site controls (or optional corrective action) are required if the excess cancer risk is greater than 1×10^{-6} for residential use and less than 1×10^{-4} for actual use and the noncancer HI is less than 1.0 for both residential and actual uses.

- Corrective action is required if the cancer risk is greater than 1×10^{-4} or the noncarcinogenic HI is greater than 1.0 for actual use.

There are no administrative rules requiring corrective action based on the results of the ecological risk assessment. Therefore, these risk results are evaluated subjectively.

In addition to the risk assessment results, the contamination assessment of the SWMU was evaluated with respect to the principle of nondegradation of the environment as required in state of UAC R315-101-3. This rule states that the unit is to be managed and closed in a way that the levels of contamination in groundwater, surface water, soil, and air will not increase after site management begins (during closure and post-closure care of the unit).

6.3 CLOSURE ACTIVITIES

Table 6.3-1 summarizes the risk assessment results and corrective action recommendations for closure of SWMU 170. The risk assessment results indicated that insignificant human health risks are associated with potential exposure to soil. Under a hypothetical residential land-use scenario, the total cancer risk is 1.5×10^{-8} and the HI is 5.2×10^{-4}. These results indicate the SWMU qualifies for risk-based closure. The results of the preliminary ecological risk assessment showed that the predicted incremental HQs are less than 10 for each COPC and receptor and that there is a lack of suitable habitat at this SWMU. Additionally, this SWMU is in a developed area and is under continual and current use by the Army.

Considering these risk assessment results, and since there is no hazardous waste at the unit, no UXO or other explosive hazard, and no contamination that could result in further degradation of

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Table 6.3-1 • Corrective Action Recommendations for SWMU 170

Human Health Criteria					
Receptors	**Cancer Risk**	**Noncancer Hazard Index**	**Major COPCs in Risk Exceedance**	**Concentrations > Background or Risk-Based Cleanup Level**	**Recommendation**
Hypothetical Residents	1.5×10^{-8}	5.2×10^{-4}	None	NA	No Action

Ecological Criteria			
Biological Receptors	**Major COPCs and Incremental Hazard Quotient**	**Concentrations > Background or Cleanup Level**	**Recommendation**
Deer Mouse	None	Yes	No Action
Horned Lark	Chromium, 3; Lead, 3	Yes	No Action

Explosive Risk		
Explosive Risk Level	**Items Expected**	**Recommendation**
None Expected	NA	No Action

Compliance with Nondegradation Principle		
Potential Degradation Mechanism and Medium	**Location**	**Recommendation**
None	NA	No Action

NA - Not applicable

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environmental media, corrective action is not required at SWMU 170. Furthermore, since there is no planned future use of the SWMU, to manage hazardous waste, no closure activities are required; therefore, no schedule of closure activities or certification of closure is required.

6.4 POST-CLOSURE PLAN

Because no hazardous waste will remain in SWMU 170 when it is closed, submission of a post-closure plan is not required.

6.5 PERMIT MODIFICATION

After the public comment period ends and the Closure Plan for SWMU 170 is approved, this SWMU will be deleted from Part A and from Tables 1 and 2 in Module IV of the DPG permit. Deletion of this unit from the permit is considered a Class 3 permit modification and may be deferred until the Phase II RFI is complete.

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7.0 CONCLUSIONS

7.1 SWMU 20—CAMELS BACK RIDGE LANDFILL

SWMU 20, which is located on the northwest side of Camels Back Ridge, consists of two low ridges and adjacent shallow trenches (west area or SWMU 20-1) and one small pit with an adjacent mound (east area or SWMU 20-2). The trenches and pit were originally believed to have been used for disposal of solid or hazardous wastes. However, further research showed that the two trenches were used only to bury instrumentation cables for the adjacent Explosive Test Shield Facility, and the small pit was an exploratory excavation for borrow gravel. No solid or hazardous waste was disposed in any of these features.

Nineteen soil samples collected from SWMU 20 contained six metals above background and three detected organic compounds, including toluene, methylene chloride, and IMPA at low concentrations. Of the six metals above background, none were retained as COPCs. The preliminary ecological risk assessment evaluated all detected chemicals at SWMU 20. The contamination assessment and risk assessment evaluating these COPCs resulted in the following conclusions:

- Human health risks qualify the SWMU for risk-based closure.
- A preliminary ecological risk assessment predicted negligible incremental risk associated with potential exposure of ecological receptors to SWMU contaminants.
  - Potential incremental risk from barium to the deer mouse was identified.
  - No significant incremental risk to the horned lark was identified.
  - This SWMU is currently inactive, does not serve as a wildlife attractant, and occupies a relatively small area within an otherwise intact and healthy community of greasewood and sagebrush.
- No contamination was detected that could be a source of environmental degradation.
- No hazardous wastes or explosive ordnance are stored at the unit.
- No continuing or future waste management use of the unit is planned.

No closure activities, closure schedule, closure certification, or post-closure care or monitoring are required at this unit. SWMU 20 should be considered closed upon approval of this closure plan module by UDEQ, Division of Solid and Hazardous Waste.

7.2 SWMU 164—AVERY WASH RACK NO. 1

This wash rack, which is located in the north-central part of Avery, was used during the 1980s for cleaning uncontaminated vehicles using water. The SWMU 164 wash rack is constructed of metal landing mat and railroad rails and is surrounded by gravel. Any contaminant releases at this SWMU would be limited to traces of vehicle fluids or lubricants.

Seven soil samples collected from this SWMU contained nine metals above background and six detected organic compounds, including toluene, xylenes, acetone, phthalates, methylene chloride, and TPHC at low concentrations. Of the nine metals above background, none were retained as COPCs. The ecological risk assessment evaluated all detected chemicals at this SWMU. The ecological risk assessment evaluated all detected chemicals at this SWMU. The contamination assessment and risk assessment evaluating these COPCs resulted in the following conclusions:

- Human health risks qualify the SWMU for risk-based closure.
- A preliminary ecological risk assessment showed no significant incremental risk to wildlife from SWMU contaminants because incremental risk calculations were low, and exposure of wildlife is unlikely.
  - No significant incremental risk to the deer mouse was identified.
  - Potential incremental risk from chromium, lead, and zinc to the horned lark was identified.
  - Receptor use of this SWMU is expected to be highly unlikely due to this nearly complete absence of any vegetation.
- No contamination was detected that could be a source of significant environmental degradation.
- No hazardous wastes or explosive ordnance are stored at the unit.

- Although the general area of this SWMU is in use for industrial purposes, there is no current plan for managing solid or hazardous waste at SWMU 164.

No closure activities, closure schedule, closure certification, or post-closure care or monitoring are required at this unit. SWMU 164 should be considered closed upon approval of this closure plan module by UDEQ, Division of Solid and Hazardous Waste.

7.3 SWMU 166—AVERY WASH RACK NO. 3

SWMU 166, which is located in the southeastern corner of Avery, consists of a wash rack that was used in the 1950s to clean small railcars involved in an irradiated-food preservation project. The unit consists of a concrete pad with a central sump that are both surrounded by a 4 ft concrete wall. The washwater drained from the sump to an underground pump room that is part of the SWMU, and was then pumped to wastewater retention tanks beneath Building 1002 to the south. Because the railcars were washed both before and after each use, any contaminant releases from this SWMU would be limited to traces of railcar lubricants in soapy washwater. This unit was later used to store containers of petroleum, oil, and lubricants for other heavy equipment.

Four soil samples collected from this SWMU contained eight metals above background and five detected organic compounds, including acetone, methylene chloride, toluene, xylenes, and TPHC. Of the eight metals above background, none was retained as a COPC. The ecological risk assessment evaluated all detected chemicals at this SWMU. A contamination assessment and risk assessment evaluating these COPCs resulted in the following conclusions:

- The noncancer hazards for residential use was below 1.0.
- A preliminary ecological risk assessment showed no significant incremental risk to wildlife from SWMU contaminants because incremental risk calculations were low, and exposure of wildlife is unlikely.
  - No incremental risk to the deer mouse or the horned lark was identified.
  - Receptor use of this SWMU is expected to be highly unlikely due to the nearly complete absence of any vegetation.

- No contamination was detected that could be a source of significant environmental degradation.
- No hazardous wastes or explosive ordnance are stored at the unit.
- Although the general area surrounding SWMU 166 is in use for industrial purposes, there is no current plan for managing solid or hazardous waste at the unit.

No closure activities, closure schedule, closure certification, or post-closure care or monitoring are required at this unit. Deed restrictions will be implemented to prevent residential use. SWMU 166 should be considered closed upon approval of this closure plan module by UDEQ, Division of Solid and Hazardous Waste.

7.4 SWMU 170—ENGLISH VILLAGE STEAM CLEANING AREA

SWMU 170, located in the southwestern part of English Village, consists of a sloped concrete pad that was previously used to clean uncontaminated vehicles and is currently used to clean dumpsters from the English Village residential area. The concrete pad supports a steel frame used to hold the dumpsters during cleaning and slopes toward a sump at the east end of the pad, which then discharges to the sanitary sewer system. Any contaminant releases at this SWMU would be limited to traces of organic and inorganic compounds commonly found in household trash.

Six soil samples collected from this SWMU contained five metals above background and five detected organic compounds, including acetone, methylene chloride, PPDDT, toluene, and xylenes, at low concentrations. Of the five metals above background, none were retained as COPCs. The ecological risk assessment evaluated all detected chemicals at this SWMU. A contamination assessment and risk assessment evaluating these COPCs resulted in the following conclusions:

- Human health risks qualify SWMU 170 for risk-based closure.

- A preliminary ecological risk assessment showed a low level of potential risk to wildlife.
  - There is no incremental risk to the deer mouse and there is incremental risk to the horned lark from chromium and lead.
  - Due to the developed nature of this SWMU, receptor use is considered to be highly unlikely.
- No contamination was detected that could be a source of significant environmental degradation.
- No hazardous wastes or explosive ordnance are stored at the unit.
- Although the unit will continue to be used as long as there is a residential community at English Village and the surrounding area is in use for industrial purposes, there is no plan to manage hazardous waste at SWMU 170 now or in the future.

No closure activities, closure schedule, closure certification, or post-closure care or monitoring is required at the unit. These results indicate that SWMU 170 should be considered closed upon approval of this closure plan module by UDEQ, Division of Solid and Hazardous Waste.

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APPENDIX A

STATISTICAL ANALYSIS
OF SOIL GEOCHEMISTRY

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STATISTICAL ANALYSIS OF SOIL GEOCHEMISTRY

A.1 BACKGROUND DATASET PREPARATION

The background dataset used for the evaluation of SWMU-specific contamination was prepared carefully to ensure that the dataset is truly representative of background conditions. The data set was prepared in the following steps.

1. All available data for background samples collected at DPG from the Consent Order and Corrective Action programs were combined.
2. Data for samples collected at depths greater than 20 feet (ft) were deleted (Section A.1.1).
3. Using a nonparametric ANOVA, the surface soil and subsurface soil datasets for each metal were compared to ensure they were not statistically different and then the data from all depth intervals were combined into one dataset (Section A.2.1)
4. Statistical outliers were removed (Section A.1.2).
5. All results for samples with at least one detection of an organic compound were deleted (Section A.1.3).
6. Spatial trends across DPG in the data were evaluated (Section A.1.4).

A.1.1 Samples Collected at Depths Greater Than 20 Feet

Background samples were collected to a maximum depth of 87 ft at Corrective Action SWMUs. Since a depth of 20 ft corresponds to likely remedial excavation depth and maximum exposure depth for ecological receptors, the 10 background samples collected more than 20 ft below ground surface were eliminated from consideration so that the final background dataset would not be biased by samples from lower stratigraphic units. Table A-1 presents the analytical data for constituents detected in these samples.

A.1.2 Outlier Testing

The background data for each metal were tested for upper extreme outliers in order to identify and then eliminate anomalous high measurements. Elimination of these upper extreme outliers resulted in a lower (more conservative) upper tolerance limit (UTL)

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The evaluation of statistical outliers in the background dataset was quantitative. Using guidance provided by Gilbert (1987) and the EPA in Statistical Analysis of Ground-Water Monitoring Data at RCRA Facilities (EPA 1992) the maximum detection of each metal was tested as a function of the size of the population, arithmetic mean, and standard deviation of the appropriate dataset. The outlier evaluation assumes that the values, excluding the outlier, were normally distributed. EPA guidance (1989, Section 6.2) states that "since lognormally distributed measurements often contain one or more values that appear high relative to the rest, it is recommended that the outlier test be run on the logarithms of the data instead of the original observations." For the purpose of the outlier test, the background dataset was assumed to be lognormally distributed, and the outlier test is performed according to the aforementioned EPA guidance.

For the calculation of mean and standard deviation of natural logarithm of values in the dataset, a value of one-half the method detection limit (MDL) was substituted in to the dataset for all nondetections. The following calculation was completed for each metal in order to determine the potential (T_n) of the maximum detection as an outlier:

$$T_n = \left(\frac{X_{max} - X_{mean}}{s} \right)$$

where:

T_n	=	Outlier statistic
n	=	Number of data points in the dataset
X_{max}	=	Natural logarithm of the maximum detected value
X_{mean}	=	Mean of the natural logarithm of values in the dataset
s	=	Standard deviation of the natural logarithm of values in the dataset

The resulting statistical T_n values are then compared to a critical value designated lambda (L). Lambda is determined as a function of probability, P (95%), and sample population size, n. To determine whether, or not , a concentration value is a statistical outlier is

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determined by comparing T_n to L. Two cases exist when comparing the calculated (T_n) to the critical L statistic:

1) $T_n \leq L$

2) $T_n > L$

Final results that fall into category 2 indicate that the concentration value is a statistical outlier.

If professional judgement found it necessary to conduct further outlier tests on the second highest value for each metal, where the maximum value was considered an outlier, these values were also tested.

Based on the final results of the outlier test, five data points can be considered statistical outliers. These statistical outliers are presented in Table A-2. Arsenic, iron, and vanadium in a sample collected at 9 ft from site EGL075SB04 were determined to be statistical outliers. Cobalt and manganese in a sample collected from 5 ft at site BKR999SB03 were also determined to be statistical outliers. The five records for these outliers will be removed from the final database prior to calculating background values. Data for other inorganic analytes in that sample were not deleted from the dataset.

The background data was also plotted on a DPG base-wide map and evaluated for spatial trends as presented in Plate A-1. Although the elevated levels of lead at SWMUs 2, 7, and 9 are not all statistical outliers, the data indicate a trend for this metal in the vicinity of Granite Peak. Therefore, where lead concentrations in samples from SWMUs 2, 7, and 9 exceed the lead background value, they will be compared to the higher lead levels in the six background samples from this part of DPG.

A.1.3 Detections of Organic Analytes in Background Samples

The presence of organic analytes in background samples indicates a potential for nonbackground conditions due to SWMU activities. Individual samples were deleted

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from the background dataset if at least one organic analyte was detected. Table A-3 presents a list of organic analytes that were detected in background samples.

A.1.4 Evaluation of Site-Wide Soil Geochemistry

All trace metal background levels (except for lead) were calculated installation-wide instead of separately for each soil type. The calculation of these values installation-wide involved combining all of the data into one dataset as stated in Section A.1. Justification of this procedure is illustrated below by breaking down this dataset by soil type for each of five metals that are important contributors to risk at the Consent Order units–arsenic, beryllium, lead, manganese, and thallium.

Objective

The purpose of the background dataset is to provide a basis for comparison with SWMU sample data. If trace metal concentrations measured in SWMU sample data are significantly elevated above levels measured in the background samples, then contamination may be indicated. However, the comparison of the SWMU and background datasets must be done with careful consideration of other site conditions that can render the comparison problematic, especially if background data are subdivided by soil type designations assigned on the basis of soil characteristics other than, and potentially unrelated to, geochemistry. Examples of these site conditions are:

- Soil type maps are typically used for planning agricultural land use. The mapped soil units do not delineate a homogeneous soil condition across the mapping unit; therefore, the soil type codes may not be accurate for an individual sample in either the SWMU or background dataset.

- An area mapped as a single soil type may contain soils with a variety of geologic parent materials with different geochemical compositions, as the maps were prepared at a subregional level of detail.

- The presence of active eolian dunes at DPG shows that in places the surface soil is transient and may not reflect the underlying soil type or map designation.

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- Many of the Consent Order SWMUs where data were collected for comparison to background are constructed in built-up areas that are composed either of extensively disturbed soils from the local area or fill brought from other areas of potentially different soil types.

- These considerations lead to uncertainty in the comparison of SWMU data to the background dataset, particularly if the data were subdivided; therefore, the comparison should be viewed as a screening process that requires subjective evaluation and cannot be used as an absolute determination of the presence or absence of contamination

Approach

Two types of soil mapping information are available for DPG. The first is illustrated in Section 1, Figure 1.1-4, and consists of a map based on the depositional environments of the unconsolidated surficial geologic materials across the installation. This information is taken from the DPG Master Plan Basic Information Maps. The second type of information is more detailed soil type mapping performed by the US Department of Agriculture, Natural Resources Conservation Service (formerly the Soil Conservation Service). The following evaluation of trace metal geochemistry with respect to soil type begins with this more detailed soil type delineation.

NRCS Soil Types

To assign soil type designations to each sample in the background geochemistry database, the sample locations were transferred onto copies of aerial photographs obtained from the NRCS in Salt Lake City. These photographs have been interpreted and marked with soil type designations by the NRCS. Table A-4 shows the name of each soil type in which a Consent Order unit or background sample is located, as well as the number of background samples in the dataset that fell in each soil type area. Mapping was not available for the areas around SWMUs 42, 43, 130, and 190. These samples were coded "Y," for unknown. After the samples were assigned to a soil type, they were subdivided by soil type for evaluation. These data were then displayed in "box and whisker" plots (Figures A-1 through A-5).

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In general, the whiskers, or vertical lines in these plots, show the range of values in each subdivided dataset. Outliers are shown as single points or small boxes beyond the length of the lines. The boxes show the range of values between the 25th and 75th percentile, and the horizontal line within each box shows the mean value. For purposes of evaluating trace metal concentrations at waste management units to background geochemistry, the most important information provided by the plots is probably the 75th percentile and maximum values. These results were evaluated subjectively by inspection and compared to the UTLs calculated on the combined dataset and presented previously in the closure plans. Emphasis is placed on diagrams for soil types in which more samples were collected, as the range of values detected appears to be related in part to the number of samples analyzed. All concentrations in this discussion and illustrated in the diagrams are in units of μg/g.

Arsenic. The 75th percentile values for most soil types are between 9 and 11. Exceptions include soil types 27 and 69, in which only 5 and 3 samples were collected respectively, and 42, which had a lower 75th percentile, but also some values above 20 and at 58. Therefore, the UTL of 13 calculated for the complete dataset seems appropriate for all of the soil types with significant numbers of samples.

Beryllium. Most of the 75th percentile and maximum values range from approximately 0.7 to 1.0. Most of the maximum values range from 0.8 and 1.1. This difference is not considered significant, and the previously presented UTL of 1 is a reasonable approximation of the maximum background concentration.

Lead. The 75th percentile values for datasets with more than 5 samples ranged from 8 to 12. The maximum concentrations ranged from 9 to 16, except in soil type 42, which was lower overall, but had one sample concentration as high as 29. The UTL of 14 is a reasonable approximation of the maximum background for these populations.

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Manganese. The 75th percentile values ranged widely from 250 to 420 in the larger datasets, and the maximum concentrations ranged from 380 to 480 in most soil types, with the highest value measured at 2,000 in one soil type. The approximation of 520 as a background level is appropriate to this wide range in values.

Thallium. Of the five metals, thallium concentrations seemed to vary the most between soil types, but this is attributed to the relatively low number of thallium detections represented in the diagrams. Whereas the datasets for each of the other metals evaluated included 80 to 86 samples, the thallium dataset includes only 39 samples and only 25 detections divided among the seven soil types and one unknown category. Therefore, this diagram probably does not show meaningful information on the distribution of thallium concentrations in the various soil types. However, the UTL of 35 compares reasonably well with the maximum detected concentration of 46.

Generalized Source Areas

To ensure complete evaluation of the spatial variability related to soil type and underlying sediment composition, the samples were also grouped according to the depositional environments and source rock areas illustrated in Figure 1.1-4. Table A-5 shows the geographic categories evaluated and number of samples in each category.

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Table A-5 Number of Background Samples by Geographic Location

Geographic Area	Number of Samples
Baker	8
Camels Back Peak	3
Carr-Ditto	34
Central	14
English Village-Fries Park	19
North Granite Peak	6
South Granite Peak	4
North	2
Tower Grid	6

When the numbers of samples and detections in each category are considered as before, the box and whisker plots for these additional dataset groupings (Figures A-6 through A-11) also show uniformity of background trace metal concentrations among the different lithic source areas and depositional environments. The only exception is lead at Granite Peak. In Figure A-11, the Granite Peak samples are separated into north and south to confirm that the samples near the northern, mineralized portion of this source area are higher in lead than those to the south. Comparison of these lead diagrams shows the type of distinction that can be interpreted as a clear indication of a different natural trace metal geochemistry in this area, whereas no clear distinction of this type is seen in any of the other diagrams representing more than five or six samples. This result confirms the previous interpretation presented in the closure plan that only lead in SWMUs near the northern part of Granite Peak should be evaluated against a local background level. All other metals at all other SWMUs should be evaluated against the combined background dataset.

A.2 STATISTICAL METHODOLOGY

Once the final background dataset was established, an approach for the statistical analysis of the DPG dataset was developed to evaluate the following:

1. Comparison of the SWMU dataset against the background dataset as a whole for each metal to identify contamination in the SWMU area.
2. Identification of relatively high measurements in the SWMU dataset that could indicate hot spot contamination.

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For the first evaluation, an analysis of variance was used to compare SWMU and background data populations. If the two datasets were shown not to be statistically different (at a given level of confidence and power), then the results were interpreted to show an absence of contamination at the SWMU. For the second type of evaluation, a background UTL was calculated for each metal; and the SWMU measurements for each metal were compared to the appropriate background UTL to identify potential hot spots for subjective evaluation. The UTL was also used to evaluate the magnitude of detections of metals identified by the ANOVA as potential SWMU contaminants. Specific information on the methodology of the background

A.2.1 Nonparametric ANOVA – Wilcoxon Rank-Sum Test

The statistical methodology followed in performing the analysis of variance (ANOVA) was also developed in accordance with guidance documents and is supported by the draft ASTM standard entitled "Guide to the Comparison of Hazardous Waste Site and Background Soil Data." These guidance documents outline several criteria that must be considered to select an appropriate ANOVA methodology. These criteria are used to match specific dataset characteristics with appropriate statistical methodologies. For parametric methods to be applicable, the background dataset and SWMU dataset must have the same distribution for a given metal. EPA guidance (1992) lists the following tests that must be satisfied by both datasets for parametric methods to be valid:

- Test for normality
- Test for lognormality
- Test for homogeneity of variances

The purpose of using a nonparametric ANOVA is to compare to separate data populations and determine whether they are similar enough to be considered one population, or different enough to be considered two distinct populations. If the populations are determined to be different, an additional comparison is completed to determine their relative ranks. First, a comparison was done for metals in the surface and subsurface background soil datasets. This ANOVA was used to evaluate surface and subsurface background data to determine

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whether the surface and subsurface metals concentrations were similar enough to combine into one dataset or if they were different enough to warrant separate evaluations. A second comparison was completed to determine the potential for contamination using site-specific SWMU data and the final background dataset by comparing the datasets and their relative ranks.

The nonparametric ANOVA is appropriate when the distribution of data cannot be verified (for example, data are not of normal distribution or have significantly different group variances) (Larsen and Marx 1990). Thus, if the sample size is sufficient, a nonparametric ANOVA can be used in any case where a parametric ANOVA is used (EPA 1989, 1992). In addition, each dataset must contain fewer than 15 percent nondetections. Table A-6 shows how the datasets for metals detected at each of the Modules 2 and 3 SWMUs compared to these criteria. The third column in Table A-6 shows which datasets contain more than 15 percent nondetections; for these datasets, nonparametric methods are recommended (EPA 1992, Section 3.2). The fourth column shows the datasets identified as non-normal by the coefficient of variance. Additional testing of the dataset distributions would probably show that several additional datasets are non-normal, as well. Nonparametric methods are recommended to evaluate non-normal datasets (EPA 1989, section 5.2; EPA 1996, Section 3.3.1.1). The fifth column shows the results of testing the SWMU and background datasets for homogeneity of variances. Where homogeneity of variances is not demonstrated, nonparametric methods are recommended.

Table A-6 shows that at least 529 of the 819 datasets evaluated in this way (or at least 65 percent) should be evaluated using nonparametric methods. Additional distribution testing would probably show that nonparametric methods would be preferred for use in evaluating many of the remaining 290 datasets. For this reason, nonparametric methods have been selected for consistent use in comparing the SWMU and background datasets. These methods are better suited to evaluating more than half of the SWMU datasets and can be used with no significant reduction in the power that would have resulted if parametric methods had been appropriate instead.

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Concept of Power in a Nonparametric Test

The concepts of confidence and power, and the ability to detect a type I and type II error, respectively, apply to both parametric tests and nonparametric tests.

Technically, power refers to the probability that a statistical testing procedure will register and identify evidence of contamination when it exists. The power of any statistical test depends on several factors (such as number of samples) that vary with each metal; and the power is not a single number, but rather a function of the level of contamination actually present (EPA 1992, Section 5.1, p. 64). However, in the 1992 guidance, EPA compared the powers of both parametric and nonparametric procedures in general by evaluating the relative efficiency. The efficiency of the nonparametric procedure is 95 percent if the data are really normal, and can be much larger than 100 percent of the efficiency of a parametric method in other non-normal cases. This means that in the worst case, when a nonparametric method is used only 5 percent efficiency is lost, but if the data include greater than 15 percent nondetections or data are not normal, the power of the nonparametric method is much greater.

After this comparison, the EPA guidance restates that the nonparametric tests such as the Wilcoxon Rank-Sum test and the Kruskal-Wallis test, such as was used for the groundwater evaluation in the Corrective Action program, is reasonably powerful for detecting concentration differences and can be used even when the data are normally distributed. When data are not normal, parametric tests tend to be more powerful for detecting differences than the usual parametric approach (EPA 1992, Section 3.1, p. 42).

The power of any testing method can be increased by relaxing the false positive rate requirement (alpha). EPA emphasized that the alpha be at least 1 percent. The rationale for this minimum requirement is motivated by statistical power. The guidance states a minimum level of acceptable power cannot be specified within the regulations because to do so would require specification of a minimum difference of environmental concern

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between the null and alternative hypotheses. Limited current knowledge about health and/or environmental effects associated with incremental changes in concentration levels constituents greatly complicates this task. Therefore, minimum false positive rates (alpha) were adopted by EPA to ensure fair power for some statistical procedures until more specific guidance could be recommended (EPA 1992, Section 5.1, p. 63).

However, to limit Type II error, the probability of Type I error must increase. This translates to the use of a higher alpha level. To balance the probabilities of the two types of errors, an approximate 5 percent alpha level is suggested as the guidance standard (EPA 1992, Section 5.1, p. 66). This recommended 5 percent alpha level was used in the closure plan.

Statistical Computations

The Wilcoxon Rank-Sum test compares two data populations by calculating a Z-statistic for each metal based on the sample size and the ranks of each concentration value within the two populations. The procedures for performing the nonparametric Wilcoxon Rank-Sum test are outlined below.

The first step in performing the Wilcoxon Rank-Sum test requires that the subsurface soil data (sample size = n) and surface soil data (sample size = m) be combined and ranked from one to N, where N = n + m. From this, the W-statistic is then calculated using the following equation:

$$W = \sum_{i=1}^{n} C_i - \frac{n}{2}(n+1)$$

where:

W	=	Calculated W-statistic
n	=	Sample size of the subsurface soil dataset
C_i	=	Ranks of the two datasets

The rationale of this nonparametric procedure is that if the ranks of the subsurface soil dataset are significantly greater, or significantly less, than the ranks of the subsurface soil

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dataset, the hypothesis of the two datasets being within the same population can be rejected. Extremely large or small values of the W-statistic indicate a statistical difference in metals concentrations for surface soil compared to subsurface soil.

Next, to find the critical value for W, a normal approximation to its distribution is assumed under the null hypothesis of no statistical difference. The following equations are used to calculate the standard deviation (S_W) and the expected value (E_W) of W:

$$E_W = \frac{mn}{2}$$

$$S_W = \sqrt{\frac{mn(N+1)}{12}}$$

where:

E_W	=	expected value of W
S_W	=	standard deviation of W
m	=	sample size of the surface soil dataset
n	=	sample size of the subsurface soil dataset
N	=	sample size of the combined datasets (m + n)

From this, the approximate Z-score for the Wilcoxon Rank-Sum Test is calculated using the following:

$$Z = \frac{(W - E_W - 0.5)}{S_W}$$

The factor of 0.5 in the numerator of the previous equation is a correction factor to account for the assumption of normal distribution of the W-statistic (Larsen and Marx 1990).

Once the Z-statistic has been calculated it is compared to the upper and lower 0.025 percentile (P = 0.025) of the standard normal distribution (Larsen and Marx 1990). If the calculated Z-lies outside of the limits of a 5 percent two-tailed probability (Z_P and Z_{P-1}) the null hypothesis at the 5 percent significance is rejected, suggesting that there a statistical difference between surface soil and subsurface soil metals concentrations for background locations at DPG.

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Background Surface Soil Data vs. Background Subsurface Soil Data

The results of the Wilcoxon Rank-Sum test can fall into one of three categories:

1) $Z < Z_{0.025}$ $[Z_{0.025} = -(Z_{0.975})]$
2) $Z_{0.025} \leq Z \leq Z_{0.975}$
3) $Z > Z_{0.975}$

The results that fall into the first and third categories indicate a significant statistical difference exists between the surface soil and subsurface soil background datasets. Table A-7 presents the results of comparing surface an subsurface soil background data.

SWMU-Specific Soil Data vs. Background Soil Data

When comparing SWMU-specific and background data in support of the contamination assessment and selection of COCs for the human health and ecological risk assessments in this report the upper limit for 5 percent one-tailed probability is evaluated. The ranks of each population are compared; while the Z-statistic is compared to $Z_{0.95}$. The results can fall into one of four categories:

1) $Z \leq Z_{crit}$, $Rank_{SWMU} \geq Rank_{background}$, and $P \geq 0.05$
2) $Z \leq Z_{crit}$, $Rank_{SWMU} < Rank_{background}$, and $P \geq 0.05$
3) $Z > Z_{crit}$, $Rank_{SWMU} \geq Rank_{background}$, and $P < 0.05$
4) $Z > Z_{crit}$, $Rank_{SWMU} < Rank_{background}$, and $P < 0.05$

The results that fall into the third and fourth categories indicate a significant statistical difference exists between the SWMU-specific and background datasets. Table A-8 presents the results of comparing SWMU-specific and background data in support of the contamination assessment and selection of COCs for the human health and ecological risk assessments in this report. Table A-9 presents the definitions of statistical terms used in the Wilcoxon Rank-Sum test.

A.2.2 Upper Tolerance Limits for Soil Background Values

For use as a comparative background value, the 95 percent UTL was calculated for each metal in final background dataset. Tolerance limits define a range that contains at least a specified percent of a population (%N) with a probability (level of confidence,

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represented as p = 1 - P-value). In the case for determining background values the 95 percent tolerance interval is constructed to contain 95 percent (%N) of the distribution with a probability of 95 percent (p).

Comparison of site metals concentrations in individual samples to the UTL is a one-sided test, designed to indicate when site analytical data exceeding background may be indicative of contamination. In contrast to the ANOVA and t-tests, the comparison to a UTL is designed to evaluate individual sampling results, not to compare mean values of site and background analytes. Comparison to the UTL is designed to detect "hot spots" of one more site samples that exceed the likely range of background values.

The calculation of the 95 percent UTL was completed using the following formula (Gilbert 1987):

$$95\%\ \text{UTL} = \text{mean}_{bkgd} + K_n s$$

where:

mean_{bkgd}	=	arithmetic mean from final background dataset
K_n	=	tolerance factor based on sample size n (Appendix Table A-3; Gilbert 1987)
s	=	standard deviation

A UTL was calculated using a methodology appropriate for a normally distributed dataset since this method also results in a lower (more conservative) UTL. The guidance cited above indicates that an assumption can be made that the data are normally distributed once the outlier is excluded. The underlying distributions for several metals in the background data set were verified to meet this assumption.

The current approach to defining background values separates metals into three groups based on the percent of nondetections:

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- Less than, or equal to, 50 percent nondetections
- Greater than 50 percent nondetections
- Few detections, equal to 99 percent nondetections

In the first case, a normal 95 percent UTL was calculated as background values for analytes with less than, or equal to, 50 percent nondetections in the background data set.

In the second case, as the percentage of nondetections increases to greater than 50 percent, the normal UTL is not an accurate measure of a 95 percent UTL for the particular analyte in the background data set. In the second case, a nonparametric UTL can be chosen for an analyte in the DPG background data set as the maximum value (EPA 1992). Two metals including antimony and selenium would require the use of a nonparametric UTL set at the maximum value in the dataset.

The third case exists for cadmium and mercury. These two metals were each detected in one sample out of 86 and 84 total background samples, respectively. In this case the nonparametric UTL should be used as in the second case, but should be equal to the second largest value in the data set. In the cases of cadmium and mercury, the second largest value is the reporting limit (RL).

The statistical summary provided in Table A-10 presents the calculated background values as normal UTLs. The alternative background values as nonparametric UTLs are presented for the antimony and selenium as the maximum detected value. Analytes cadmium and mercury with only 1 percent detections have background values designated as the RL. The calculated background values presented in Table A-10 have been compared to site-specific data to determine areas potentially impacted by site activities as discussed in this report.

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APPENDIX A

Tables

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Table A-1 Analyte Detections for Background Soil Samples Collected Deeper Than 20 Feet

Site ID	Depth (ft)	Analyte	Analytical Method	Concentration (µg/g)	Background Area
CRR999SB01	53.0	AL	JS12	10600	Carr
CRR999SB01	53.0	B	JS12	18.6	Carr
CRR999SB01	53.0	BA	JS12	82.9	Carr
CRR999SB01	53.0	CA	JS12	43400	Carr
CRR999SB01	53.0	CR	JS12	11.6	Carr
CRR999SB01	53.0	CU	JS12	4.67	Carr
CRR999SB01	53.0	FE	JS12	11400	Carr
CRR999SB01	53.0	K	JS12	3890	Carr
CRR999SB01	53.0	MG	JS12	9920	Carr
CRR999SB01	53.0	MN	JS12	114	Carr
CRR999SB01	53.0	NA	JS12	1170	Carr
CRR999SB01	53.0	NI	JS12	4.33	Carr
CRR999SB01	53.0	PB	JD21	3.48	Carr
CRR999SB01	53.0	TOC	LKTC	5280	Carr
CRR999SB01	53.0	V	JS12	17.9	Carr
CRR999SB01	53.0	ZN	JS12	20.1	Carr
CRR999SB02	49.5	AL	JS12	10100	Carr
CRR999SB02	49.5	B	JS12	17.4	Carr
CRR999SB02	49.5	BA	JS12	81.5	Carr
CRR999SB02	49.5	BE	JS12	0.54	Carr
CRR999SB02	49.5	CA	JS12	42500	Carr
CRR999SB02	49.5	CR	JS12	10.2	Carr
CRR999SB02	49.5	CU	JS12	5.51	Carr
CRR999SB02	49.5	FE	JS12	10000	Carr
CRR999SB02	49.5	K	JS12	3390	Carr
CRR999SB02	49.5	MG	JS12	6520	Carr
CRR999SB02	49.5	MN	JS12	164	Carr
CRR999SB02	49.5	NA	JS12	928	Carr
CRR999SB02	49.5	NI	JS12	5.78	Carr
CRR999SB02	49.5	PB	JD21	3.19	Carr
CRR999SB02	49.5	V	JS12	17	Carr
CRR999SB02	49.5	ZN	JS12	19.3	Carr
CRR999SB03	49.0	AL	JS12	7530	Carr
CRR999SB03	49.0	B	JS12	14.3	Carr
CRR999SB03	49.0	BA	JS12	56.3	Carr
CRR999SB03	49.0	CA	JS12	46200	Carr
CRR999SB03	49.0	CR	JS12	7.32	Carr
CRR999SB03	49.0	DNBP	LM25	3.6	Carr
CRR999SB03	49.0	FE	JS12	8170	Carr
CRR999SB03	49.0	K	JS12	2220	Carr
CRR999SB03	49.0	MG	JS12	7500	Carr
CRR999SB03	49.0	MN	JS12	97.8	Carr
CRR999SB03	49.0	NA	JS12	1180	Carr
CRR999SB03	49.0	NI	JS12	4.27	Carr
CRR999SB03	49.0	PB	JD21	2.03	Carr
CRR999SB03	49.0	PHANTR	LM25	0.13	Carr
CRR999SB03	49.0	TOC	LKTC	3570	Carr
CRR999SB03	49.0	V	JS12	15.8	Carr
CRR999SB03	49.0	ZN	JS12	13.6	Carr
EGL044MW02	87.0	AL	JS12	33300	English Village
EGL044MW02	87.0	AS	B9	9.02	English Village
EGL044MW02	87.0	B	JS12	50.1	English Village
EGL044MW02	87.0	BA	JS12	507	English Village
EGL044MW02	87.0	BE	JS12	4.35	English Village
EGL044MW02	87.0	CA	JS12	8490	English Village
EGL044MW02	87.0	CO	JS12	7.66	English Village
EGL044MW02	87.0	CR	JS12	143	English Village

Table A-1 Analyte Detections for Background Soil Samples Collected Deeper Than 20 Feet

Site ID	Depth (ft)	Analyte	Analytical Method	Concentration (µg/g)	Background Area
EGL044MW02	87.0	CU	JS12	16.5	English Village
EGL044MW02	87.0	FE	JS12	39800	English Village
EGL044MW02	87.0	K	JS12	1590	English Village
EGL044MW02	87.0	MG	JS12	5380	English Village
EGL044MW02	87.0	MN	JS12	43.7	English Village
EGL044MW02	87.0	NA	JS12	1210	English Village
EGL044MW02	87.0	NI	JS12	24.1	English Village
EGL044MW02	87.0	PB	JD21	13	English Village
EGL044MW02	87.0	V	JS12	67.7	English Village
EGL044MW02	87.0	ZN	JS12	55.7	English Village
EGL069MW03	81.5	AL	JS12	4010	English Village
EGL069MW03	81.5	BA	JS12	62.6	English Village
EGL069MW03	81.5	CA	JS12	2540	English Village
EGL069MW03	81.5	CR	JS12	4.96	English Village
EGL069MW03	81.5	FE	JS12	6830	English Village
EGL069MW03	81.5	K	JS12	1300	English Village
EGL069MW03	81.5	MG	JS12	1850	English Village
EGL069MW03	81.5	MN	JS12	49.7	English Village
EGL069MW03	81.5	NA	JS12	468	English Village
EGL069MW03	81.5	PB	JD21	5.17	English Village
EGL069MW03	81.5	V	JS12	4.75	English Village
EGL069MW03	81.5	ZN	JS12	11.3	English Village
EGL075SB04	27.0	AL	JS12	16600	English Village
EGL075SB04	27.0	AS	B9	13.9	English Village
EGL075SB04	27.0	B	JS12	13.1	English Village
EGL075SB04	27.0	BA	JS12	179	English Village
EGL075SB04	27.0	BE	JS12	1.25	English Village
EGL075SB04	27.0	CA	JS12	67000	English Village
EGL075SB04	27.0	CO	JS12	8.19	English Village
EGL075SB04	27.0	CR	JS12	17.4	English Village
EGL075SB04	27.0	CU	JS12	10.3	English Village
EGL075SB04	27.0	FE	JS12	26300	English Village
EGL075SB04	27.0	K	JS12	2770	English Village
EGL075SB04	27.0	MG	JS12	6020	English Village
EGL075SB04	27.0	MN	JS12	457	English Village
EGL075SB04	27.0	NA	JS12	2900	English Village
EGL075SB04	27.0	NI	JS12	20.2	English Village
EGL075SB04	27.0	PB	JS12	9.52	English Village
EGL075SB04	27.0	PB	JD21	12.3	English Village
EGL075SB04	27.0	V	JS12	30.6	English Village
EGL075SB04	27.0	ZN	JS12	61.2	English Village
EGL075SB04	42.0	AL	JS12	15100	English Village
EGL075SB04	42.0	AS	B9	15.8	English Village
EGL075SB04	42.0	B	JS12	12.1	English Village
EGL075SB04	42.0	BA	JS12	151	English Village
EGL075SB04	42.0	BE	JS12	1.06	English Village
EGL075SB04	42.0	CA	JS12	70000	English Village
EGL075SB04	42.0	CO	JS12	8.03	English Village
EGL075SB04	42.0	CR	JS12	21.8	English Village
EGL075SB04	42.0	CU	JS12	9.6	English Village
EGL075SB04	42.0	FE	JS12	24900	English Village
EGL075SB04	42.0	K	JS12	3350	English Village
EGL075SB04	42.0	MG	JS12	7780	English Village
EGL075SB04	42.0	MN	JS12	254	English Village
EGL075SB04	42.0	NA	JS12	1830	English Village
EGL075SB04	42.0	NI	JS12	15.9	English Village
EGL075SB04	42.0	PB	JD21	10.1	English Village

Table A-1 Analyte Detections for Background Soil Samples Collected Deeper Than 20 Feet

Site ID	Depth (ft)	Analyte	Analytical Method	Concentration (µg/g)	Background Area
EGL075SB04	42.0	V	JS12	44.1	English Village
EGL075SB04	42.0	ZN	JS12	59.6	English Village
EGL075SB04	61.0	AL	JS12	22300	English Village
EGL075SB04	61.0	AS	B9	10.1	English Village
EGL075SB04	61.0	B	JS12	17.2	English Village
EGL075SB04	61.0	BA	JS12	268	English Village
EGL075SB04	61.0	BE	JS12	1.39	English Village
EGL075SB04	61.0	CA	JS12	81000	English Village
EGL075SB04	61.0	CO	JS12	6.56	English Village
EGL075SB04	61.0	CR	JS12	17.7	English Village
EGL075SB04	61.0	CU	JS12	12.4	English Village
EGL075SB04	61.0	FE	JS12	21800	English Village
EGL075SB04	61.0	K	JS12	4640	English Village
EGL075SB04	61.0	MG	JS12	10600	English Village
EGL075SB04	61.0	MN	JS12	234	English Village
EGL075SB04	61.0	NA	JS12	1090	English Village
EGL075SB04	61.0	NI	JS12	15.1	English Village
EGL075SB04	61.0	PB	JD21	12.9	English Village
EGL075SB04	61.0	V	JS12	33.5	English Village
EGL075SB04	61.0	ZN	JS12	58.3	English Village
TWR016MW03	84.0	AL	JS12	20900	Tower Grid
TWR016MW03	84.0	AS	JS12	25.8	Tower Grid
TWR016MW03	84.0	AS	B9	4.75	Tower Grid
TWR016MW03	84.0	BA	JS12	232	Tower Grid
TWR016MW03	84.0	BE	JS12	1.04	Tower Grid
TWR016MW03	84.0	CA	JS12	150000	Tower Grid
TWR016MW03	84.0	CO	JS12	6.51	Tower Grid
TWR016MW03	84.0	CR	JS12	16.7	Tower Grid
TWR016MW03	84.0	CU	JS12	17.3	Tower Grid
TWR016MW03	84.0	FE	JS12	20000	Tower Grid
TWR016MW03	84.0	K	JS12	7230	Tower Grid
TWR016MW03	84.0	MG	JS12	26900	Tower Grid
TWR016MW03	84.0	MN	JS12	418	Tower Grid
TWR016MW03	84.0	NA	JS12	14100	Tower Grid
TWR016MW03	84.0	NI	JS12	12.8	Tower Grid
TWR016MW03	84.0	PB	JS12	10.1	Tower Grid
TWR016MW03	84.0	PB	JD21	3.25	Tower Grid
TWR016MW03	84.0	V	JS12	31.2	Tower Grid
TWR016MW03	84.0	ZN	JS12	53.9	Tower Grid
TWR017MW01	84.0	AL	JS12	10900	Tower Grid
TWR017MW01	84.0	AS	B9	3.33	Tower Grid
TWR017MW01	84.0	BA	JS12	154	Tower Grid
TWR017MW01	84.0	CA	JS12	61600	Tower Grid
TWR017MW01	84.0	CR	JS12	8.72	Tower Grid
TWR017MW01	84.0	CU	JS12	7.71	Tower Grid
TWR017MW01	84.0	FE	JS12	12600	Tower Grid
TWR017MW01	84.0	K	JS12	2360	Tower Grid
TWR017MW01	84.0	MG	JS12	12000	Tower Grid
TWR017MW01	84.0	MN	JS12	279	Tower Grid
TWR017MW01	84.0	NA	JS12	1720	Tower Grid
TWR017MW01	84.0	NI	JS12	5.99	Tower Grid
TWR017MW01	84.0	PB	JD21	5.92	Tower Grid
TWR017MW01	84.0	V	JS12	24.4	Tower Grid
TWR017MW01	84.0	ZN	JS12	25.4	Tower Grid

Table A-2 Statistical Outliers in the Final Background Soil Data Set

SITE_TYPE	SITE_ID	TESTNAME	METHOD	FC	DATA_QUALS	DEPTH	BOOLEAN	VALUE	UNITS	EPAQUAL	PRIM_CONT
BORE	EGL075SB04	AS	B9			9		58.00000000	UGG	NA	EZ
BORE	EGL075SB04	FE	JS12			9		54900.00000000	UGG	NA	EZ
BORE	BKR999SB03	MN	JS12			5		2000.00000000	UGG	NA	EZ
BORE	EGL075SB04	V	JS12			9		57.00000000	UGG	NA	EZ
BORE	BKR999SB03	CO	JS12			5		27.20000000	UGG	NA	EZ

NA - EPAQUAL for EPA qualifier is not applicable for Parsons Engineering Science data
EZ - Parsons Engineering Science
FC - Flagging code
DATA_QUALS - Data qualifiers
PRIM_CONT - Prime contractor
µg/g - Micrograms per gram
Analyte acronyms are defined in the Acronym List of the main report.

Table A-3 Detections of Organic Compounds in Background Samples

SITE_TYPE	SITE_ID	TESTNAME	METHOD	FC	DATA_QUAL	DEPT	BOOLEAN	VALUE	UNITS
BORE	020BG01	CH2CL2	8240	S			2	.0054	UGG
BORE	020BG01	MEC6H5	8270	S	S		2	.51	UGG
BORE	020BG01	MEC6H5	8240	S			2	.0087	UGG
BORE	033BG01	ACET	8240	S			3	.03	UGG
BORE	036BG01	B2EHP	8270	S			4	.97	UGG
GRAB	037BG01	LIN	8080	S			0	.0006	UGG
GRAB	037BG01	PPDDT	8080	S			0	.00088	UGG
BORE	037BG01	ACET	8240	S			3	.026	UGG
GRAB	038BG01	TPHC	4181	S			0	31	UGG
GRAB	039BG01	DNBP	8270	S			0	.27	UGG
BORE	039BG01	DNBP	8270	S			3	.37	UGG
GRAB	040BG01	ACLDAN	8080	S			0	.0018	UGG
GRAB	040BG01	B2EHP	8270	S			0	.11	UGG
GRAB	040BG01	GCLDAN	8080	S			0	.0022	UGG
GRAB	040BG01	PPDDE	8080	S	P		0	.0029	UGG
GRAB	040BG01	PPDDT	8080	S			0	.0061	UGG
BORE	040BG01	ACLDAN	8080	S			3	.014	UGG
BORE	040BG01	B2EHP	8270	S			3	.22	UGG
BORE	040BG01	DLDRN	8080	S	P		3	.001	UGG
BORE	040BG01	GCLDAN	8080	S			3	.015	UGG
BORE	040BG01	PPDDD	8080	S	P		3	.0023	UGG
BORE	042BG01	DEP	8270	S			3	.012	UGG
GRAB	043BG01	TPHC	4181	S			0	12	UGG
BORE	043BG01	DLDRN	8080	S			3	.0023	UGG
GRAB	046BG01	PPDDT	8080	S			0	.0013	UGG
GRAB	048BG01	PCB260	8080	S			0	.244	UGG
BORE	048BG01	ACET	8240	S			3	.041	UGG
BORE	048BG01	ENDRN	8080	S			3	.00225	UGG
GRAB	051BG01	C16A	8270	S	S		0	.32	UGG
GRAB	051BG01	EICOSL	8270	S	S		0	2.1	UGG
GRAB	055BG01	DNBP	8270	S			0	.032	UGG
GRAB	055BG01	PPDDT	8080	S			0	.0013	UGG
BORE	055BG01	TRCLE	8240	S			3	.0015	UGG

Table A-3 Detections of Organic Compounds in Background Samples

SITE_TYPE	SITE_ID	TESTNAME	METHOD	FC	DATA_QUAL	DEPT	BOOLEAN	VALUE	UNITS
BORE	059BG01	CH2CL2	8240	S			3	.0013	UGG
GRAB	063BG01	AENSLF	8080	S			0	.00088	UGG
BORE	063BG01	AENSLF	8080	S			3	.00065	UGG
BORE	063BG02	CH2CL2	8240	S			3	.0096	UGG
BORE	124BG01	B2EHP	8270	S			3	.042	UGG
BORE	124BG01	ENDRN	8080	S		Q	3	.0017	UGG
BORE	128BG01	BBZP	8270	S			3	.014	UGG
BORE	128BG01	PYR	8270	S			3	.006	UGG
BORE	130BG01	MEC6H5	8270	S	S		3	.31	UGG
GRAB	163BG01	246TCA	8270	S	S		0	1.1	UGG
GRAB	164BG01	B2EHP	8270	S			0	.19	UGG
GRAB	164BG01	TPHC	4181	S			0	140	UGG
BORE	164BG01	ACET	8240	S			3	.015	UGG
BORE	164BG01	B2EHP	8270	S			3	.32	UGG
BORE	165BG01	ACET	8240	S			3	.027	UGG
BORE	165BG01	TPHC	4181	S			3	170	UGG
GRAB	167BG01	TPHC	4181	S			0	140	UGG
BORE	167BG01	ACET	8240	S			3	.13	UGG
BORE	167BG01	CH2CL2	8240	S			3	.016	UGG
BORE	APG999SB01	BZALC	LM25	S			2	0.05600000	UGG
BORE	APG999SB03	MEC6H5	LM23	S			10	0.12000000	UGG
BORE	BKR999SB01	BZALC	LM25	S			4	0.17000000	UGG
BORE	BKR999SB03	BZALC	LM25	S			2	0.14000000	UGG
BORE	BKR999SB03	DEP	LM25	S			2	5.00000000	UGG
BORE	BKR999SB03	BZALC	LM25	S			4	0.17000000	UGG
BORE	BKR999SB03	BZALC	LM25	S			5	0.23000000	UGG
BORE	CRR999SB01	DNBP	LM25	S			15	4.30000000	UGG
BORE	CRR999SB03	DNBP	LM25	S			49	3.60000000	UGG
BORE	CRR999SB03	PHANTR	LM25	S			49	0.13000000	UGG
BORE	DTO999SB01	BZALC	LM25	S			15	0.22000000	UGG
BORE	DTO999SB01	BZALC	LM25	S			2.5	0.18000000	UGG
BORE	DTO999SB02	BZALC	LM25	S			15	0.06700000	UGG
BORE	DTO999SB02	BZALC	LM25	S			20	0.05500000	UGG

Table A-3 Detections of Organic Compounds in Background Samples

SITE_TYPE	SITE_ID	TESTNAME	METHOD	FC	DATA_QUAL	DEPT	BOOLEAN	VALUE	UNITS
BORE	DTO999SB03	NNDPA	LM25	S			19	0.69000000	UGG
BORE	DWD999SB03	DNBP	LM25	S			15	4.00000000	UGG
BORE	DWD999SB03	DNBP	LM25	S			17	2.30000000	UGG
BORE	DWD999SB03	DNBP	LM25	S			2.5	1.90000000	UGG
AHOL	GRM999HA01	DNBP	LM25	S			3.5	2.90000000	UGG

EB - Ebasco
EZ - Parsons Engineering Science
FC - Flagging code
DATA_QUALS - Data qualifiers
PRIM_CONT - Prime contractor
µg/g - Micrograms per gram
Analyte acronyms are defined in the Acronym List of the main report.

Table A-4 Dugway Proving Ground Background Soil Types

Soil Type	Number of Samples	Soil Type Name
27	5	Izamatch-Cliffdown alkali complex
42	17	Medburn fine sandy loam
56	32	Skumpah silt loam
59	10	Skumpah silt loam, saline
60	6	Skumpah-Yanrab complex, saline
67	15	Timpie silt loam, saline
69	3	Tooele fine sandy loam
70	4	Tooele fine sandy loam, saline
Y	4	Soil types not determined for Primary soil type Y SWMUs 42, 43, 130, and 190

Table A-6 Parametric Test Criteria for Background Metals and Metals Detected in Soil at Dugway Proving Ground

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
Background	AG	> 15%		---
	AL			---
	AS			---
	BA			---
	BE	> 15%		---
	CA			---
	CD	> 15%		---
	CO	> 15%		---
	CR			---
	CU			---
	FE			---
	HG	> 15%		---
	K			---
	MG			---
	MN			---
	NA			---
	NI			---
	PB			---
	SB	> 15%		---
	SE	> 15%	Non-normal	---
	TL	> 15%		---
	V			---
	ZN			---
SWMU 2	AG	> 15%	Non-normal	No
	AL			No
	AS		Non-normal	No
	BA			
	BE	> 15%		
	CA			No
	CD	> 15%	Non-normal	
	CO			No
	CR			No
	CU		Non-normal	No
	FE			No
	K			No
	MG			
	MN			
	NA			
	NI			
	PB		Non-normal	No
	SB	> 15%		No
	SE	> 15%		No
	TL	> 15%		
	V			
	ZN		Non-normal	No
SWMU 7	AG	> 15%	Non-normal	No
	AL			
	AS			No
	BA			No
	BE			
	CA			No
	CO			
	CR			
	CU			
	FE			
	K			
	MG			
	MN			No
	NA			No
	NI			No
	PB			No
	SB	> 15%		No
	TL	> 15%		
	V			
	ZN			
SWMU 9	AG	> 15%	Non-normal	No
	AL			No
	AS			No
	BA			No
	BE			No
	CA			No
	CD	> 15%		
	CO	> 15%		No
	CR			No

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	CU			
	FE			No
	K			No
	MG			No
	MN			No
	NA			No
	NI			
	PB			No
	TL	> 15%		No
	V			
	ZN			No
SWMU 14	AL			No
	AS			No
	BA			No
	BE			No
	CA			No
	CO			No
	CR			No
	CU			No
	FE			No
	K			No
	MG			No
	MN			No
	NA			No
	NI			No
	PB			No
	TL			No
	V			No
	ZN			No
SWMU 20	AL			No
	AS			No
	BA			No
	BE	> 15%		No
	CA			
	CO	> 15%		
	CR			No
	CU			

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	FE			No
	K			No
	MG			No
	MN			No
	NA			
	NI			No
	PB			No
	SB	> 15%		No
	TL	> 15%		No
	V			
	ZN			No
SWMU 33	AG	> 15%		
	AL			
	AS			No
	BA			
	BE	> 15%		
	CA			No
	CD	> 15%	Non-normal	No
	CO			
	CR		Non-normal	No
	CU		Non-normal	No
	FE			
	HG	> 15%	Non-normal	No
	K			
	MG			No
	MN			
	NA			
	NI			
	PB		Non-normal	No
	SB	> 15%		
	SE	> 15%	Non-normal	
	TL	> 15%		No
	V			
	ZN		Non-normal	No
SWMU 34	AL			
	AS	> 15%		
	BA			No

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	BE			No
	CA			No
	CO			No
	CR			
	CU			
	FE			
	HG	> 15%		
	K			
	MG			No
	MN			No
	NA			No
	NI			
	PB		Non-normal	No
	SB	> 15%		
	SE	> 15%		
	TL	> 15%		
	V			
	ZN			
SWMU 36	AG	> 15%	Non-normal	No
	AL			No
	AS		Non-normal	No
	BA			No
	BE	> 15%		
	CA			No
	CD	> 15%	Non-normal	No
	CO			No
	CR		Non-normal	
	CU		Non-normal	No
	FE			No
	HG	> 15%	Non-normal	No
	K			
	MG			No
	MN			No
	NA			
	NI			No
	PB		Non-normal	No
	SB	> 15%		
	SE	> 15%	Non-normal	No
	TL	> 15%	Non-normal	No
	V			
	ZN		Non-normal	No
SWMU 37	AG	> 15%		No
	AL			
	AS			
	BA			
	BE			
	CA			
	CD	> 15%	Non-normal	No
	CO			No
	CR			
	CU		Non-normal	
	FE		Non-normal	No
	HG	> 15%	Non-normal	No
	K			
	MG			No
	MN		Non-normal	No
	NA			
	NI			
	PB		Non-normal	No
	SB	> 15%		No
	SE	> 15%	Non-normal	No
	TL	> 15%	Non-normal	
	V			
	ZN		Non-normal	
SWMU 38	AL			No
	AS			
	BA			
	BE	> 15%		
	CA			
	CO			No
	CR			No
	CU			No
	FE			No
	HG	> 15%	Non-normal	No

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	K			No
	MG			No
	MN			
	NA			No
	NI			No
	PB			
	SB	> 15%		No
	SE	> 15%		
	TL			No
	V			No
	ZN			No
SWMU 39	AG	> 15%	Non-normal	No
	AL		Non-normal	No
	AS		Non-normal	No
	BA			
	BE	> 15%		
	CA			
	CD	> 15%	Non-normal	
	CO	> 15%	Non-normal	No
	CR		Non-normal	No
	CU		Non-normal	No
	FE			No
	K			No
	MG			No
	MN			No
	NA			
	NI		Non-normal	No
	PB		Non-normal	No
	SB	> 15%		No
	SE	> 15%	Non-normal	No
	TL	> 15%	Non-normal	No
	V		Non-normal	No
	ZN			
SWMU 40	AL			No
	AS			
	BA			
	BE	> 15%		

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	CA			
	CD	> 15%		No
	CO			No
	CR			No
	CU			No
	FE			No
	K			No
	MG			
	MN			No
	NA		Non-normal	No
	NI			No
	PB			
	SB	> 15%		No
	SE	> 15%	Non-normal	
	TL	> 15%		
	V			No
	ZN		Non-normal	No
SWMU 42	AG	> 15%		No
	AL			
	AS			
	BA			No
	BE	> 15%		No
	CA			No
	CD	> 15%		No
	CO			
	CR			
	CU			
	FE			
	K			
	MG			No
	MN			
	NA			No
	NI			
	PB			
	SB	> 15%		No
	SE	> 15%		
	TL			

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	V			
	ZN			No
SWMU 43	AL			
	AS	> 15%		
	BA			No
	BE	> 15%		
	CA			
	CO			
	CR			
	CU			
	FE			
	HG	> 15%		No
	K			
	MG			No
	MN			
	NA			No
	NI			
	PB			
	SB	> 15%		No
	SE	> 15%		
	TL	> 15%		No
	V			
	ZN			
SWMU 46	AG	> 15%	Non-normal	No
	AL			No
	AS		Non-normal	No
	BA			
	BE	> 15%		No
	CA			
	CD	> 15%		No
	CO	> 15%		No
	CR			
	CU		Non-normal	
	FE			No
	HG	> 15%		No
	K			No
	MG			No
	MN		Non-normal	No
	NA			No
	NI			No
	PB	> 15%	Non-normal	No
	SB	> 15%		No
	SE	> 15%	Non-normal	No
	TL	> 15%	Non-normal	
	V			
	ZN		Non-normal	No
SWMU 48	AL			No
	AS			No
	BA			No
	BE	> 15%		No
	CA			No
	CD	> 15%		No
	CO	> 15%		No
	CR			
	CU		Non-normal	
	FE			No
	HG	> 15%		No
	K			No
	MG			
	MN			No
	NA			No
	NI			No
	PB		Non-normal	No
	SB	> 15%		No
	SE	> 15%		
	TL	> 15%		
	V			
	ZN			No
SWMU 51	AL			
	AS			
	BA			
	BE			No
	CA			No
	CD	> 15%		No

Table A-6 Parametric Test Criteria for Background Metals and Metals Detected in Soil at Dugway Proving Ground

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	CO			
	CR			
	CU			No
	FE			No
	K			No
	MG			No
	MN			
	NA			
	NI			No
	PB			
	SB	> 15%		
	SE	> 15%		
	TL	> 15%		
	V			
	ZN			No
SWMU 55	AL			
	AS			
	BA			
	BE	> 15%		No
	CA			
	CD	> 15%		No
	CO			
	CR			
	CU			No
	FE			No
	HG	> 15%		No
	K			
	MG			No
	MN			No
	NA			No
	NI			
	PB			
	SB	> 15%		No
	SE	> 15%		No
	TL	> 15%		
	V			
	ZN			

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
SWMU 58	AL			
	AS			
	BA			
	BE	> 15%		
	CA			
	CD	> 15%		No
	CO			
	CR			
	CU			
	FE			
	K			
	MG			
	MN			
	NA			
	NI			
	PB			
	SB	> 15%		
	SE	> 15%		
	TL	> 15%		No
	V			
	ZN			
SWMU 59	AL			
	AS			
	BA			
	BE	> 15%		
	CA			No
	CO			No
	CR			
	CU			No
	FE			No
	HG	> 15%		No
	K			No
	MG			No
	MN			No
	NA			No
	NI			
	PB			

Table A-6 Parametric Test Criteria for Background Metals and Metals Detected in Soil at Dugway Proving Ground

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	SB	> 15%		
	SE	> 15%		No
	TL	> 15%		No
	V			No
	ZN			No
SWMU 63-1	AL			No
	AS			
	BA			
	BE	> 15%		
	CA			No
	CO			No
	CR			No
	CU			No
	FE			No
	HG	> 15%		No
	K			No
	MG			No
	MN			No
	NA			No
	NI			No
	PB			
	SB	> 15%		
	SE	> 15%		No
	TL	> 15%		
	V			No
	ZN			No
SWMU 63-1	AL			
	AS			No
	BA			No
	BE			No
	CA			No
	CO			No
	CR			
	CU			No
	FE			No
	K			No
	MG			No
	MN			No
	NA			No
	NI			No
	PB			No
	SB	> 15%		No
	SE	> 15%	Non-normal	No
	TL	> 15%		
	V			
	ZN		Non-normal	
SWMU 99	AL			No
	AS			No
	BA			No
	BE	> 15%		No
	CA			
	CO	> 15%		No
	MN			No
	NA			No
	NI			No
	PB			
	SB	> 15%		No
	TL	> 15%		No
	V			
	ZN			No
SWMU 90	AG	> 15%	Non-normal	
	AL			
	AS		Non-normal	No
	BA			
	BE	> 15%		No
	CA			No
	CD	> 15%	Non-normal	No
	CO			No
	CR			No
	CU			
	FE			No
	HG	> 15%	Non-normal	No
	K			No
	MG			No
	MN			No
	NA			No
	NI			No
	PB		Non-normal	No
	SB	> 15%		No
	SE	> 15%	Non-normal	No
	TL	> 15%		
	V			
	ZN		Non-normal	
SWMU 99	AL			No
	AS			No
	BA			No
	BE	> 15%		No
	CA			
	CO	> 15%		No

Table A-6 Parametric Test Criteria for Background Metals and Metals Detected in Soil at Dugway Proving Ground

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	CR			No
	CU			No
	FE			No
	K			No
	MG			
	MN			No
	NA			No
	NI			No
	PB		Non-normal	No
	SB	> 15%		No
	SE	> 15%	Non-normal	No
	TL	> 15%		
	V			
	ZN			No
SWMU 124	AL			
	AS			
	BA			
	BE	> 15%		
	CA			No
	CD	> 15%		
	CO	> 15%		
	CR			
	CU			
	FE			
	K			
	MG			
	MN			
	NA		Non-normal	No
	NI			
	PB			
	SB	> 15%		No
	TL	> 15%		
	V			
	ZN			
SWMU 128	AG	> 15%	Non-normal	
	AL			No
	AS		Non-normal	No

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	BA			
	BE	> 15%	Non-normal	No
	CA			
	CD	> 15%		No
	CO	> 15%		
	CR			
	CU			
	FE			
	HG	> 15%		No
	K			
	MG			No
	MN			
	NA			No
	NI			No
	PB			
	SB	> 15%		No
	SE	> 15%		No
	TL	> 15%		No
	V			No
	ZN			
SWMU 130	AL			No
	AS			
	BA			
	BE	> 15%		No
	CA			No
	CD	> 15%		No
	CO	> 15%		
	CR			
	CU			
	FE			No
	HG	> 15%	Non-normal	No
	K			
	MG			No
	MN			
	NA			No
	NI	> 15%		
	PB		Non-normal	No

Table A-6 Parametric Test Criteria for Background Metals and Metals Detected in Soil at Dugway Proving Ground

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	SE	> 15%		
	TL	> 15%		
	V			
	ZN			No
SWMU 158	AL			No
	AS			
	BA			
	BE			No
	CA			
	CO			
	CR			No
	CU			
	FE			No
	K			
	MG			
	MN			No
	NA			No
	NI			
	PB			
	SB	> 15%		No
	SE	> 15%	Non-normal	
	TL			
	V			
	ZN			No
SWMU 162	AL			No
	AS			No
	BA			
	BE	> 15%		No
	CA			
	CO			
	CR			
	CU			
	FE			No
	K			
	MG			No
	MN			
	NA			

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	NI			
	PB			
	TL			No
	V			
	ZN			No
SWMU 163	AL			
	AS			No
	BA			
	BE	> 15%		No
	CA			No
	CO	> 15%		
	CR			
	CU			No
	FE			No
	K			No
	MG			
	MN			
	NA			No
	NI			No
	PB			
	SB	> 15%		
	SE	> 15%		
	TL	> 15%		No
	V			
	ZN			No
SWMU 164	AL			
	AS			
	BA			
	BE	> 15%		
	CA			
	CD	> 15%	Non-normal	No
	CO			
	CR			
	CU			
	FE			
	HG	> 15%		No
	K			

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	MG			
	MN			
	NA			No
	NI			
	PB		Non-normal	No
	SB	> 15%		
	TL			
	V			
	ZN			No
	AL			
SWMU 165	AS			No
	BA			
	BE	> 15%		
	CA			No
	CD	> 15%	Non-normal	No
	CO			
	CR			
	CU			
	FE			
	K			No
	MG			No
	MN			
	NA			No
	NI			
	PB			
	SB	> 15%		
	SE	> 15%		No
	TL	> 15%		No
	V			
	ZN			No
SWMU 166	AL			
	AS			
	BA			
	BE	> 15%		
	CA			
	CD	> 15%	Non-normal	No
	CO	> 15%		

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	CR			
	CU			
	FE			
	K			
	MG			
	MN			
	NA			No
	NI			
	PB		Non-normal	No
	SB	> 15%		
	TL	> 15%		No
	V			
	ZN			No
SWMU 167	AL			
	AS			
	BA			
	BE	> 15%		No
	CA			No
	CD	> 15%	Non-normal	No
	CO			
	CR			
	CU			
	FE			
	HG	> 15%		No
	K			
	MG			No
	MN			
	NA			No
	NI			
	PB			
	SB	> 15%		
	SE	> 15%		No
	TL	> 15%		
	V			
	ZN			
SWMU 168	AG	> 15%	Non-normal	No
	AL			No

Table A-6 Parametric Test Criteria for Background Metals and Metals Detected in Soil at Dugway Proving Ground

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	AS			No
	BA			
	BE	> 15%		No
	CA			No
	CD	> 15%	Non-normal	No
	CO	> 15%		
	CR			No
	CU			
	FE			No
	HG	> 15%	Non-normal	No
	K			
	MG			
	MN			No
	NA			No
	NI			
	PB		Non-normal	No
	SB	> 15%		
	SE	> 15%	Non-normal	
	TL	> 15%		
	V			
	ZN		Non-normal	No
SWMU 169	AG	> 15%		No
	AL			
	AS			No
	BA			No
	BE	> 15%		
	CA			No
	CD	> 15%	Non-normal	No
	CO			
	CR			No
	CU		Non-normal	No
	FE			
	K			
	MG			No
	MN			
	NA			No
	NI			
	PB			No
	SB	> 15%		
	TL			No
	V			
	ZN		Non-normal	No
SWMU 170	AL			
	AS			No
	BA			
	BE	> 15%		
	CA			
	CO			
	CR			
	CU			
	FE			
	HG	> 15%		No
	K			
	MG			No
	MN			
	NA		Non-normal	
	NI			
	PB			No
	TL	> 15%		
	V			
	ZN			
SWMU 190	AL			No
	AS	> 15%		No
	BA			No
	CA			No
	CD	> 15%	Non-normal	No
	CO	> 15%		
	CR			
	CU			
	FE			No
	K			No
	MG			No
	MN			No
	NA			No

Table A-6 Parametric Test Criteria for Background Metals and Metals Detected in Soil at Dugway Proving Ground

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]
	NI	> 15%		No
	PB		Non-normal	No
	TL	> 15%		No
	V			No
	ZN		Non-normal	No

SWMU	Analyte	Percent of Nondetections [1]	Distribution [2]	Homogeneous Variances [3]

1 Parametric tests are appropriate if there are less than 15 percent nondetections (EPA 1992, Section 3.2). If there are greater than 15 percent nondetections nonparametric tests can be used.

2 Parametric tests can only be used when data have a normal or lognormal distribution. Data sets being compared *must have the same distribution* (EPA 1989, Section 5.2; EPA 1996, Section 3.3.1.1). Coefficient of variance used to make gross determination of non-normality (CV > 1). CV results are questionable for data sets with a high number of nondetections. Other methods are preferred for determining data distributions (EPA 1992, Section 1.1.1; EPA 1996, Section 4.2.4)

3 The parametric Student's t-test requires that the variances of the data sets (site and background) are homogeneous (EPA 1992, Section 1.2; EPA 1996, Section 3.3.1.1).

Table [redacted] Wilcoxon Rank-Sum Test Results for Subsurface (B) and Surface [redacted] Data

TESTNAME	N_B	N_S	ND_B	ND_S	N_GP	MRANK_B	MRANK_S	SUMCI	W	EW	SDW	SDW_AJT	Z_CAL	Z_95	Z_975	Z_99	REMARK	ZCALGTZ975	Difference?
AG																	NO HIT		
AL	67	19	0	0	13	44.5448	39.8158	756.500	566.50	636.50	96.06	96.041	-0.734	1.645	1.960	2.326			
AS	67	19	6	0	9	44.5522	39.7895	756.000	566.00	636.50	96.06	96.041	-0.739	1.645	1.960	2.326			
BA	67	19	0	0	11	42.4478	47.2105	897.000	707.00	636.50	96.06	96.047	0.729	1.645	1.960	2.326			
BE	67	19	30	7	6	43.3284	44.1053	838.000	648.00	636.50	96.06	95.005	0.115	1.645	1.960	2.326			
CA	67	19	0	0	14	44.4030	40.3158	766.000	576.00	636.50	96.06	95.984	-0.635	1.645	1.960	2.326			
CD	67	19	67	18	2	49.3134	23.0000	437.000	247.00	636.50	96.06	83.232	-4.060	1.645	1.960	2.326		Y	Y
CO	67	19	14	0	13	45.6119	36.0526	685.000	495.00	636.50	96.06	95.958	-1.478	1.645	1.960	2.326			
CR	67	19	0	0	13	45.4403	36.6579	696.500	506.50	636.50	96.06	96.046	-1.358	1.645	1.960	2.326			
CU	67	19	3	0	10	42.4627	47.1579	896.000	706.00	636.50	96.06	96.045	0.718	1.645	1.960	2.326			
CYN																	NO HIT		
FE	67	19	0	0	11	46.7313	32.1053	610.000	420.00	636.50	96.06	96.045	-2.259	1.645	1.960	2.326		Y	Y
HG																	NO HIT		
K	67	19	0	0	9	40.7836	53.0789	1008.50	818.50	636.50	96.06	96.064	1.889	1.645	1.960	2.326			
MG	67	19	0	0	13	41.1343	51.8421	985.000	795.00	636.50	96.06	96.015	1.645	1.645	1.960	2.326			
MN	67	19	0	0	11	41.8955	49.1579	934.000	744.00	636.50	96.06	96.039	1.114	1.645	1.960	2.326			
NA	67	19	0	0	8	48.9403	24.3158	462.000	272.00	636.50	96.06	96.061	-3.799	1.645	1.960	2.326		Y	Y
NI	67	19	6	0	10	43.4701	43.6053	828.500	638.50	636.50	96.06	96.042	0.016	1.645	1.960	2.326			
PB	64	16	0	0	8	39.2578	45.4688	727.500	591.50	512.00	83.13	83.130	0.950	1.645	1.960	2.326			
SB	20	19	16	17	2	20.9000	19.0526	362.000	172.00	190.00	35.59	22.346	-0.520	1.645	1.960	2.326			
SE																	NO HIT		
TL	20	19	8	6	6	19.6750	20.3421	386.500	196.50	190.00	35.59	34.746	0.169	1.645	1.960	2.326			
V	67	19	0	0	17	46.0970	34.3421	652.500	462.50	636.50	96.06	96.046	-1.816	1.645	1.960	2.326			
ZN	67	19	0	0	12	41.8657	49.2632	936.000	746.00	636.50	96.06	96.061	1.135	1.645	1.960	2.326			

Table A-8 Wilcoxon Rank-Sum Results for SWMU-Specific (S) and Background (B) Data

SWMU	SOILLAYER	TESTNAME	N_B	N_S	ND_B	ND_S	N_GP	MRANK_B	MRANK_S	SUMCI	W	EW	SDW	SDW_AJT	Z_CAL	Z_95	Z_975	Z_99	REMARK	ZCALGTZ95	MRSGTMRB	COC
020	POOLED	AG																	NO HIT			
020	POOLED	AL	86	18	0	0	19	58.6686	23.0278	414.500	243.500	774.000	116.383	116.359	-4.563	1.645	1.960	2.326				
020	POOLED	AS	86	18	6	0	17	50.2849	63.0833	1135.500	964.500	774.000	116.383	116.361	1.633	1.645	1.960	2.326			Y	
020	POOLED	BA	86	18	0	0	15	48.7965	70.1944	1263.500	1092.500	774.000	116.383	116.358	2.732	1.645	1.960	2.326		Y	Y	Y
020	POOLED	BE	86	18	37	14	6	53.7558	46.5000	837.000	666.000	774.000	116.383	114.556	-0.932	1.645	1.960	2.326				
020	POOLED	CA	86	18	0	0	17	46.5465	80.9444	1457.000	1286.000	774.000	116.383	116.279	4.395	1.645	1.960	2.326		Y	Y	Y
020	POOLED	CD																	NO HIT			
020	POOLED	CO	87	18	14	7	16	58.0862	28.4167	511.500	340.500	783.000	117.614	117.484	-3.767	1.645	1.960	2.326				
020	POOLED	CR	86	18	0	0	18	58.5814	23.4444	422.000	251.000	774.000	116.383	116.366	-4.498	1.645	1.960	2.326				
020	POOLED	CU	86	18	3	0	13	56.3837	33.9444	611.000	440.000	774.000	116.383	116.358	-2.874	1.645	1.960	2.326				
020	POOLED	CYN																	NO HIT			
020	POOLED	FE	86	18	0	0	15	59.1105	20.9167	376.500	205.500	774.000	116.383	116.364	-4.889	1.645	1.960	2.326				
020	POOLED	HG																	NO HIT			
020	POOLED	K	86	18	0	0	15	56.5581	33.1111	596.000	425.000	774.000	116.383	116.371	-3.003	1.645	1.960	2.326				
020	POOLED	MG	86	18	0	0	17	48.0291	73.8611	1329.500	1158.500	774.000	116.383	116.305	3.299	1.645	1.960	2.326		Y	Y	Y
020	POOLED	MN	87	18	0	0	18	55.5920	40.4722	728.500	557.500	783.000	117.614	117.579	-1.922	1.645	1.960	2.326				
020	POOLED	NA	86	18	0	0	15	53.1047	49.6111	893.000	722.000	774.000	116.383	116.374	-0.451	1.645	1.960	2.326				
020	POOLED	NI	86	18	6	0	14	56.1221	35.1944	633.500	462.500	774.000	116.383	116.362	-2.681	1.645	1.960	2.326				
020	POOLED	PB	86	18	0	0	19	56.3953	33.8889	610.000	439.000	774.000	116.383	116.372	-2.883	1.645	1.960	2.326				
020	POOLED	SB	39	18	33	7	2	24.6026	38.5278	693.500	522.500	351.000	58.249	47.121	2.936	1.645	1.960	2.326		Y	Y	Y
020	POOLED	SE																	NO HIT			
020	POOLED	TL	39	18	14	14	8	32.9231	20.5000	369.000	198.000	351.000	58.249	54.679	-2.635	1.645	1.960	2.326				
020	POOLED	V	86	18	0	0	19	57.7267	27.5278	495.500	324.500	774.000	116.383	116.350	-3.867	1.645	1.960	2.326				
020	POOLED	ZN	86	18	0	0	17	58.2733	24.9167	448.500	277.500	774.000	116.383	116.374	-4.270	1.645	1.960	2.326				
164	POOLED	AG																	NO HIT			
164	POOLED	AL	86	5	0	0	13	47.2267	24.9000	124.500	109.500	215.000	57.417	57.401	-1.846	1.645	1.960	2.326				
164	POOLED	AS	86	5	6	0	9	45.2151	59.5000	297.500	282.500	215.000	57.417	57.399	1.167	1.645	1.960	2.326			Y	
164	POOLED	BA	86	5	0	0	13	45.9128	47.5000	237.500	222.500	215.000	57.417	57.400	0.122	1.645	1.960	2.326			Y	
164	POOLED	BE	86	5	37	3	7	45.8779	48.1000	240.500	225.500	215.000	57.417	56.821	0.174	1.645	1.960	2.326			Y	
164	POOLED	CA	86	5	0	0	14	45.6628	51.8000	259.000	244.000	215.000	57.417	57.361	0.496	1.645	1.960	2.326			Y	
164	POOLED	CD	86	5	85	3	3	45.8372	48.8000	244.000	229.000	215.000	57.417	50.411	0.235	1.645	1.960	2.326			Y	
164	POOLED	CO	87	5	14	0	16	47.0345	37.2000	186.000	171.000	217.500	58.062	58.007	-0.809	1.645	1.960	2.326				
164	POOLED	CR	86	5	0	0	13	44.9477	64.1000	320.500	305.500	215.000	57.417	57.403	1.567	1.645	1.960	2.326			Y	
164	POOLED	CU	86	5	3	0	11	44.9535	64.0000	320.000	305.000	215.000	57.417	57.399	1.559	1.645	1.960	2.326			Y	
164	POOLED	CYN																	NO HIT			
164	POOLED	FE	86	5	0	0	12	46.9477	29.7000	148.500	133.500	215.000	57.417	57.402	-1.428	1.645	1.960	2.326				
164	POOLED	HG	84	5	83	4	2	45.8393	30.9000	154.500	139.500	210.000	56.125	49.289	-1.265	1.645	1.960	2.326				
164	POOLED	K	86	5	0	0	12	46.8779	30.9000	154.500	139.500	215.000	57.417	57.411	-1.324	1.645	1.960	2.326				
164	POOLED	MG	86	5	0	0	13	45.5349	54.0000	270.000	255.000	215.000	57.417	57.385	0.688	1.645	1.960	2.326			Y	
164	POOLED	MN	87	5	0	0	12	46.8046	41.2000	206.000	191.000	217.500	58.062	58.046	-0.465	1.645	1.960	2.326				
164	POOLED	NA	86	5	0	0	8	47.7791	15.4000	77.000	62.000	215.000	57.417	57.412	-2.673	1.645	1.960	2.326				
164	POOLED	NI	86	5	6	0	14	45.5756	53.3000	266.500	251.500	215.000	57.417	57.402	0.627	1.645	1.960	2.326			Y	
164	POOLED	PB	86	5	0	0	9	44.4128	73.3000	366.500	351.500	215.000	57.417	57.410	2.369	1.645	1.960	2.326		Y	Y	Y
164	POOLED	SB	39	5	33	4	2	21.8846	27.3000	136.500	121.500	97.500	27.042	18.188	0.869	1.645	1.960	2.326			Y	
164	POOLED	SE																	NO HIT			
164	POOLED	TL	39	5	14	0	8	21.8718	27.4000	137.000	122.000	97.500	27.042	26.595	0.888	1.645	1.960	2.326			Y	
164	POOLED	V	86	5	0	0	18	46.0116	45.8000	229.000	214.000	215.000	57.417	57.401	-0.026	1.645	1.960	2.326				
164	POOLED	ZN	86	5	0	0	15	45.1163	61.2000	306.000	291.000	215.000	57.417	57.412	1.315	1.645	1.960	2.326			Y	

SWMU	SOILLAYER	TESTNAME	N_B	N_S	ND_B	ND_S	N_GP	MRANK_B	MRANK_S	SUMCI	W	EW	SDW	SDW_AJT	Z_CAL	Z_95	Z_975	Z_99	REMARK	ZCALGTZ95	MRSGTMRB	COC
166	POOLED	AG																	NO HIT			
166	POOLED	AL	86	4	0	0	14	46.4826	24.3750	97.500	87.500	172.000	51.075	51.061	-1.664	1.645	1.960	2.326				
166	POOLED	AS	86	4	6	0	10	44.4593	67.8750	271.500	261.500	172.000	51.075	51.061	1.743	1.645	1.960	2.326		Y	Y	Y
166	POOLED	BA	86	4	0	0	13	44.3488	70.2500	281.000	271.000	172.000	51.075	51.065	1.929	1.645	1.960	2.326		Y	Y	Y
166	POOLED	BE	86	4	37	1	8	45.0930	54.2500	217.000	207.000	172.000	51.075	50.556	0.675	1.645	1.960	2.326			Y	
166	POOLED	CA	86	4	0	0	14	44.3198	70.8750	283.500	273.500	172.000	51.075	51.029	1.977	1.645	1.960	2.326		Y	Y	Y
166	POOLED	CD	86	4	85	3	2	45.6163	43.0000	172.000	162.000	172.000	51.075	44.815	-0.206	1.645	1.960	2.326				
166	POOLED	CO	87	4	14	1	15	46.2931	39.6250	158.500	148.500	174.000	51.653	51.601	-0.503	1.645	1.960	2.326				
166	POOLED	CR	86	4	0	0	14	45.6628	42.0000	168.000	158.000	172.000	51.075	51.062	-0.284	1.645	1.960	2.326				
166	POOLED	CU	86	4	3	0	12	45.1163	53.7500	215.000	205.000	172.000	51.075	51.061	0.636	1.645	1.960	2.326			Y	
166	POOLED	CYN																	NO HIT			
166	POOLED	FE	86	4	0	0	12	46.0407	33.8750	135.500	125.500	172.000	51.075	51.064	-0.920	1.645	1.960	2.326				
166	POOLED	HG																	NO HIT			
166	POOLED	K	86	4	0	0	11	46.2151	30.1250	120.500	110.500	172.000	51.075	51.072	-1.214	1.645	1.960	2.326				
166	POOLED	MG	86	4	0	0	14	44.8256	60.0000	240.000	230.000	172.000	51.075	51.049	1.126	1.645	1.960	2.326			Y	
166	POOLED	MN	87	4	0	0	11	45.8908	48.3750	193.500	183.500	174.000	51.653	51.638	0.174	1.645	1.960	2.326			Y	
166	POOLED	NA	86	4	0	0	9	47.1919	9.1250	36.500	26.500	172.000	51.075	51.071	-2.859	1.645	1.960	2.326				
166	POOLED	NI	86	4	6	0	10	45.3023	49.7500	199.000	189.000	172.000	51.075	51.063	0.323	1.645	1.960	2.326			Y	
166	POOLED	PB	86	4	0	0	10	45.2267	51.3750	205.500	195.500	172.000	51.075	51.070	0.450	1.645	1.960	2.326			Y	
166	POOLED	SB	39	4	33	2	3	20.8205	33.5000	134.000	124.000	78.000	23.917	17.007	1.902	1.645	1.960	2.326		Y	Y	Y
166	POOLED	SE																	NO HIT			
166	POOLED	TL	39	4	14	2	6	22.9231	13.0000	52.000	42.000	78.000	23.917	23.287	-1.526	1.645	1.960	2.326				
166	POOLED	V	86	4	0	0	19	45.7733	39.6250	158.500	148.500	172.000	51.075	51.062	-0.470	1.645	1.960	2.326				
166	POOLED	ZN	86	4	0	0	13	44.9128	58.1250	232.500	222.500	172.000	51.075	51.071	0.979	1.645	1.960	2.326			Y	
170	POOLED	AG																	NO HIT			
170	POOLED	AL	86	6	0	0	15	46.7558	42.8333	257.000	236.000	258.000	63.238	63.220	-0.356	1.645	1.960	2.326				
170	POOLED	AS	86	6	6	0	11	46.3023	49.3333	296.000	275.000	258.000	63.238	63.222	0.261	1.645	1.960	2.326			Y	
170	POOLED	BA	86	6	0	0	12	45.3256	63.3333	380.000	359.000	258.000	63.238	63.221	1.589	1.645	1.960	2.326			Y	
170	POOLED	BE	86	6	37	3	8	46.6105	44.9167	269.500	248.500	258.000	63.238	62.603	-0.158	1.645	1.960	2.326				
170	POOLED	CA	86	6	0	0	16	46.2209	50.5000	303.000	282.000	258.000	63.238	63.184	0.372	1.645	1.960	2.326			Y	
170	POOLED	CD																	NO HIT			
170	POOLED	CO	87	6	14	0	15	46.7184	51.0833	306.500	285.500	261.000	63.945	63.885	0.375	1.645	1.960	2.326			Y	
170	POOLED	CR	86	6	0	0	13	44.8256	70.5000	423.000	402.000	258.000	63.238	63.215	2.269	1.645	1.960	2.326		Y	Y	Y
170	POOLED	CU	86	6	3	0	12	45.3430	63.0833	378.500	357.500	258.000	63.238	63.215	1.566	1.645	1.960	2.326			Y	
170	POOLED	CYN																	NO HIT			
170	POOLED	FE	86	6	0	0	12	46.1860	51.0000	306.000	285.000	258.000	63.238	63.217	0.419	1.645	1.960	2.326			Y	
170	POOLED	HG	84	8	83	7	2	48.1786	28.8750	231.000	195.000	336.000	72.166	63.448	-1.961	1.645	1.960	2.326				
170	POOLED	K	86	6	0	0	12	46.2151	50.5833	303.500	282.500	258.000	63.238	63.232	0.380	1.645	1.960	2.326			Y	
170	POOLED	MG	86	6	0	0	15	47.4477	32.9167	197.500	176.500	258.000	63.238	63.196	-1.297	1.645	1.960	2.326				
170	POOLED	MN	87	6	0	0	12	46.7701	50.3333	302.000	281.000	261.000	63.945	63.921	0.305	1.645	1.960	2.326			Y	
170	POOLED	NA	86	6	0	0	9	47.2849	35.2500	211.500	190.500	258.000	63.238	63.230	-1.075	1.645	1.960	2.326				
170	POOLED	NI	86	6	6	0	10	45.4651	61.3333	368.000	347.000	258.000	63.238	63.217	1.399	1.645	1.960	2.326			Y	
170	POOLED	PB	86	6	0	0	10	44.8256	70.5000	423.000	402.000	258.000	63.238	63.228	2.269	1.645	1.960	2.326		Y	Y	Y
170	POOLED	SB																	NO HIT			

Table Heading	Explanation
SWMU	SWMU
SOILLAYER	Surface, subsurface or pooled (surface+subsurface) soil
TESTNAME	Analyte
N_B	Number of background or subsurface soil samples
N_S	Number of SWMU-specific or surface soil samples
ND_B	Number of nondetections for background or subsurface soil samples
ND_S	Number of nondetections for SWMU-specific or surface soil samples
N_GP	Number of groups containing unique values
MRANK_B	Mean rank for population B
MRANK_S	Mean rank for population S
SUMCI	Sum of ranks for B and S
W	W statistic
EW	Expected value for W statistic
SDW	Standard deviation of W
SDW_AJT	Adjusted standard deviation of W
Z_CAL	Calculated Z statistic
Z_95	Z statistic at a 95% confidence
ZCALGTZ95	Is calculated Z statistic greater than the Z statistic at a 95% confidence?
MRSGTMRB	Is the mean rank of population S greater than the mean rank of population B?
Contaminant?	Does the result indicate SWMU-specific contamination?
Difference?	Is there a statistical difference between surface and subsurface background data?

Table A-10 Summary of Results and Upper Tolerance Values for the Background Soil Data Set

Analyte	Number of Samples	Number of Detections	Percent Detections	Minimum Detected Concentration (µg/g)	Maximum Detected Concentration (µg/g)	Location of Maximum Concentration	Arithmetic Mean	Standard Deviation	Background Value 95% Upper Tolerance Limit (µg/g)
AG	86	0	0	ND	ND	- -	0.32	0.086	0.94 RL
AL	86	86	100	2300	27000	DTO999SB03	11000	4700	19,000
AS	85	79	93	1.3	21	046BG01	6.5	3.7	13
BA	86	86	100	19	630	046BG01	210	110	400
BE	86	49	57	0.5	1.1	TWR017MW01	0.53	0.3	1
CA	86	86	100	2300	240000	BKR999SB02	100000	51000	190,000
CD	86	1	1	0.68	0.68	124BG01	0.44	0.19	0.46 RL
CO	86	72	84	2.2	17	EGL075SB04	4.1	2.3	7.9
CR	86	86	100	2.9	20	DTO999SB03	10	3.9	17
CU	86	83	97	1.9	69	036BG01	11	8.2	25
FE	85	85	100	3400	28000	EGL075SB04	11000	4600	19,000
HG	84	1	1	0.061	0.061	EGL069MW03	0.039	0.003	0.073 RL
K	86	86	100	640	10000	APG999SB03	3900	2100	7,400
MG	86	86	100	2000	33000	165BG01	16000	8000	29,000
MN	86	86	100	47	1100	APG999SB02	280	140	520
NA	86	86	100	460	15000	DTO999SB03	4600	3600	11,000
NI	86	80	93	3.0	36	EGL075SB04	8.6	5.2	17
PB	80	80	100	2.7	29	EGL075SB04	7.4	3.8	14
SB	39	6	15	6.1	9.1	059BG01	3.1	2	9.1 MAX
SE	63	4	6	0.61	2.9	EGL075SB04	0.29	0.42	2.9 MAX
TL	39	25	64	10	46	165BG01	16	11	35
V	86	86	100	9.0	57	EGL075SB04	20	6.9	29
ZN	86	86	100	9.3	70	EGL075SB04	35	15	59

µg/g - Micrograms per gram
RL - Method reporting limit
ND - Not detected
MAX - Maximum value
Analyte acronyms are defined in the Acronym List of the main report.

APPENDIX A

Figures

Recycled Paper

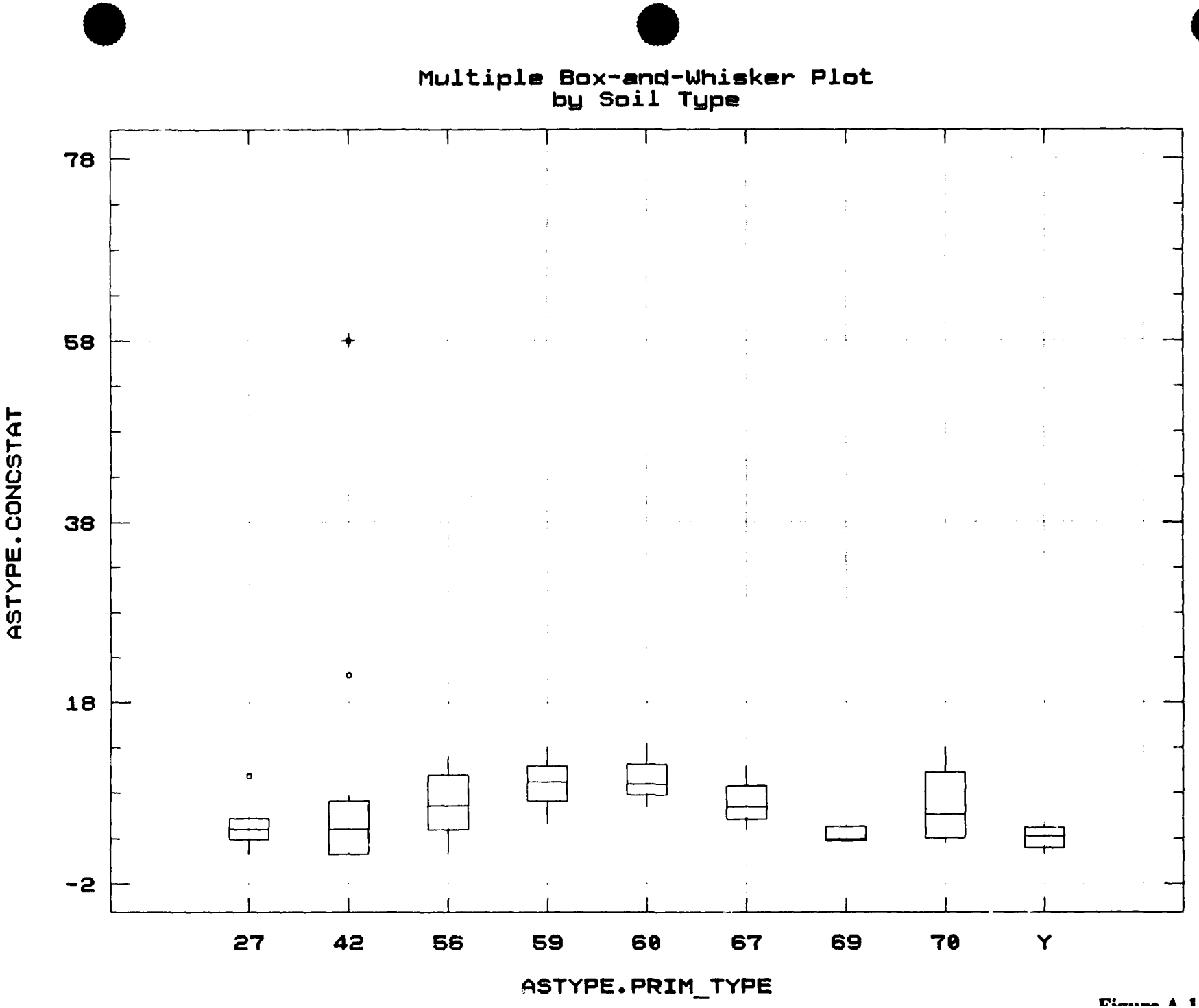

Multiple Box-and-Whisker Plot
by Soil Type
78
58
38
18
-2
ASTYPE.CONCSTAT
27
42
56
59
60
67
69
70
Y
ASTYPE.PRIM_TYPE

Figure A-1

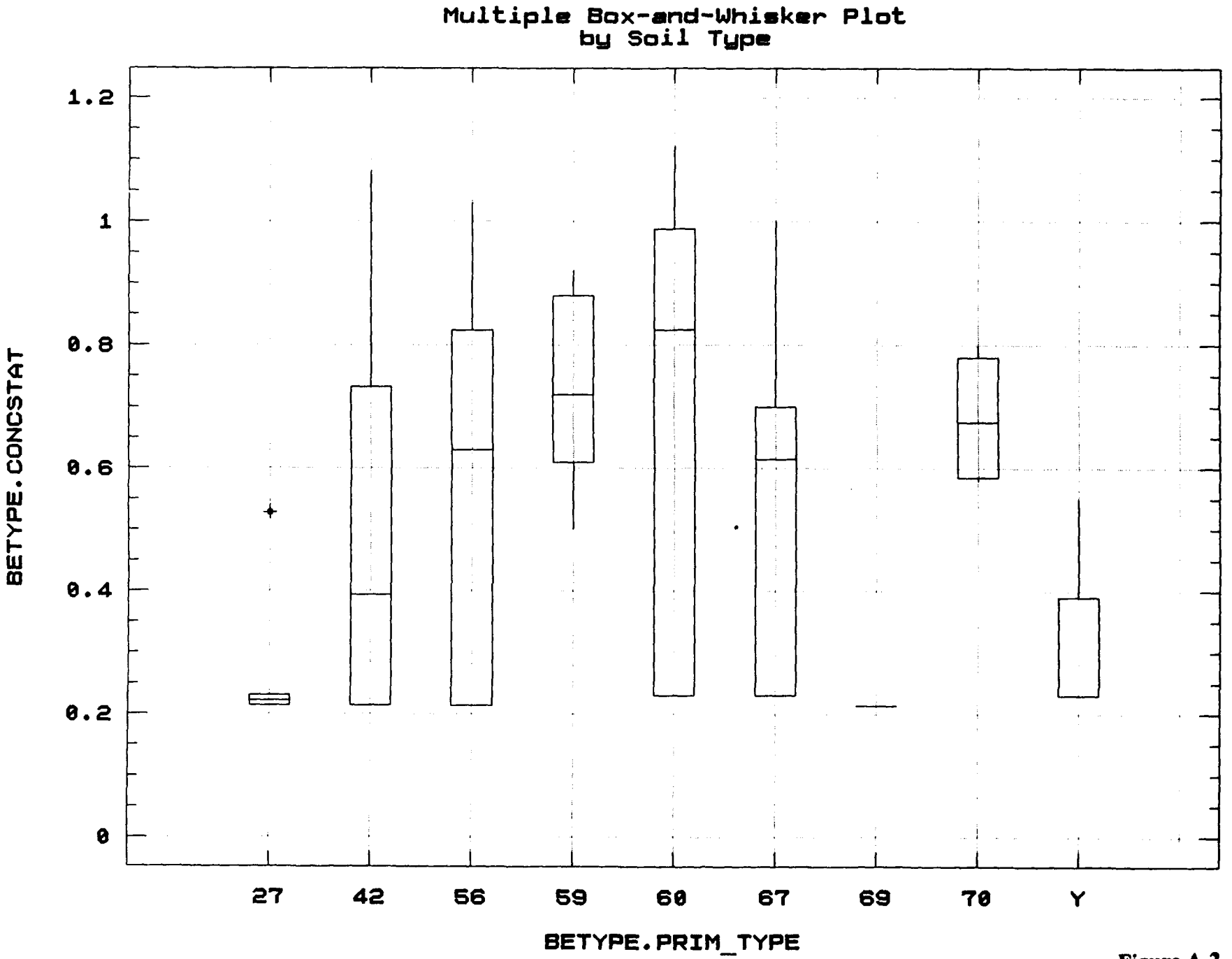
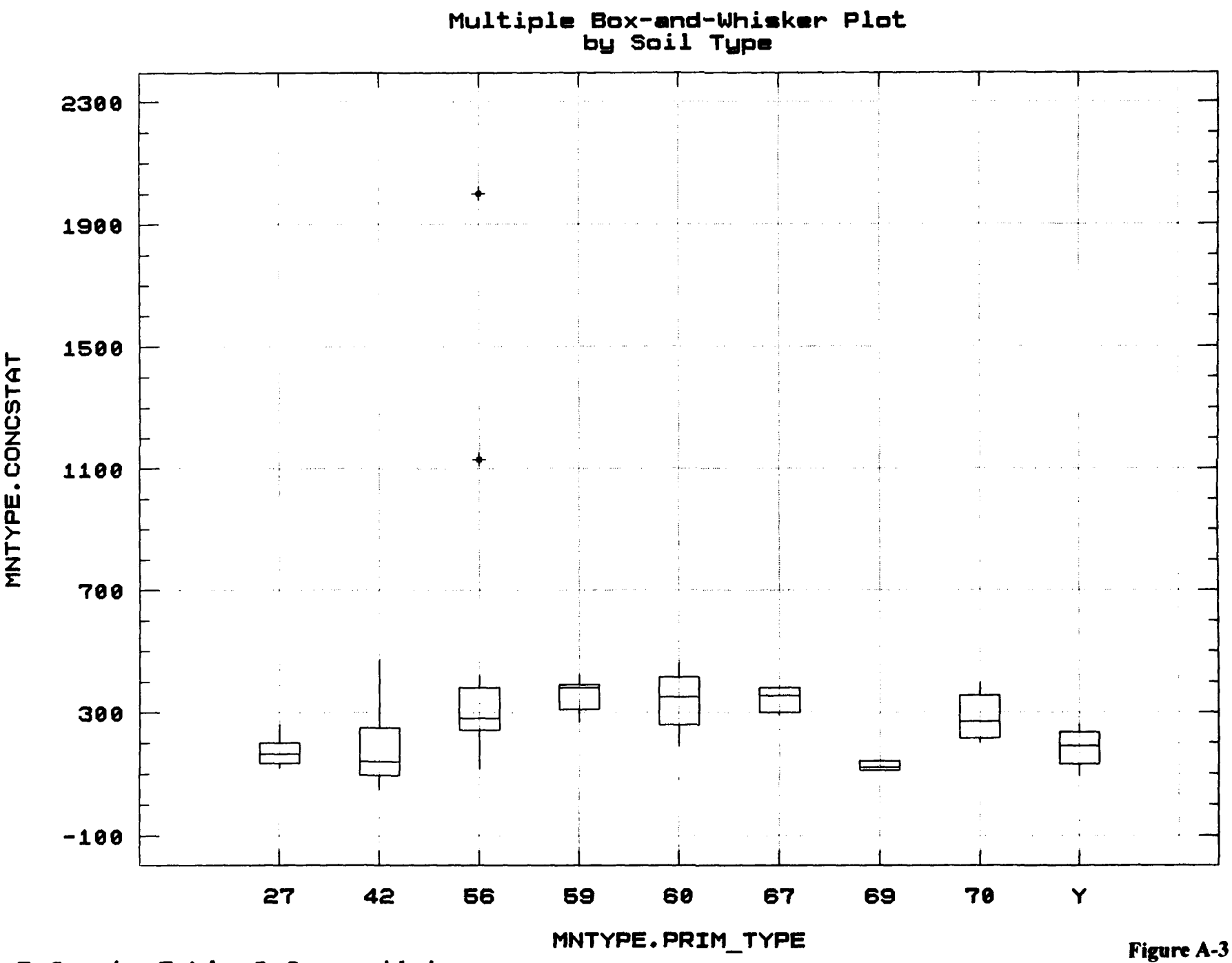
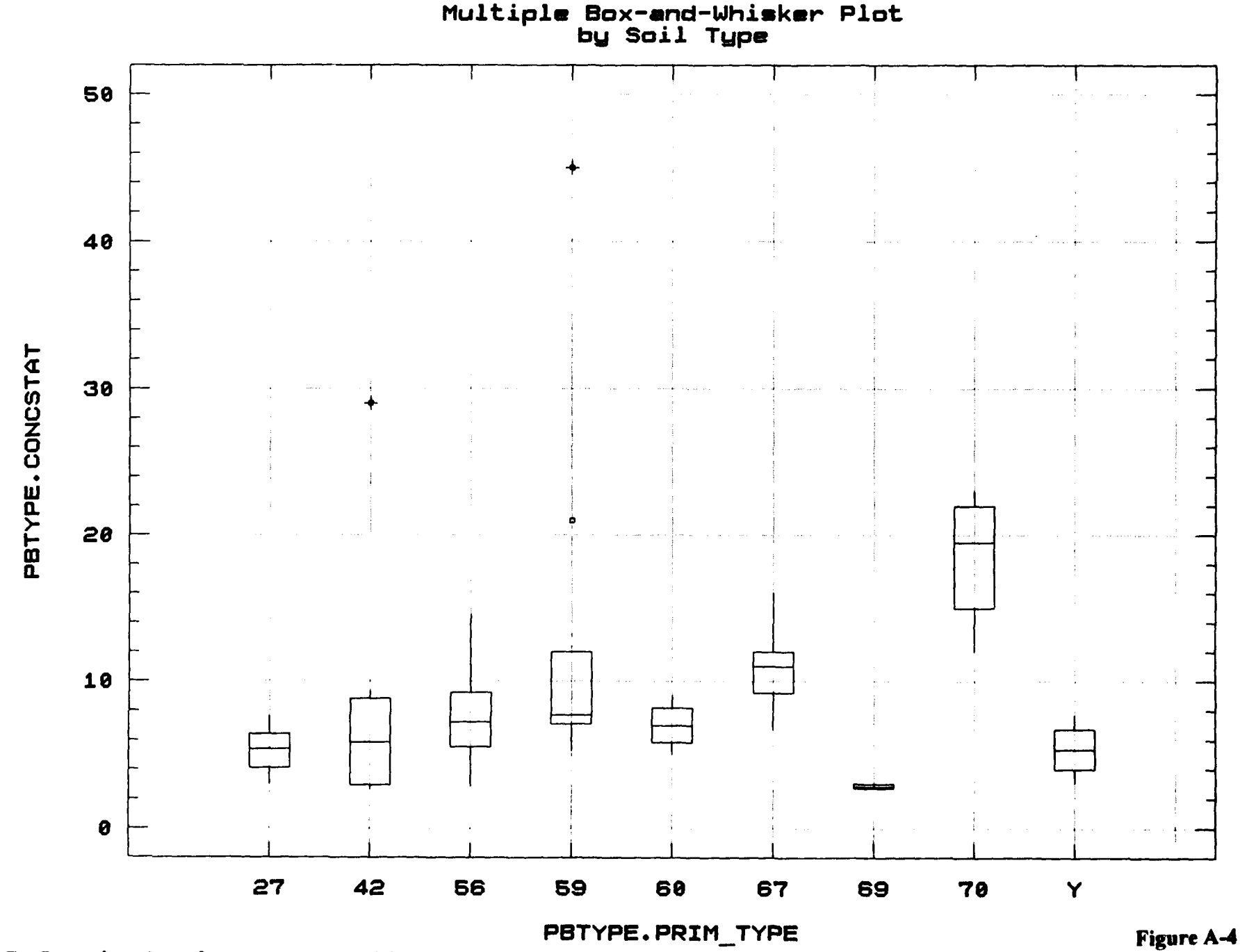

Multiple Box-and-Whisker Plot
by Soil Type
BETYPE.CONCSTAT
1.2
1
0.8
0.6
0.4
0.2
0
27
42
56
59
60
67
69
70
Y
BETYPE.PRIM_TYPE

Figure A-2



Refer to Table 2 for soil types

Figure A-3



Refer to Table 2 for soil types

Figure A-4

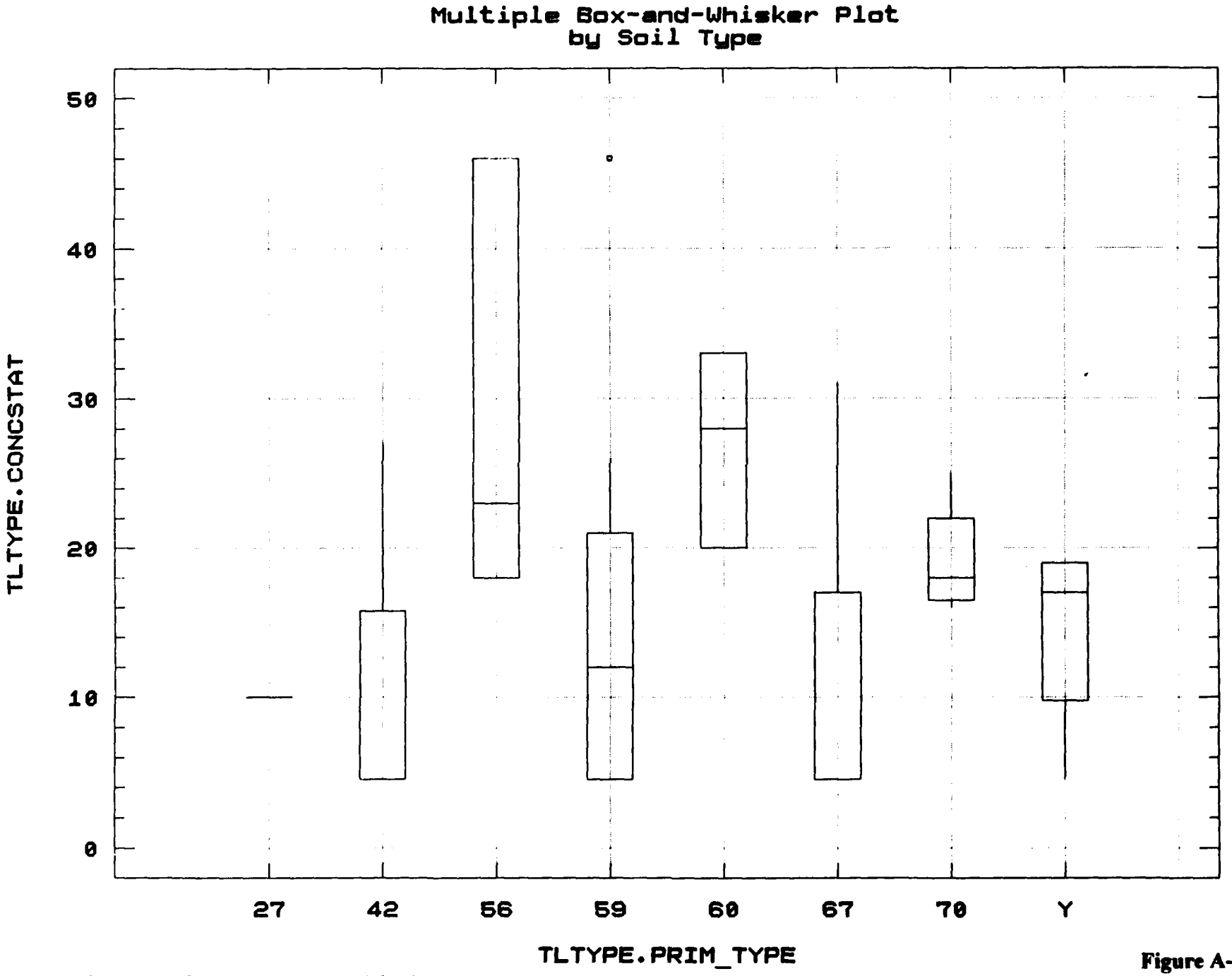
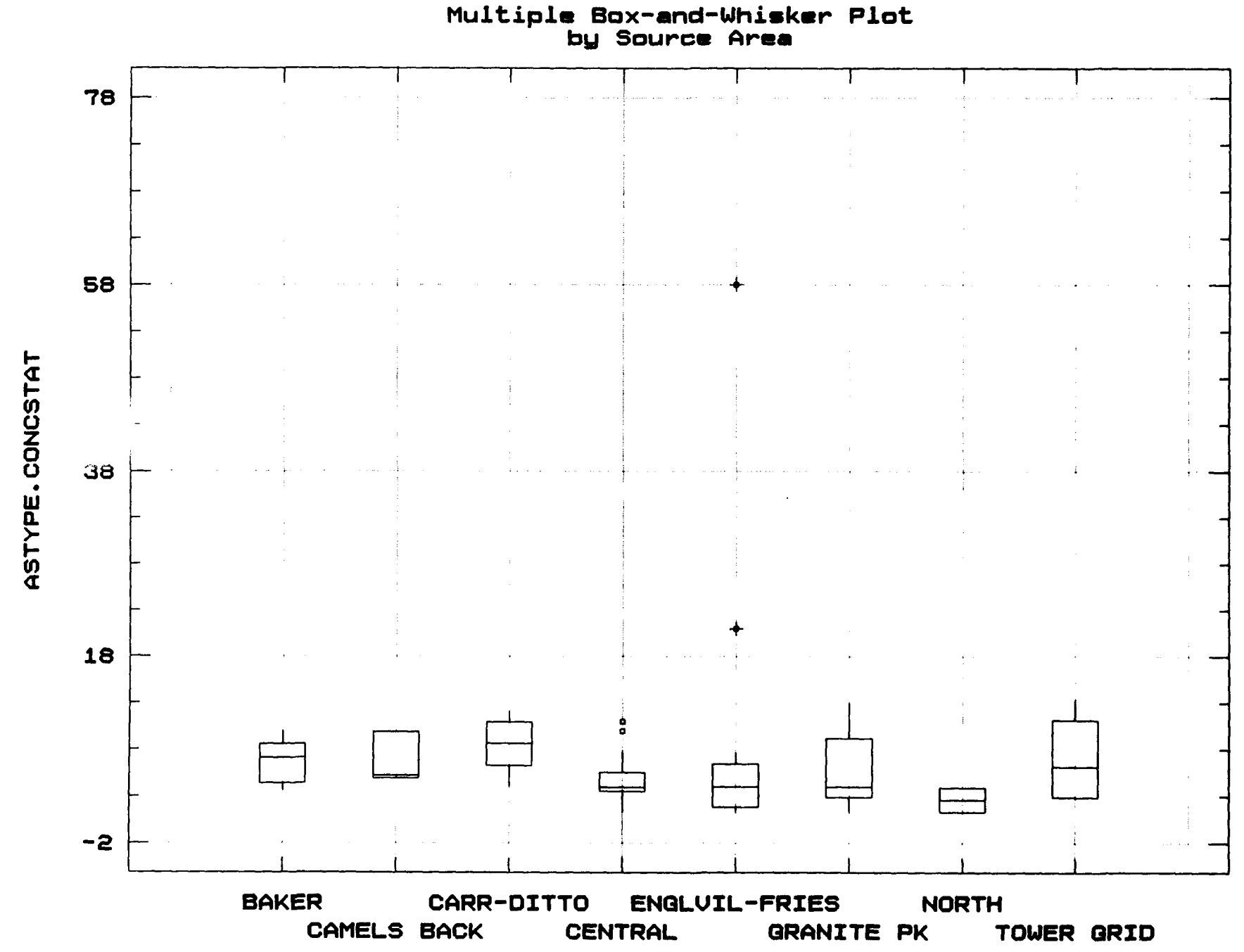


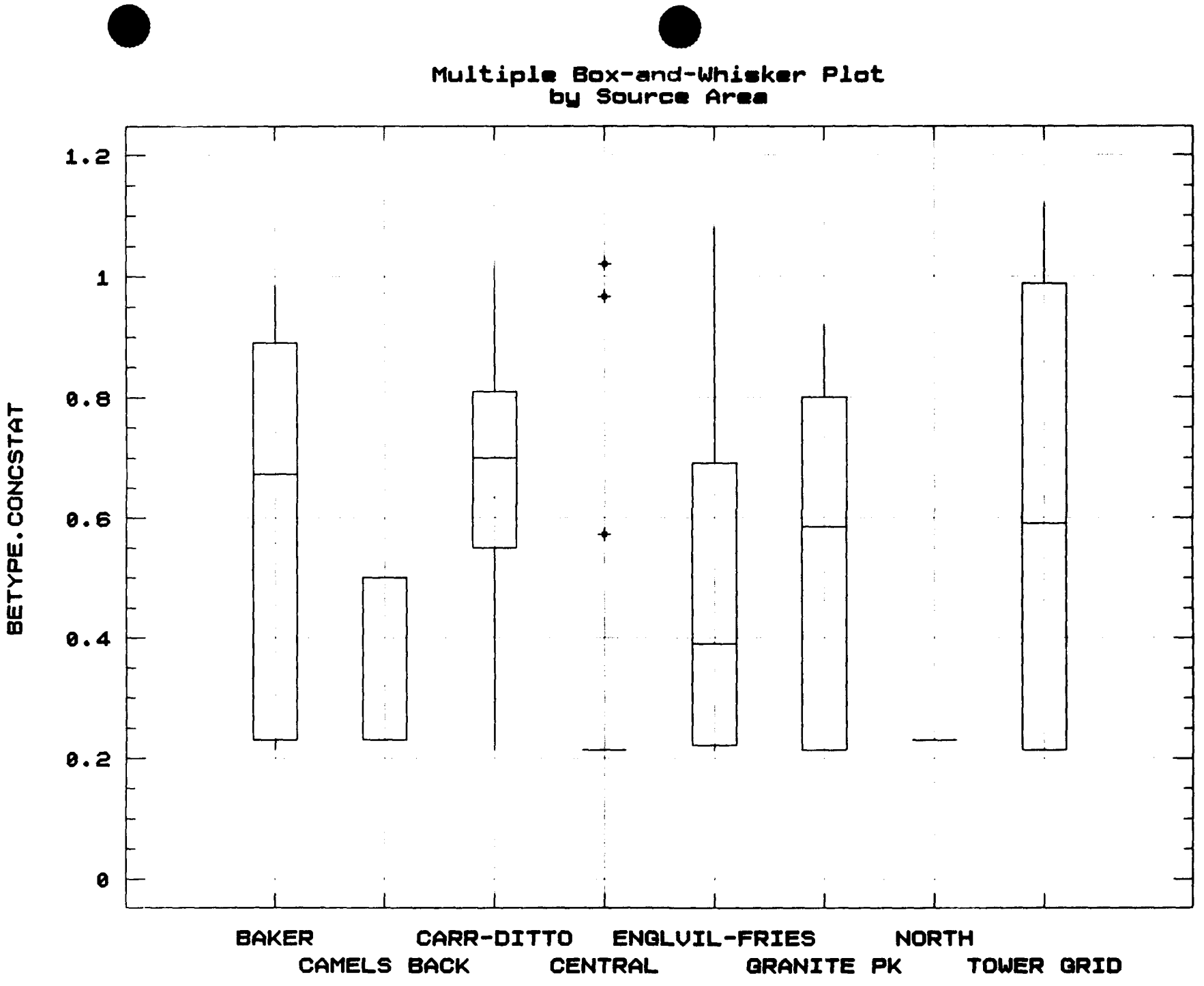
Figure A-5

Refer to Table 2 for soil types



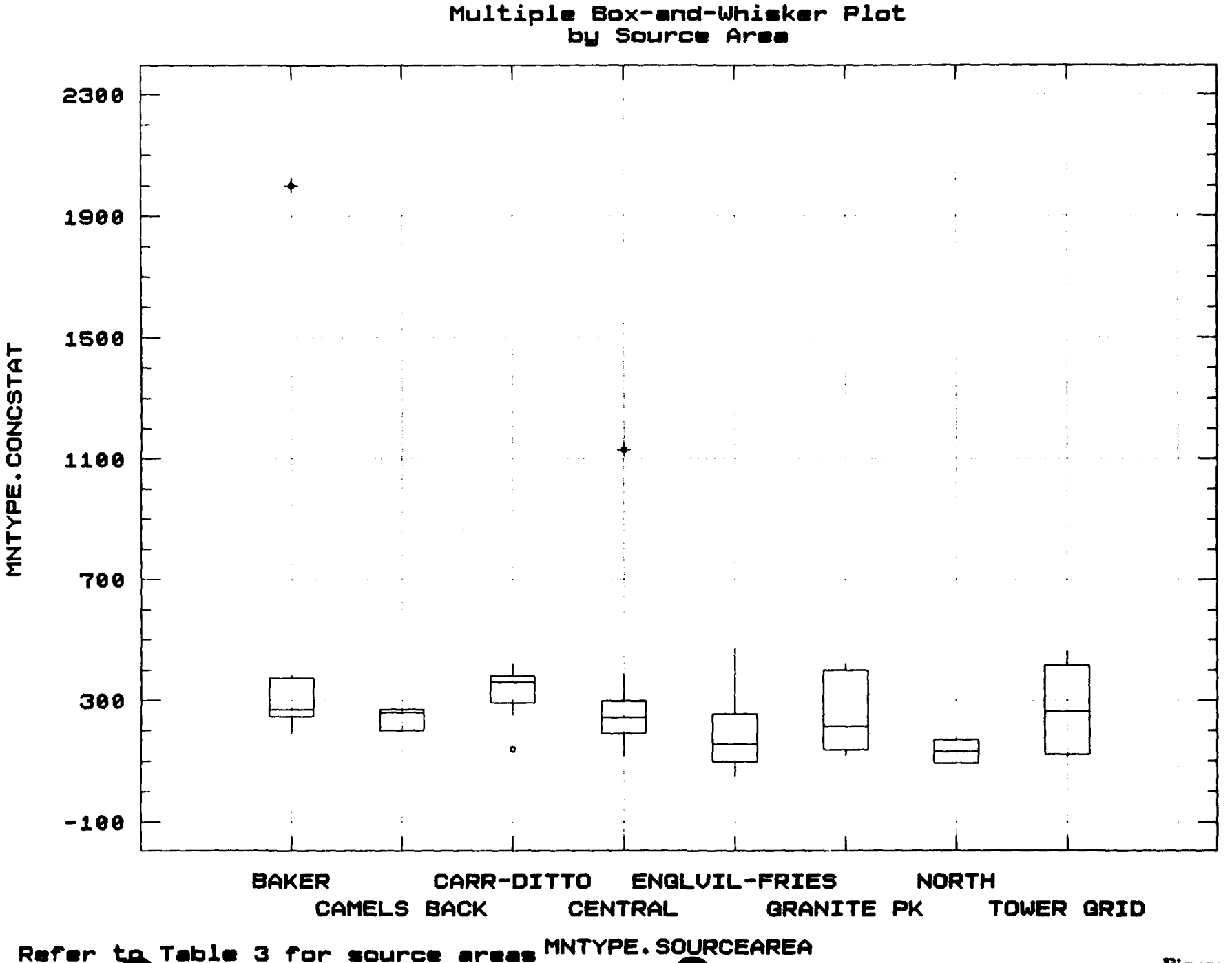
Refer to Table 3 for source areas

Figure A-



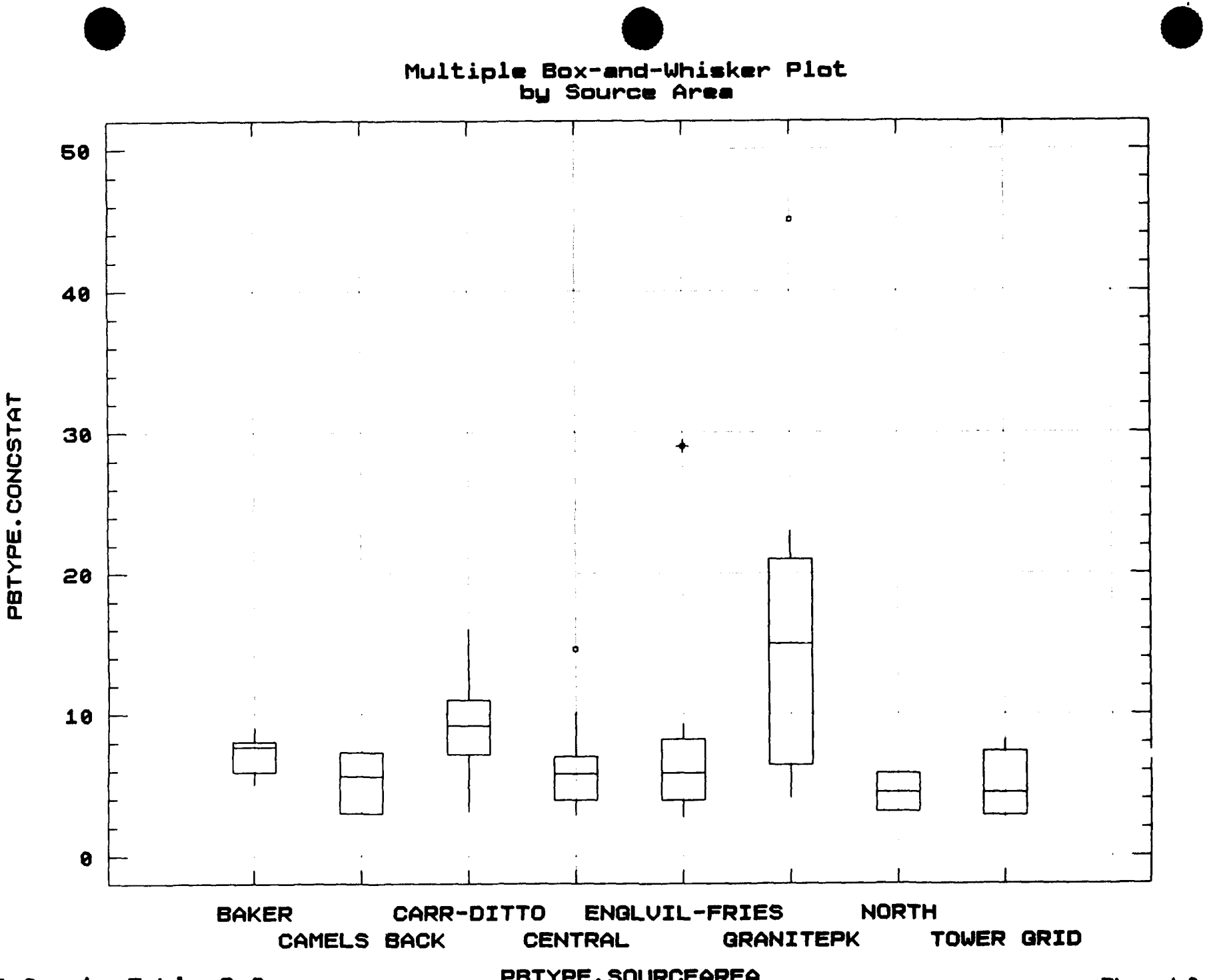
Refer to Table 3 for source areas

Figure A-7



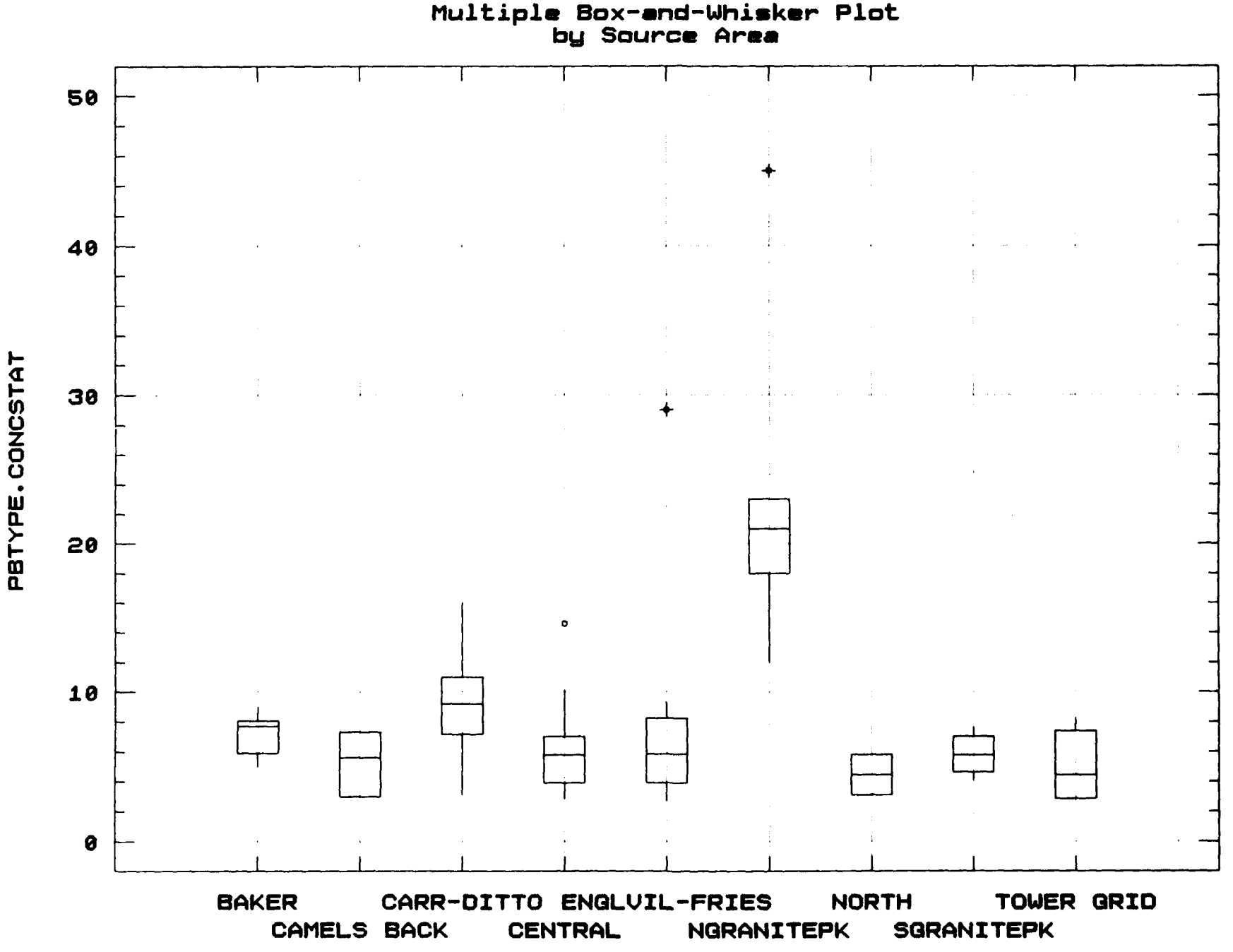
Refer to Table 3 for source areas

Figure



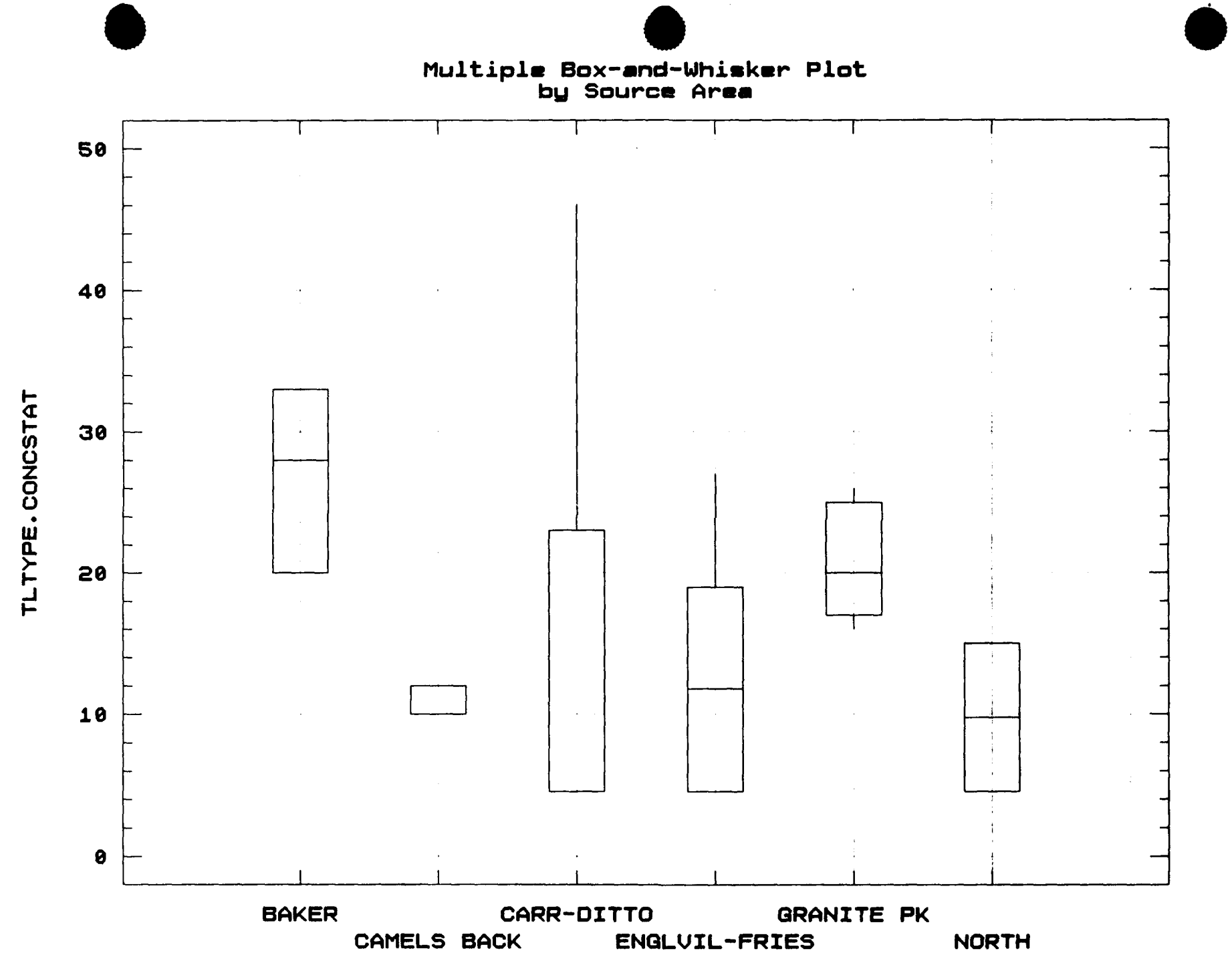
Refer to Table 3 for source areas

Figure A-9



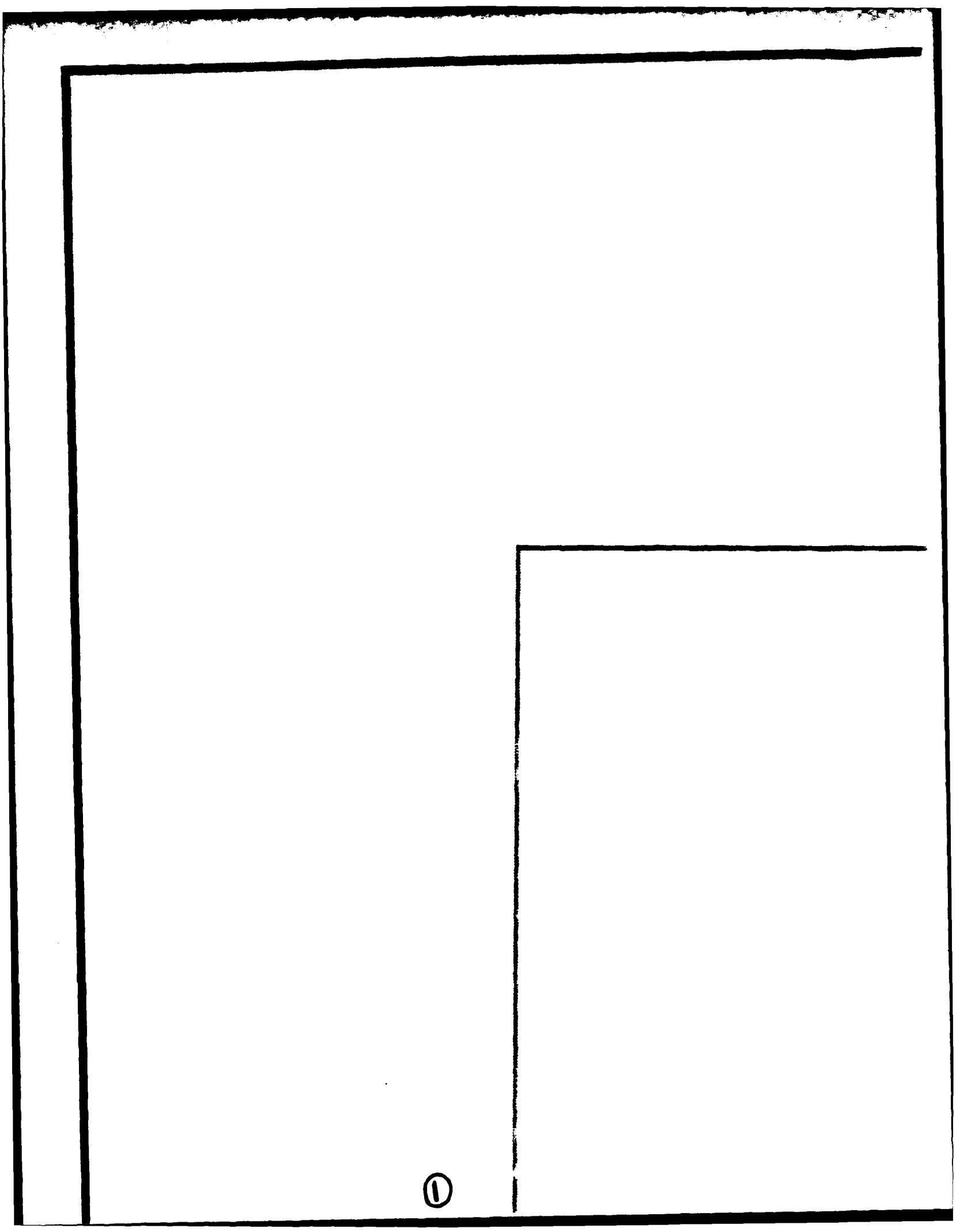
Refer to Table 3 for source areas

Figure



Refer to Table 3 for source areas

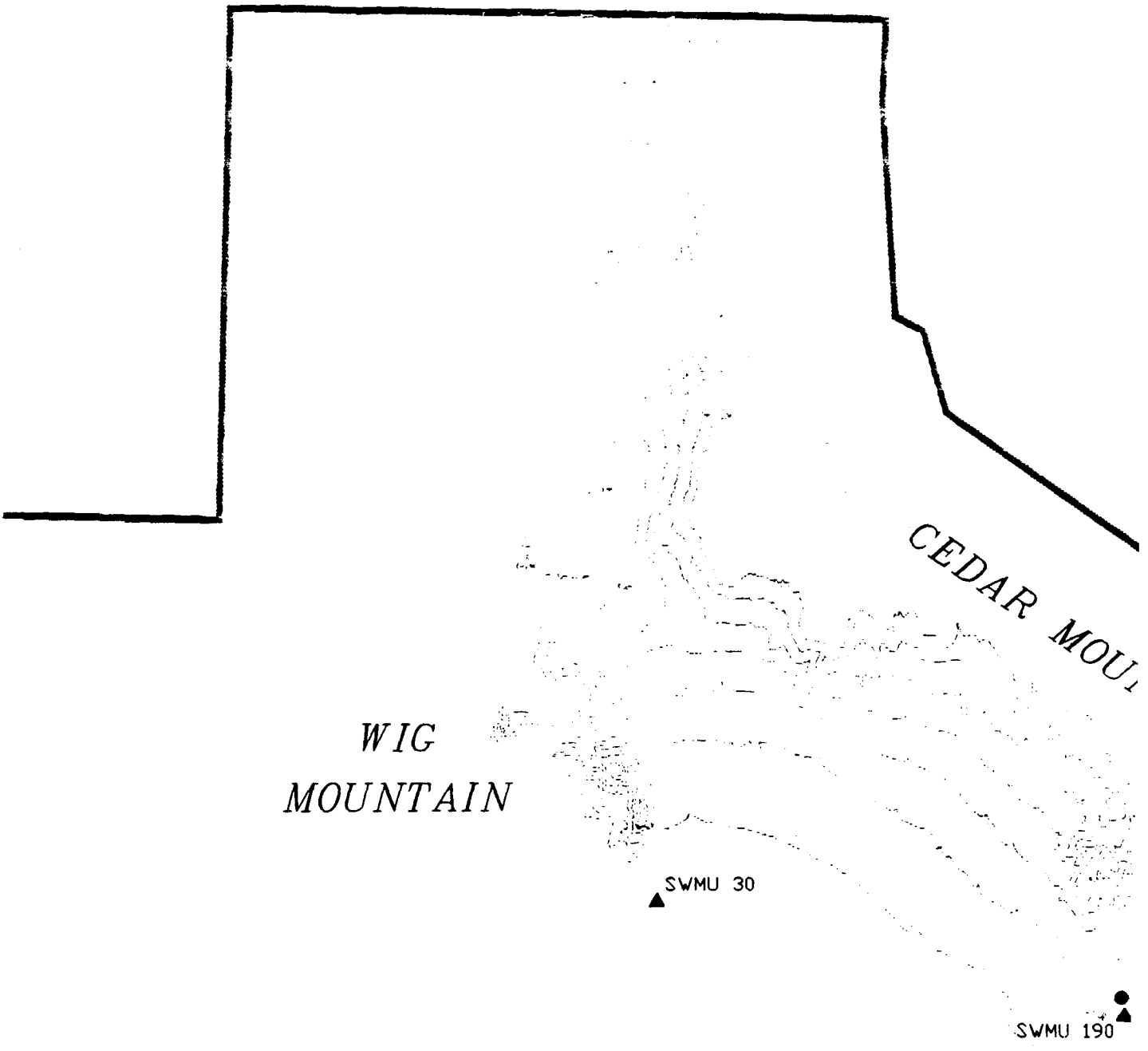
Figure A-11



DUGWAY PROVING

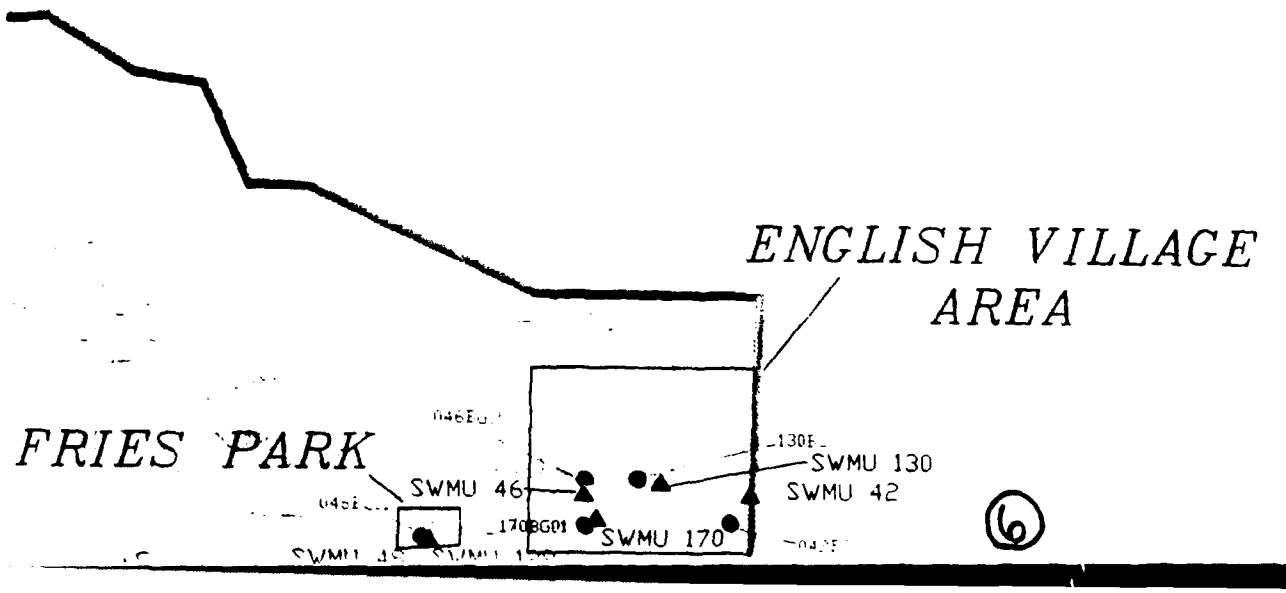
ING GROUND

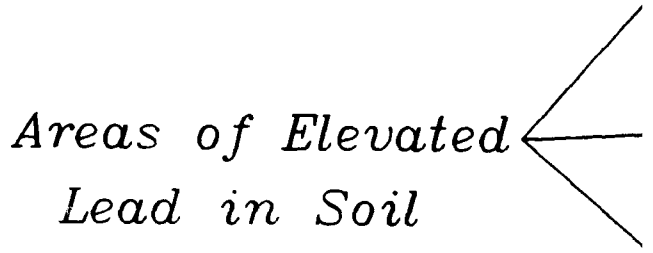
APG999SE
APG999SR
APG999SP

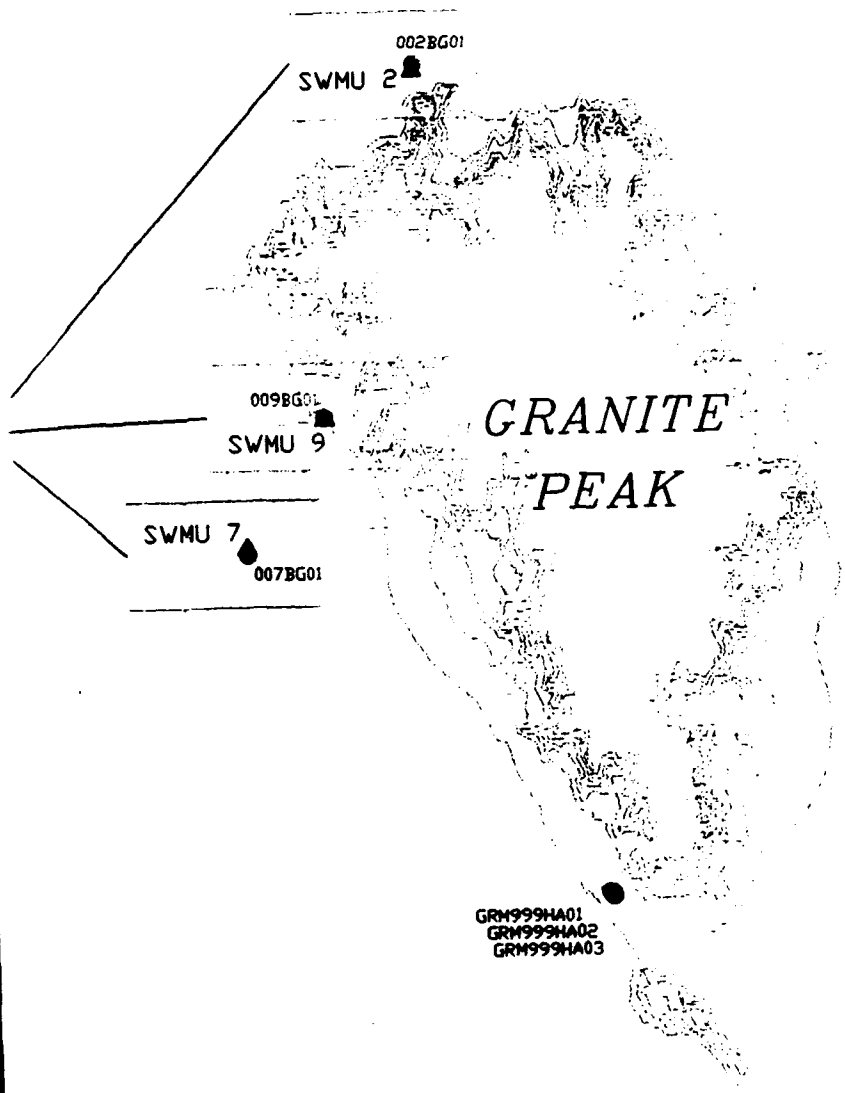


APG999SB0
APG999SB02
APG999SB01


UNTAINS
190BG01
SWMU 39
039BG01
FRIES PARK
5
046BG01
SWMU 46
048BG01
170BG01


ENGLISH VILLAGE
AREA
FRIES PARK
SWMU 46
SWMU 130
SWMU 42
SWMU 170


Areas of Elevated
Lead in Soil



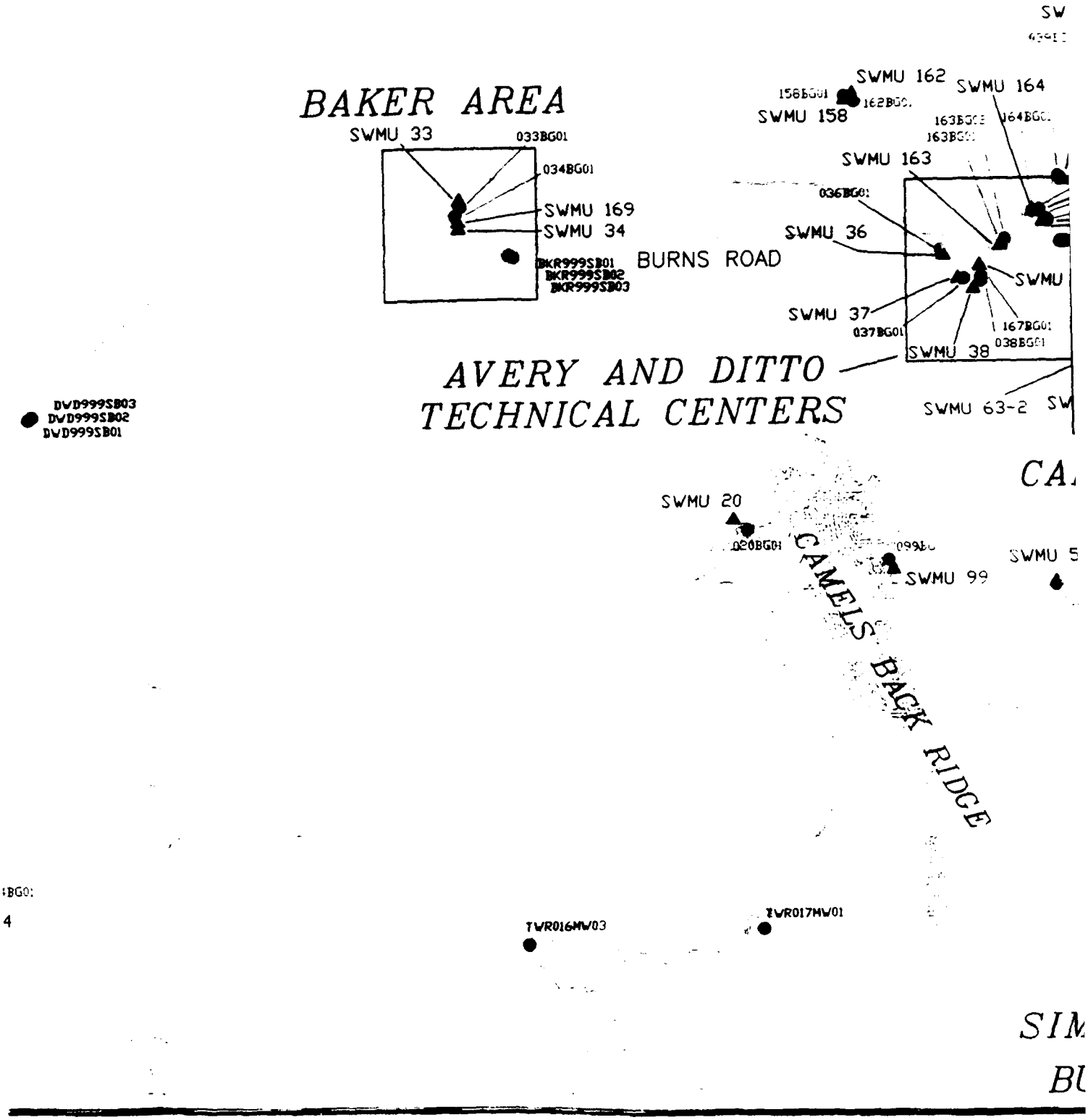
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APG999SD02
APG999SD01

DW
DWD
DWDS

014BG
SWMU 14

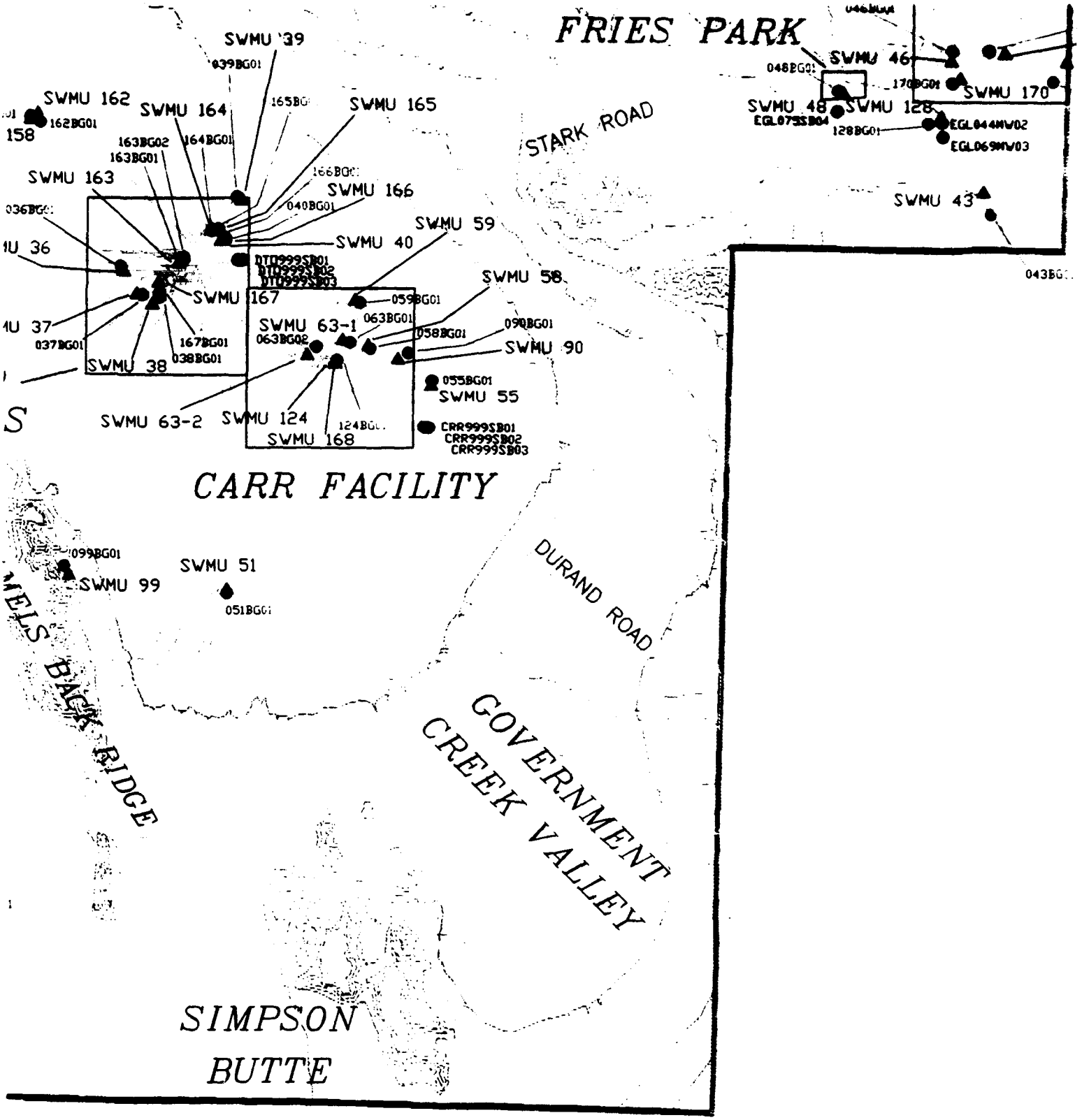
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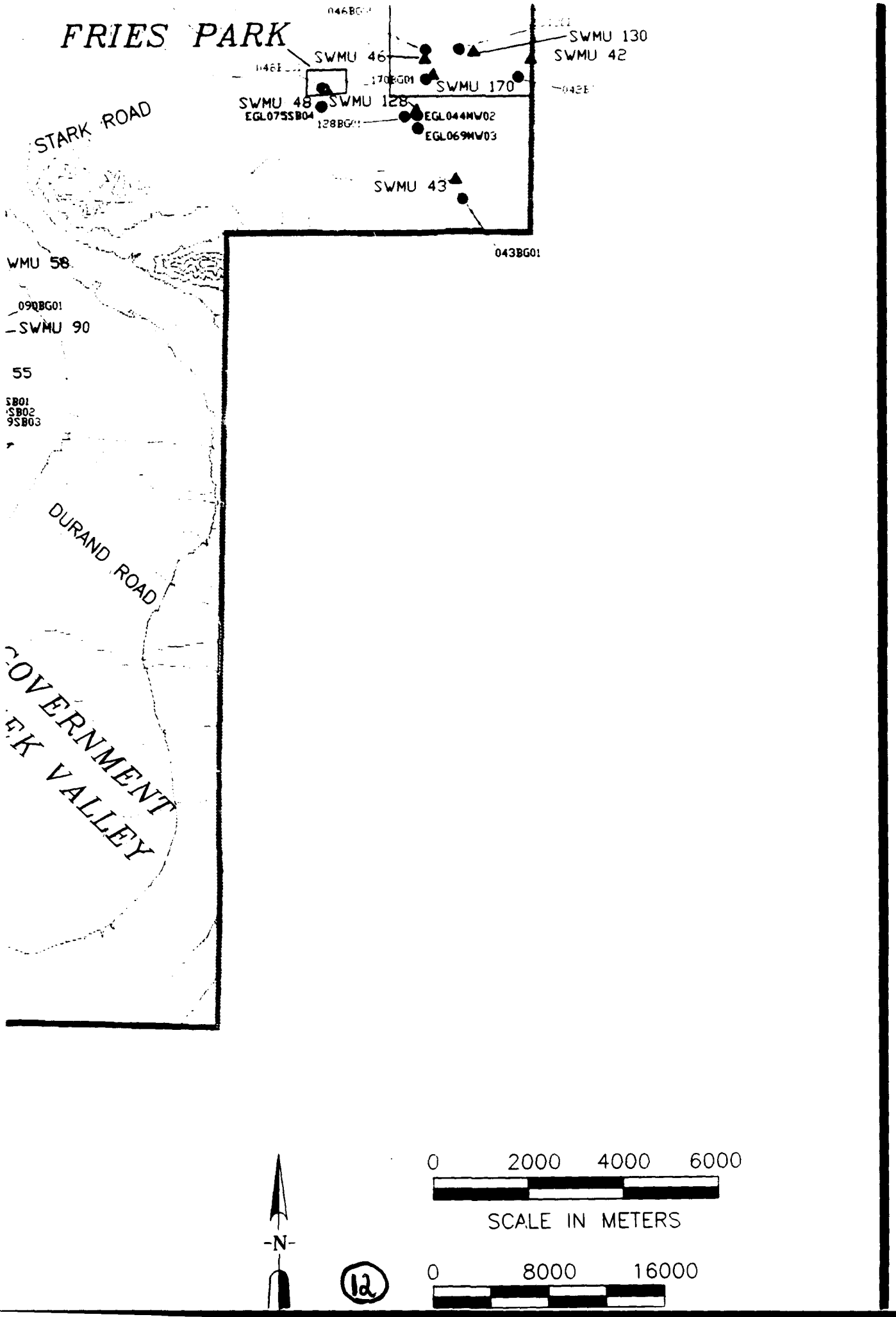
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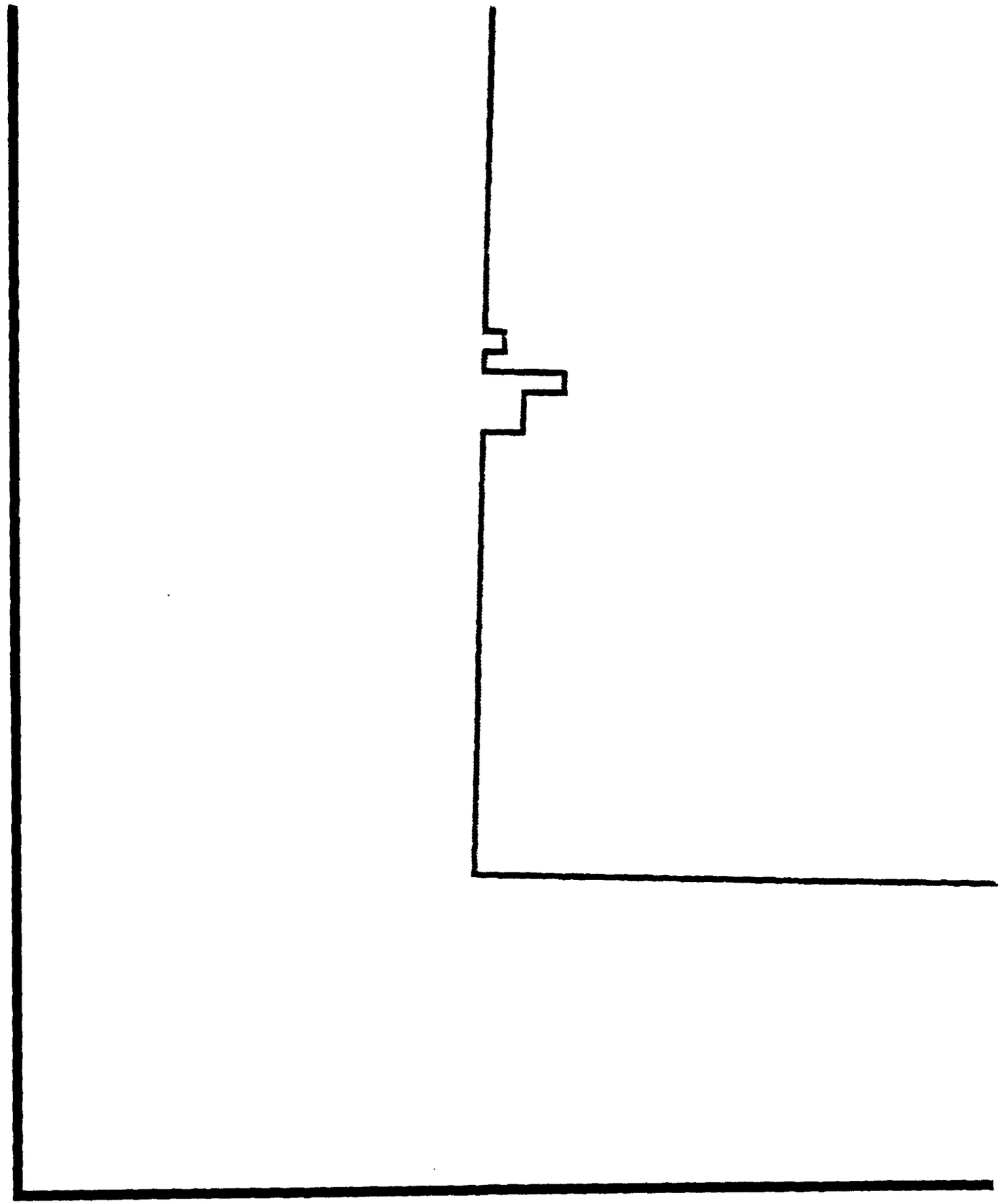


Consent Order SWMU Background Location

(10)


FRIES PARK
SWMU 39
039BG01
SWMU 162
162BG01
158
SWMU 164
165BG
SWMU 165
163BG02
163BG01
164BG01
SWMU 163
166BG
SWMU 166
040BG01
036BG
1U 36
SWMU 40
SWMU 59
DTD999SB01
DTD999SB02
DTD999SB03
SWMU 167
059BG01
SWMU 58
1U 37
037BG01
063BG01
SWMU 63-1
063BG02
058BG01
090BG01
SWMU 90
167BG01
038BG01
SWMU 38
055BG01
SWMU 55
S
SWMU 63-2
SWMU 124
124BG
SWMU 168
CRR999SB01
CRR999SB02
CRR999SB03
CARR FACILITY
099BG01
SWMU 99
SWMU 51
051BG01
MELS BACK RIDGE
DURAND ROAD
GOVERNMENT CREEK VALLEY
SIMPSON BUTTE
STARK ROAD
048BG01
SWMU 46
170BG01
SWMU 170
SWMU 48
EGL075SB04
SWMU 128
128BG01
EGL044MW02
EGL069MW03
SWMU 43
043BG
-N-
0
20
SCAL
0
8


FRIES PARK
SWMU 46
SWMU 130
SWMU 42
SWMU 170
SWMU 48
SWMU 128
EGL075SB04
EGL044MW02
EGL069MW03
STARK ROAD
SWMU 43
043BG01
WMU 58
090BG01
SWMU 90
55
SB01
SB02
9SB03
DURAND ROAD
OVERNMENT
K VALLEY
-N-
0 2000 4000 6000
SCALE IN METERS
0 8000 16000



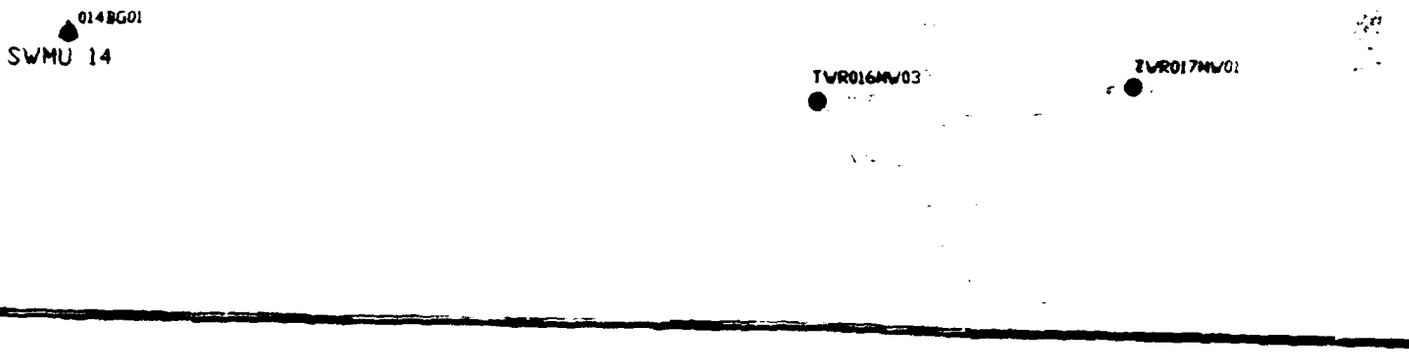
SWMU

Legend

D

R

C



ıd

Drainage

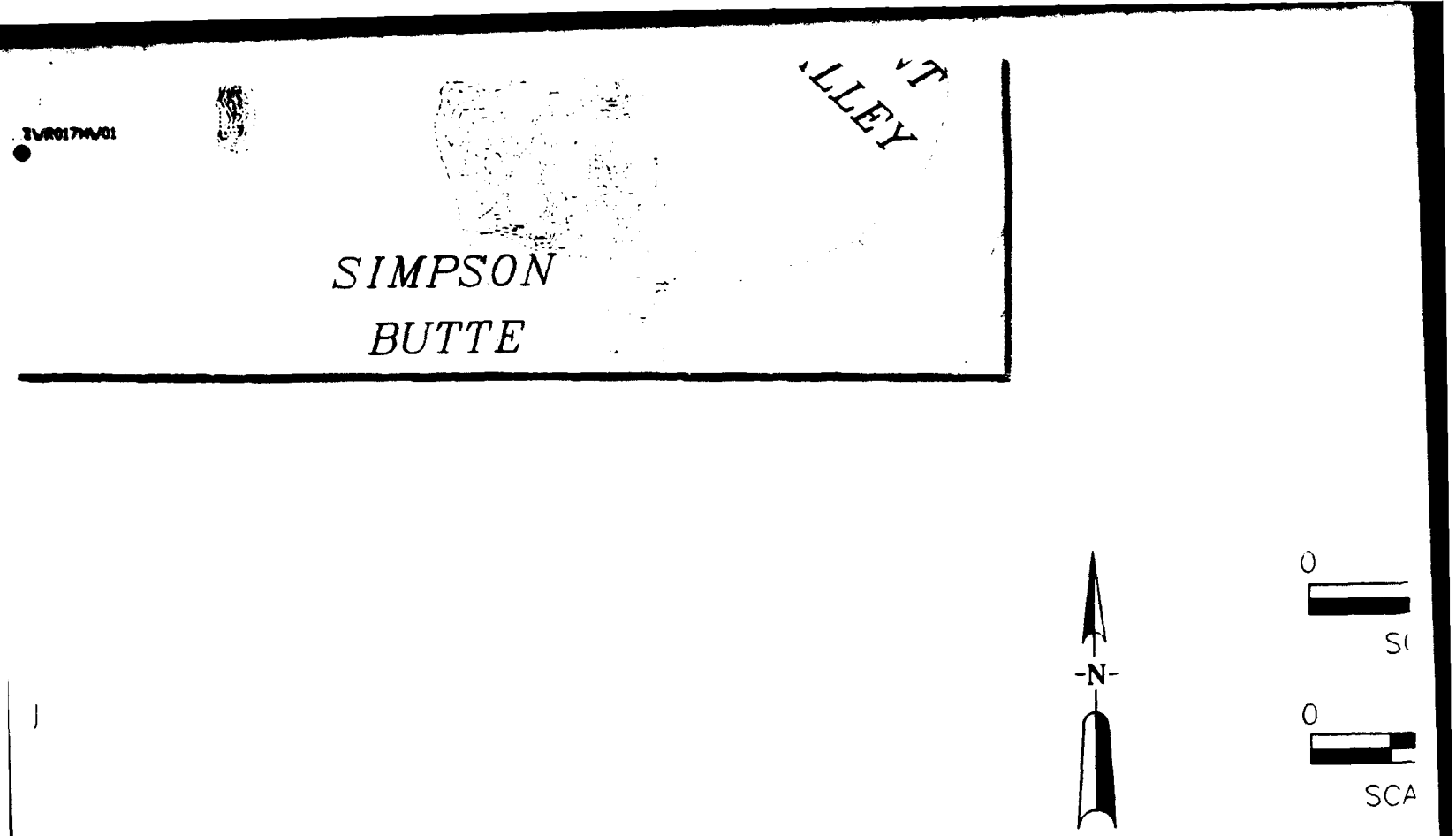
Road

Contour

Consent Order SWMU Background Location

CRR999SB01 Corrective Action SWMU Background Location

SWMU 14 SWMU Monument Location



0

S(

0

SCA

MU

:ation

Prepared for:

U.S. Army Environmental Cente
Aberdeen Proving Ground, Mar

Prepared October 1995

Plate A–1

Location of Backgrounc at Dugway Proving Gro

Dugway Proving Ground
Prepared by: Ebasco Services Inc



0 2000 4000 6000

SCALE IN METERS

0 8000 16000

SCALE IN FEET

F:\PROJECTS\DUGWAY\GRAPHICS\CLOSURE\DUG_STP.DWG

Prepared for:

U.S. Army Environmental Center
Aberdeen Proving Ground, Maryland

Prepared October 1995

Plate A–1

Location of Background Soil Samples at Dugway Proving Ground

Dugway Proving Ground
Prepared by: Ebasco Services Inc.

APPENDIX B

HUMAN HEALTH RISK ASSESSMENT

CALCULATION DOCUMENTATION AND SUPPORTING INFORMATION

APPENDIX B.1

RBSL CALCULATION DOCUMENTATION

RESIDENTIAL LAND-USE SCENARIO

LIST OF TABLES

Table B.1-1 ation of Soil Risk-Based Screening Levels (RBSLs) for Residential Use Scenario, Carcinogenic Endpoints

Table B.1-2 Derivation of Soil Risk-Based Screening Levels (RBSLs) for Residential Land-Use Scenario, Noncarcinogenic Endpoints

Table B.2-1 Derivation of Soil Risk-Based Screening Levels (RBSLs) for Industrial Land-Use Scenario, Carcinogenic Endpoints

Table B.2-2 Derivation of Soil Risk-Based Screening Levels (RBSLs) for Industrial Land-Use Scenario, Noncarcinogenic Endpoints

Appendix Table B.1-1 Derivation of Soil Risk-Based Screening Levels (RBSLs) for Residential Land-Use Scenario, Carcinogenic Endpoints

Exposed Population: Residents (childhood to adult exposures, 30-year exposure period)

Exposure Pathway: Ingestion of soil, dermal contact with soil, inhalation of soil particulates, and vapor inhalation*

**vapor inhalation evaluated for volatile constituents only, as defined by chemicals with Henry's Law constants (H) greater than 10^{-5}*

Cumulative RBSL Equation: Cum-RBSL = 1 / [(1/SI-RBSL) + (1/DC-RBSL) + (1/INH-RBSL)]

where:

SI-RBSL = Soil Ingestion Risk-Based Screening Level
DC-RBSL = Dermal Contact Risk-Based Screening Level
INH-RBSL = Inhalation Risk-Based Screening Level

Soil Ingestion Component:

Soil Ingestion RBSL (SI-RBSL) in mg/kg =

$$\frac{TR \times ATc \times 365\ days/year}{EF \times SFo \times 10^{-6}\ kg/mg \times IR_{soil/adj}}$$

Dermal Contact Component:

Dermal Contact RBSL (DC-RBSL) in mg/kg =

$$\frac{TR \times BW \times ATc \times 365\ days/year}{EF \times ED \times SFo \times 10^{-6}\ kg/mg \times SA \times AF \times RAF_d}$$

Inhalation Component:

Inhalation RBSL (INH-RBSL) in mg/kg =

$$\frac{TR \times ATc \times 365\ days/year}{EF \times ED \times URF \times 1{,}000\ ug/mg \times [1/VF + 1/PEF]}$$

(include VF component only for volatile chemicals with $H > 10^{-5}$)

Parameter	Acronym		Assumed Value	Units
Target Cancer Risk	TR		1.0E-06	unitless
Body Weight	BW		70	kg
Averaging Time (carcinogens)	ATc		70	years
Exposure Frequency	EF		350	days/year
Exposure Duration	ED		30	years
Oral cancer slope factor	SFo		chem-specific	$(mg/kg/day)^{-1}$
Soil ingestion rate, age-adjusted	$IR_{soil/adj}$		114	(mg-year)/(kg-day)
Skin surface area	SA		5,800	cm^2/day
Soil to skin adherence factor	AF		1.0	mg/cm^2
Dermal relative absorption factor	RAF_d	*inorganics:*	0.001	unitless
		organics:	0.05	unitless
Inhalation unit risk factor	URF		chem-specific	$(ug/m^3)^{-1}$
Soil-to-air volatilization factor	VF		chem-specific	m^3/kg (for chemicals with $H > 10^{-5}$)
Particulate emission factor	PEF		6.79E+08	m^3/kg

Source of equations and input parameters: EPA 1994 w/ exception of dermal component

Appendix Table B.1-1 Derivation of Soil RBSLs for Residential Land-Use Scenario, Carcinogenic Endpoints -- Inorganic Constituents

Constituent	SFo $(mg/kg/day)^{-1}$	URF $(ug/m^3)^{-1}$	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Aluminum	--	--	--	--	--	--
Antimony	--	--	--	--	--	--
Arsenic	1.75E+00	4.30E-03	3.7E-01	1.7E+01	3.8E+02	3.6E-01
Barium	--	--	--	--	--	--
Beryllium	4.30E+00	2.40E-03	1.5E-01	6.8E+00	6.9E+02	1.5E-01
Cadmium	--	1.80E-03	--	--	9.2E+02	9.2E+02
Chromium III	--	--	--	--	--	--
Chromium VI	--	1.20E-02	--	--	1.4E+02	1.4E+02
Cobalt	--	--	--	--	--	--
Copper	--	--	--	--	--	--
Cyanide	--	--	--	--	--	--
Lead	--	--	--	--	--	--
Manganese	--	--	--	--	--	--
Mercury	--	--	--	--	--	--
Nickel	--	--	--	--	--	--
Selenium	--	--	--	--	--	--
Silver	--	--	--	--	--	--
Thallium	--	--	--	--	--	--
Vanadium	--	--	--	--	--	--
Zinc	--	--	--	--	--	--

-- Carcinogenic endpoint not applicable, or toxicity data not available or pending.

Appendix Table B.1-1 Derivation of Soil RBSLs for Residential Land-Use Scenario, Carcinogenic Endpoints -- Organic Non-Volatile Constituents

Constituent	H (atm-m^3/mol)	SFo $(mg/kg/day)^{-1}$	URF $(ug/m^3)^{-1}$	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Bis(2-ethylhexyl) phthalate	8.36E-06	1.4E-02	--	4.6E+01	4.2E+01	--	2.2E+01

Where available, source of H values is EPA 1994, otherwise taken from Table 2.6-1 of the Interim Report.

Appendix Table B.1-1 Derivation of Soil RBSLs for Residential Land-Use Scenario, Carcinogenic Endpoints -- Organic Volatile Constituents

Constituent	H ($atm\text{-}m^3/mol$)	SFo $(mg/kg/day)^{-1}$	URF $(ug/m^3)^{-1}$	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Acetone	2.88E-05	--	--	--	--	--	--
PPDDT	5.37E-05	3.4E-01	9.7E-05	1.9E+00	1.7E+00	8.0E+01	8.9E-01
Methylene chloride	2.37E-03	7.5E-03	4.7E-07	8.5E+01	7.8E+01	7.1E+00	6.0E+00
Tetrachloroethane (1,1,2,2-)	3.72E-04	2.0E-01	5.8E-05	3.2E+00	2.9E+00	4.1E-01	3.2E-01
Toluene	6.14E-03	--	--	--	--	--	--
Xylenes	6.04E-03	--	--	--	--	--	--

-- Carcinogenic endpoint not applicable.

Where available, source of H values is EPA 1994, otherwise taken from Table 2.6-1 of the Interim Report.

Appendix Table B.1-1 Derivation of Soil RBSLs for Residential Land-Use Scenario, Carcinogenic Endpoints -- VF and SSL Calculations

Volatilization Factor (VF) Equation

$VF = Q/C * (10^{-4}\ m^2/cm^2) * (3.14 * \alpha * T)^{1/2} / (2 * Dei * \theta_a * Kas)$

where:

$\alpha\ (cm^2/s) = (Dei * \theta_a^{3.33} / (\theta_a + ((\rho s)(1 - \theta_a) / Kas))$

$Dei\ (cm^2/s) = Di * (\theta_a^{3.33} / n^2)$

$\theta_a = n - w\rho b = 0.28$ Lair/Lsoil

$n = 1 - (\rho b/\rho s) = 0.43$ Lpore/Lsoil

Soil Saturation Limit (Csat) Equation

$Csat = S/\rho b * [(Kd * \rho b) + \theta_w + (H' * \theta a)]$

where:		Units
S = solubility in water	= chemical-specific	mg/L-water
ρb = dry soil bulk density	= 1.5	kg/L
θ_w = water-filled soil porosity	= 0.15	L water / L soil
w = average soil moisture content	= 0.1 or 10 percent	unitless

VF Input Parameter Definitions		Value	Units
VF = Volatilization Factor			m^3/kg
Q/C = inverse of the mean conc. at the center of a 30-acre-square source	=	35.1	g/m^2-s per kg/m^3
T = exposure interval	=	9.5E+08	s
Dei = effective diffusivity	=	i * ($\theta_a^{3.33}$/ n^2)	cm^2/s
θ_a = air-filled soil porosity	=	0.28	Lair / Lsoil
Di = diffusivity in air	=	chemical-specific	cm^2/s
n = total soil porosity (for loam)	=	0.43	Lpore / Lsoil
w = average soil moisture content	=	0.10	cm3 water / g soil
ρb = dry soil bulk density		1.5	g/cm^3 or kg/L
ρs = soil particle density		2.65	g/cm^3
Kas = soil-air partition coefficient	=	H'/Kd	g-soil / cm^3-air
H = Henry's law constant	=	chemical-specific	atm-m^3/mol
H' = Henry's law constant, unitless	=	H * 41	unitless*
**41 is a units conversion factor (where units = mol/atm-m3)*			
Kd = soil-water partition coefficient	=	chemical-specific	cm^3/g or L/kg**
**or, in lieu of chem-specific data, Kd = Koc * foc*			
Koc = organic carbon partition coeffici	=	chem-specific	cm^3/g or L/kg
foc = organic carbon content of soil	=	0.006	g/g (0.6%)

Source of Equations and Default Input Parameters: EPA Draft Soil Screening Guidance (EPA/540/R-94/106, December 1994)

Chemical-Specific Volatilization Factors (VFs) and Soil Saturation Limit (Csat) Derivations

Parameter	Di (cm^2/s)	Dei * (cm^2/s)	H (atm-m^3/mol)	H' (unitless)	Koc (cm3/g)	Kd* (cm^3/g)	Kas * (g/cm3)	a * (cm^2/s)	S (mg/L-water)	VF (m^3/kg)	Csat (mg/kg)
Acetone	1.24E-01	9.7E-03	2.88E-05	1.2E-03	4.60E-01	2.76E-03	4.3E-01	5.7E-04	6.04E+05	2.0E+03	6.22E+04
PPDDT	1.37E-02	1.1E-03	5.37E-05	2.2E-03	2.37E+05	1.42E+03	1.5E-06	2.4E-10	3.41E-03	3.2E+06	4.85E+00
Methylene chloride	1.01E-01	7.9E-03	2.37E-03	9.7E-02	1.60E+01	9.60E-02	1.0E+00	1.0E-03	1.74E+04	1.4E+03	3.73E+03
1,2,2-Tetrachloroethane	7.10E-02	5.5E-03	3.72E-04	1.5E-02	7.90E+01	4.74E-01	3.2E-02	2.6E-05	3.07E+03	9.8E+03	1.77E+03
Toluene	8.70E-02	6.8E-03	6.14E-03	2.5E-01	1.31E+02	7.86E-01	3.2E-01	3.0E-04	5.58E+02	2.7E+03	5.21E+02
Xylenes	7.20E-02	5.6E-03	6.04E-03	2.5E-01	2.60E+02	1.56E+00	1.6E-01	1.3E-04	1.86E+02	4.3E+03	3.17E+02

* refers to calculated value.

Where available, source of H values is EPA 1994, Table 5-5 ; otherwise taken from Table 2.6-1 of Interim Report

Source of Koc, Di, and S values is EPA 1994, Table 3-2, Chemical-Specific Properties Used in SSL Calculations

Appendix Table B.1-2 Derivation of Soil Risk-Based Screening Levels (RBSLs) for Residential Land-Use Scenario, Noncarcinogenic Endpoints

Exposed Population: Child residents, assuming 6-year childhood exposure

Exposure Pathway: Ingestion of soil, dermal contact with soil, inhalation of soil particulates, and vapor inhalation*

**vapor inhalation evaluated for volatile constituents only, as defined by chemicals with Henry's Law constants (H) greater than 10^{-5}*

Cumulative RBSL Equation: Cum-RBSL = 1 / [(1/SI-RBSL) + (1/DC-RBSL) + (1/INH-RBSL)]

where:

SI-RBSL = Soil Ingestion Risk-Based Screening Level
DC-RBSL = Dermal Contact Risk-Based Screening Level
INH-RBSL = Inhalation Risk-Based Screening Level

Soil Ingestion Component:

Soil Ingestion RBSL (SI-RBSL) in mg/kg =

$$\frac{THQ \times BW \times AT \times 365\ days/year \times RfDo}{EF \times ED \times 10^{-6}\ kg/mg \times IR_{soil}}$$

Dermal Contact Component:

Dermal Contact RBSL (DC-RBSL) in mg/kg =

$$\frac{THQ \times BW \times AT \times 365\ days/year \times RfDo}{EF \times ED \times 10^{-6}\ kg/mg \times SA \times AF \times RAF_d}$$

Inhalation Component:

Inhalation RBSL (INH-RBSL) in mg/kg =

$$\frac{THQ \times AT \times 365\ days/year}{EF \times ED \times [\,1/RfC * (1/VF + 1/PEF)]}$$

(include VF component only for volatile chemicals with $H > 10^{-5}$)

Parameter	Acronym		Assumed Value	Units
Target Hazard Quotient	THQ		1.0	unitless
Body Weight	BW		15	kg
Averaging Time	AT		6	years
Exposure Frequency	EF		350	days/year
Exposure Duration	ED		6	years
Oral reference dose	RfDo		chem-specific	mg/kg/day
Soil ingestion rate	IR_{soil}		200	mg/day
Skin surface area	SA		2,650	cm^2/day
Soil to skin adherence factor	AF		1.0	mg/cm^2
Dermal relative absorption factor	RAF_d	*inorganics:*	0.001	unitless
		organics:	0.05	unitless
Inhalation reference concentration	RfC		chem-specific	mg/m^3
Soil-to-air volatilization factor	VF		chem-specific	m^3/kg (for chemicals with $H > 10^{-5}$)
Particulate emission factor	PEF		6.79E+08	m^3/kg

Source of equations and input parameters: EPA 1994 w/ exception of dermal component

Appendix Table B.1-2 Derivation of Soil RBSLs for Residential Land-Use Scenario, Noncarcinogenic Endpoints -- Inorganic Constituents

Constituent	RfD (mg/kg/day)	RfC (mg/m^3)	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Aluminum	1.0E+00	--	7.8E+04	5.9E+06	--	7.7E+04
Antimony	4.00E-04	--	3.1E+01	2.4E+03	--	3.1E+01
Arsenic	3.00E-04	--	2.3E+01	1.8E+03	--	2.3E+01
Barium	7.00E-02	--	5.5E+03	4.1E+05	--	5.4E+03
Beryllium	5.00E-03	--	3.9E+02	3.0E+04	--	3.9E+02
Cadmium	5.00E-04	--	3.9E+01	3.0E+03	--	3.9E+01
Chromium III	1.00E+00	--	7.8E+04	5.9E+06	--	7.7E+04
Chromium VI	5.00E-03	--	3.9E+02	3.0E+04	--	3.9E+02
Copper	--	--	--	--	--	--
Cyanide	2.00E-02	--	1.6E+03	1.2E+05	--	1.5E+03
Lead	--	--	--	--	--	--
Manganese	5.00E-03	5.00E-05	3.9E+02	3.0E+04	3.5E+04	3.8E+02
Mercury	3.0E-04	3.00E-01	2.3E+01	1.8E+03	2.1E+08	2.3E+01
Nickel	2.00E-02	--	1.6E+03	1.2E+05	--	1.5E+03
Selenium	5.00E-03	--	3.9E+02	3.0E+04	--	3.9E+02
Silver	5.00E-03	--	3.9E+02	3.0E+04	--	3.9E+02
Thallium [1]	8.00E-05	--	6.3E+00	4.7E+02	--	6.2E+00
Vanadium [2]	9.00E-03	--	7.0E+02	5.3E+04	--	6.9E+02
Zinc	3.00E-01	--	2.3E+04	1.8E+06	--	2.3E+04

-- No toxicity data available.

[1] RfD for thallium is that reported in IRIS for thallium chloride (Oct 95).

[2] RfD for vanadium is that reported in IRIS for vanadium pentoxide (Oct 95).

Appendix Table B.1-2 Derivation of Soil RBSLs for Residential Land-Use Scenario, Noncarcinogenic Endpoints -- Organic Non-Volatile Constituents

Constituent	H ($atm\text{-}m^3/mol$)	RfD (mg/kg/day)	RfC (mg/m^3)	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Bis(2-ethylhexyl) phthalate	8.36E-06	2.0E-02	--	1.6E+03	2.4E+03	--	9.4E+02

Where available, source of H values is EPA 1994, otherwise taken from Table 2.6-1 of the Interim Report.

Appendix Table B.1-2 Derivation of Soil RBSLs for Residential Land-Use Scenario, Noncarcinogenic Endpoints -- Organic Volatile Constituents

Constituent	H (atm-m^3/mol)	RfD (mg/kg/day)	RfC (mg/m^3)	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Acetone	2.88E-05	1.0E-01	--	7.8E+03	1.2E+04	--	4.7E+03
DDT	5.37E-05	5.0E-04	--	3.9E+01	5.9E+01	--	2.4E+01
Methylene chloride	2.37E-03	6.0E-02	--	4.7E+03	7.1E+03	--	2.8E+03
Tetrachloroethane (1,1,2,2-)	3.72E-04	--	--	--	--	--	--
Toluene	6.14E-03	2.0E-01	4.0E-01	1.6E+04	2.4E+04	1.1E+03	1.0E+03
Xylenes	6.04E-03	2.0E+00	--	1.6E+05	2.4E+05	--	9.4E+04

-- Toxicity data not applicable.

Where available, source of H values is EPA 1994, otherwise taken from Table 2.6-1 of the Interim Report.

Appendix Table B.1-2 Derivation of Soil RBSLs for Residential Land-Use Scenario, Volatilization Factor (VF) and Soil Saturation Limit (SSL) Calculations

Volatilization Factor (VF) Equation

$VF = Q/C * (10^{-4}\ m^2/cm^2) * (3.14 * \alpha * T)^{1/2} / (2 * Dei * \theta_a * Kas)$

where:

$\alpha\ (cm^2/s) = (Dei * \theta_a) / (\theta_a + ((\rho s)(1 - \theta_a) / Kas))$

$Dei\ (cm^2/s) = Di * (\theta_a^{3.33} / n^2)$

θ_a = n - w ρb = 0.28 Lair/Lsoil

n = 1 - (ρb/ρs) = 0.43 Lpore/Lsoil

Soil Saturation Limit (Csat) Equation

$Csat = S/\rho b * [(Kd * \rho b) + \theta_w + (H' * \theta a)]$

where:		Units
S = solubility in water	= chemical-specific	mg/L-water
ρb = dry soil bulk density	= 1.5	kg/L
θ_w = water-filled soil porosity	= 0.15	L water / L soil
w = average soil moisture content	= 0.1 or 10 percent	unitless

VF Input Parameter Definitions		Value	Units
VF = Volatilization Factor			m^3/kg
Q/C = inverse of the mean conc. at the center of a 30-acre-square source	=	35.1	g/m^2-s per kg/m^3
T = exposure interval	=	9.5E+08	s
Dei = effective diffusivity	=	i * ($\theta_a^{3.33}$/ n^2)	cm^2/s
θ_a = air-filled soil porosity	=	0.28	Lair / Lsoil
Di = diffusivity in air	=	chemical-specific	cm^2/s
n = total soil porosity (for loam)	=	0.43	Lpore / Lsoil
w = average soil moisture content	=	0.10	cm3 water / g soil
ρb = dry soil bulk density		1.5	g/cm^3 or kg/L
ρs = soil particle density		2.65	g/cm^3
Kas = soil-air partition coefficient	=	H'/Kd	g-soil / cm^3-air
H = Henry's law constant	=	chemical-specific	atm-m^3/mol
H' = Henry's law constant, unitless	=	H * 41	unitless*
**41 is a units conversion factor (where units = mol/atm-m3)*			
Kd = soil-water partition coefficient	=	chemical-specific	cm^3/g or L/kg**
***or, in lieu of chem-specific data, Kd = Koc * foc*			
Koc = organic carbon partition coeffici	=	chem-specific	cm^3/g or L/kg
foc = organic carbon content of soil	=	0.006	g/g (0.6%)

Source of Equations and Default Input Parameters: ***EPA Draft Soil Screening Guidance (EPA/540/R-94/106, December 1994)***

Chemical-Specific Volatilization Factors (VFs) and Soil Saturation Limit (Csat) Derivations

Parameter	Di (cm^2/s)	Dei * (cm^2/s)	H (atm-m^3/mol)	H' (unitless)	Koc (cm3/g)	Kd* (cm^3/g)	Kas * (g/cm3)	a * (cm^2/s)	S (mg/L-water)	VF (m^3/kg)	Csat (mg/kg)
Acetone	1.24E-01	9.7E-03	2.88E-05	1.2E-03	4.60E-01	2.76E-03	4.3E-01	5.7E-04	6.04E+05	2.0E+03	6.22E+04
PPDDT	1.37E-02	1.1E-03	5.37E-05	2.2E-03	2.37E+05	1.42E+03	1.5E-06	2.4E-10	3.41E-03	3.2E+06	4.85E+00
Methylene chloride	1.01E-01	7.9E-03	2.37E-03	9.7E-02	1.60E+01	9.60E-02	1.0E+00	1.0E-03	1.74E+04	1.4E+03	3.73E+03
Tetrachloroethane (1,1,2,2)	7.10E-02	5.5E-03	3.72E-04	1.5E-02	7.90E+01	4.74E-01	3.2E-02	2.6E-05	3.07E+03	9.8E+03	1.77E+03
Toluene	8.70E-02	6.8E-03	6.14E-03	2.5E-01	1.31E+02	7.86E-01	3.2E-01	3.0E-04	5.58E+02	2.7E+03	5.21E+02
Xylenes	7.20E-02	5.6E-03	6.04E-03	2.5E-01	2.60E+02	1.56E+00	1.6E-01	1.3E-04	1.86E+02	4.3E+03	3.17E+02

* refers to calculated value.

Where available, source of H values is EPA 1994, Table 5-5 ; otherwise taken from Table 2.6-1 of Interim Report

Source of Koc, Di, and S values is EPA 1994, Table 3-2, Chemical-Specific Properties Used in SSL Calculations

APPENDIX B.2

RBSL DOCUMENTATION

INDUSTRIAL LAND-USE SCENARIO

Appendix Table B.2-1 Derivation of Soil Risk-Based Screening Levels (RBSLs) for Industrial Land-Use Scenario, Carcinogenic Endpoints

Exposed Population: Adult workers under industrial land-use scenario

Exposure Pathway: Ingestion of soil, dermal contact with soil, inhalation of soil particulates, and vapor inhalation*

**vapor inhalation evaluated for volatile constituents only, as defined by chemicals with Henry's Law constants (H) greater than 10^{-5}*

Cumulative RBSL Equation: Cum-RBSL = 1 / [(1/SI-RBSL) + (1/DC-RBSL) + (1/INH-RBSL)]

where:

SI-RBSL = Soil Ingestion Risk-Based Screening Level
DC-RBSL = Dermal Contact Risk-Based Screening Level
INH-RBSL = Inhalation Risk-Based Screening Level

Soil Ingestion Component:

Soil Ingestion RBSL (SI-RBSL) in mg/kg =

$$\frac{TR \times BW \times ATc \times 365\ days/year}{EF \times ED \times SFo \times 10^{-6}\ kg/mg \times IR_{soil}}$$

Dermal Contact Component:

Dermal Contact RBSL (DC-RBSL) in mg/kg =

$$\frac{TR \times BW \times ATc \times 365\ days/year}{EF \times ED \times SFo \times 10^{-6}\ kg/mg \times SA \times AF \times RAF_d}$$

Inhalation Component:

Inhalation RBSL (INH-RBSL) in mg/kg =

$$\frac{TR \times ATc \times 365\ days/year}{EF \times ED \times URF \times 1{,}000\ ug/mg \times [1/VF + 1/PEF]}$$

(include VF component only for volatile chemicals with $H > 10^{-5}$)

Parameter	Acronym		Assumed Value	Units
Target Cancer Risk	TR		1.0E-06	unitless
Body Weight	BW		70	kg
Averaging Time (carcinogens)	ATc		70	years
Exposure Frequency	EF		250	days/year
Exposure Duration	ED		25	years
Oral cancer slope factor	SFo		chem-specific	$(mg/kg/day)^{-1}$
Soil ingestion rate	IR_{soil}		50	mg/day
Skin surface area	SA		5,800	cm^2/day
Soil to skin adherence factor	AF		1.0	mg/cm^2
Dermal relative absorption factor	RAF_d	*inorganics:*	0.001	unitless
		organics:	0.05	unitless
Inhalation unit risk factor	URF		chem-specific	$(ug/m^3)^{-1}$
Soil-to-air volatilization factor	VF		chem-specific	m^3/kg (for chemicals with $H > 10^{-5}$)
Particulate emission factor	PEF		6.79E+08	m^3/kg

Source of equations and input parameters: EPA 1994 w/ exception of dermal component

Appendix Table B.2-1 Derivation of Soil RBSLs for Industrial Land-Use Scenario, Carcinogenic Endpoints -- Inorganic Constituents

Constituent	SFo $(mg/kg/day)^{-1}$	URF $(ug/m^3)^{-1}$	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Aluminum	--	--	--	--	--	--
Antimony	--	--	--	--	--	--
Arsenic	1.75E+00	4.30E-03	3.3E+00	2.8E+01	6.5E+02	2.9E+00
Barium	--	--	--	--	--	--
Beryllium	4.30E+00	2.40E-03	1.3E+00	1.1E+01	1.2E+03	1.2E+00
Cadmium	--	1.80E-03	--	--	1.5E+03	1.5E+03
Chromium III	--	--	--	--	--	--
Chromium VI	--	1.20E-02	--	--	2.3E+02	2.3E+02
Copper	--	--	--	--	--	--
Cyanide	--	--	--	--	--	--
Lead	--	--	--	--	--	--
Manganese	--	--	--	--	--	--
Mercury	--	--	--	--	--	--
Nickel	--	--	--	--	--	--
Selenium	--	--	--	--	--	--
Silver	--	--	--	--	--	--
Thallium	--	--	--	--	--	--
Vanadium	--	--	--	--	--	--
Zinc	--	--	--	--	--	--

-- Carcinogenic endpoint not applicable, or toxicity data not available or pending.

Appendix Table B.2-1 Derivation of Soil RBSLs for Industrial Land-Use Scenario,
Carcinogenic Endpoints -- Organic Non-Volatile Constituents

Constituent	H ($atm\text{-}m^3/mol$)	SFo $(mg/kg/day)^{-1}$	URF $(ug/m^3)^{-1}$	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Bis(2-ethylhexyl) phthalate	8.36E-06	1.4E-02	--	4.1E+02	7.0E+01	--	6.0E+01

Appendix Table B.2-1 Derivation of Soil RBSLs for Industrial Land-Use Scenario, Carcinogenic Endpoints -- Organic Volatile Constituents

Constituent	H (atm-m^3/mol)	SFo $(mg/kg/day)^{-1}$	URF $(ug/m^3)^{-1}$	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Acetone	2.88E-05	--	--	--	--	--	--
PPDDT	5.37E-05	3.4E-01	9.7E-05	1.7E+01	2.9E+00	1.4E+02	2.4E+00
Methylene chloride	2.37E-03	7.5E-03	4.7E-07	7.6E+02	1.3E+02	1.2E+01	1.1E+01
Tetrachloroethane (1,1,2,2-)	3.72E-04	2.0E-01	5.8E-05	2.9E+01	4.9E+00	6.9E-01	5.9E-01
Toluene	6.14E-03	--	--	--	--	--	--
Xylenes	6.04E-03	--	--	--	--	--	--

-- Carcinogenic endpoint not applicable.

Appendix Table B.2-1 Derivation of Soil RBSLs for Industrial Land-Use Scenario, Carcinogenic Endpoints -- VF and SSL Calculations

Volatilization Factor (VF) Equation

$VF = Q/C * (10^{-4} m^2/cm^2) * (3.14 * \alpha * T)^{1/2} / (2 * Dei * \theta_a * Kas)$

where:

$\alpha\ (cm^2/s) = (Dei * \theta_a^{3.33} / (\theta_a + ((\rho s)(1 - \theta_a) / Kas))$

$Dei\ (cm^2/s) = Di * (\theta_a^{3.33} / n^2)$

$\theta_a = n - w\,\rho b = 0.28$ Lair/Lsoil

$n = 1 - (\rho b/\rho s) = 0.43$ Lpore/Lsoil

Soil Saturation Limit (Csat) Equation

$Csat = S/\rho b * [(Kd * \rho b) + \theta_w + (H' * \theta a)]$

where:

		Units
S = solubility in water	= chemical-specific	mg/L-water
ρb = dry soil bulk density	= 1.5	kg/L
θ_w = water-filled soil porosity	= 0.15	L water / L soil
w = average soil moisture content	= 0.1 or 10 percent	unitless

VF Input Parameter Definitions

		Value	Units
VF = Volatilization Factor			m^3/kg
Q/C = inverse of the mean conc. at the center of a 30-acre-square source	=	35.1	g/m^2-s per kg/m^3
T = exposure interval	=	9.5E+08	s
Dei = effective diffusivity	=	i * ($\theta_a^{3.33}$/ n^2)	cm^2/s
θ_a = air-filled soil porosity	=	0.28	Lair / Lsoil
Di = diffusivity in air	=	chemical-specific	cm^2/s
n = total soil porosity (for loam)	=	0.43	Lpore / Lsoil
w = average soil moisture content	=	0.10	cm3 water / g soil
ρb = dry soil bulk density		1.5	g/cm^3 or kg/L
ρs = soil particle density		2.65	g/cm^3
Kas = soil-air partition coefficient	=	H'/Kd	g-soil / cm^3-air
H = Henry's law constant	=	chemical-specific	atm-m^3/mol
H' = Henry's law constant, unitless	=	H * 41	unitless*

**41 is a units conversion factor (where units = mol/atm-m3)*

Kd = soil-water partition coefficient	=	chemical-specific	cm^3/g or L/kg**
Koc = organic carbon partition coeffici	=	chem-specific	cm^3/g or L/kg
foc = organic carbon content of soil	=	0.006	g/g (0.6%)

***Source of Equations and Default Input Parameters:** EPA Draft Soil Screening Guidance (EPA/540/R-94/106, December 1994)*

Chemical-Specific Volatilization Factors (VFs) and Soil Saturation Limit (Csat) Derivations

Parameter	Di (cm^2/s)	Dei * (cm^2/s)	H (atm-m^3/mol)	H' (unitless)	Koc (cm3/g)	Kd* (cm^3/g)	Kas * (g/cm3)	a * (cm^2/s)	S (mg/L-water)	VF (m^3/kg)	Csat (mg/kg)
Acetone	1.24E-01	9.7E-03	2.88E-05	1.2E-03	4.60E-01	2.76E-03	4.3E-01	5.7E-04	6.04E+05	2.0E+03	6.22E+04
PPDDT	1.37E-02	1.1E-03	5.37E-05	2.2E-03	2.37E+05	1.42E+03	1.5E-06	2.4E-10	3.41E-03	3.2E+06	4.85E+00
Methylene chloride	1.01E-01	7.9E-03	2.37E-03	9.7E-02	1.60E+01	9.60E-02	1.0E+00	1.0E-03	1.74E+04	1.4E+03	3.73E+03
Tetrachloroethane (1,1,2,2)	7.10E-02	5.5E-03	3.72E-04	1.5E-02	7.90E+01	4.74E-01	3.2E-02	2.6E-05	3.07E+03	9.8E+03	1.77E+03
Toluene	8.70E-02	6.8E-03	6.14E-03	2.5E-01	1.31E+02	7.86E-01	3.2E-01	3.0E-04	5.58E+02	2.7E+03	5.21E+02
Xylenes	7.20E-02	5.6E-03	6.04E-03	2.5E-01	2.60E+02	1.56E+00	1.6E-01	1.3E-04	1.86E+02	4.3E+03	3.17E+02

* refers to calculated value.

Where available, source of H values is EPA 1994, Table 5-5 ; otherwise taken from Table 2.6-1 of Interim Report

Source of Koc, Di, and S values is EPA 1994, Table 3-2, Chemical-Specific Properties Used in SSL Calculations

Appendix Table B.2-2 Derivation of Soil Risk-Based Screening Levels (RBSLs) for Industrial Land-Use Scenario, Noncarcinogenic Endpoints

Exposed Population: Adult workers under industrial land-use scenario

Exposure Pathway: Ingestion of soil, dermal contact with soil, inhalation of soil particulates, and vapor inhalation*

**vapor inhalation evaluated for volatile constituents only, as defined by chemicals with Henry's Law constants (H) greater than 10^{-5}*

Cumulative RBSL Equation: Cum-RBSL = 1 / [(1/SI-RBSL) + (1/DC-RBSL) + (1/INH-RBSL)]

where:

SI-RBSL = Soil Ingestion Risk-Based Screening Level
DC-RBSL = Dermal Contact Risk-Based Screening Level
INH-RBSL = Inhalation Risk-Based Screening Level

Soil Ingestion Component:

Soil Ingestion RBSL (SI-RBSL) in mg/kg =

$$\frac{THQ \times BW \times AT \times 365\ days/year \times RfDo}{EF \times ED \times 10^{-6}\ kg/mg \times IR_{soil}}$$

Dermal Contact Component:

Dermal Contact RBSL (DC-RBSL) in mg/kg =

$$\frac{THQ \times BW \times AT \times 365\ days/year \times RfDo}{EF \times ED \times 10^{-6}\ kg/mg \times SA \times AF \times RAF_d}$$

Inhalation Component:

Inhalation RBSL (INH-RBSL) in mg/kg =

$$\frac{THQ \times AT \times 365\ days/year}{EF \times ED \times [\ 1/RfC * (1/VF + 1/PEF)]}$$

(include VF component only for volatile chemicals with $H > 10^{-5}$)

Parameter	Acronym		Assumed Value	Units
Target Hazard Quotient	THQ		1.0	unitless
Body Weight	BW		70	kg
Averaging Time	AT		25	years
Exposure Frequency	EF		250	days/year
Exposure Duration	ED		25	years
Oral reference dose	RfDo		chem-specific	mg/kg/day
Soil ingestion rate	IR_{soil}		50	mg/day
Skin surface area	SA		5,800	cm^2/day
Soil to skin adherence factor	AF		1.0	mg/cm^2
Dermal relative absorption factor	RAF_d	*inorganics:*	0.001	unitless
		organics:	0.05	unitless
Inhalation reference concentration	RfC		chem-specific	mg/m^3
Soil-to-air volatilization factor	VF		chem-specific	m^3/kg (for chemicals with $H > 10^{-5}$)
Particulate emission factor	PEF		6.79E+08	m^3/kg

Source of equations and input parameters: EPA 1994 w/ exception of dermal component

Appendix Table B.2-2 Derivation of Soil RBSLs for Industrial Land-Use Scenario, Noncarcinogenic Endpoints -- Inorganic Constituents

Constituent	RfD (mg/kg/day)	RfC (mg/m^3)	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Aluminum	1.0E+00		2.0E+06	1.8E+07	--	1.8E+06
Antimony	4.00E-04	--	8.2E+02	7.0E+03	--	7.3E+02
Arsenic	3.00E-04	--	6.1E+02	5.3E+03	--	5.5E+02
Barium	7.00E-02	--	1.4E+05	1.2E+06	--	1.3E+05
Beryllium	5.00E-03	--	1.0E+04	8.8E+04	--	9.2E+03
Cadmium	5.00E-04	--	1.0E+03	8.8E+03	--	9.2E+02
Chromium III	1.00E+00	--	2.0E+06	1.8E+07	--	1.8E+06
Chromium VI	5.00E-03	--	1.0E+04	8.8E+04	--	9.2E+03
Copper	--	--	--	--	--	--
Cyanide	2.00E-02	--	4.1E+04	3.5E+05	--	3.7E+04
Lead	--	--	--	--	--	--
Manganese	5.00E-03	5.00E-05	1.0E+04	8.8E+04	5.0E+04	7.7E+03
Mercury	3.0E-04	3.00E-01	6.1E+02	5.3E+03	3.0E+08	5.5E+02
Nickel	2.00E-02	--	4.1E+04	3.5E+05	--	3.7E+04
Selenium	5.00E-03	--	1.0E+04	8.8E+04	--	9.2E+03
Silver	5.00E-03	--	1.0E+04	8.8E+04	--	9.2E+03
Thallium [1]	8.00E-05	--	1.6E+02	1.4E+03	--	1.5E+02
Vanadium [2]	9.00E-03	--	1.8E+04	1.6E+05	--	1.6E+04
Zinc	3.00E-01	--	6.1E+05	5.3E+06	--	5.5E+05

-- No toxicity data available.

[1] RfD for thallium is that reported in IRIS for thallium chloride (Oct 95).

[2] RfD for vanadium is that reported in IRIS for vanadium pentoxide (Oct 95).

Appendix Table B.2-2 Derivation of Soil RBSLs for Industrial Land-Use Scenario, Noncarcinogenic Endpoints -- Organic Non-Volatile Constituents

Constituent	H (atm-m^3/mol)	RfD (mg/kg/day)	RfC (mg/m^3)	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Bis(2-ethylhexyl) phthalate	8.36E-06	2.0E-02	--	4.1E+04	7.0E+03	--	6.0E+03

Appendix Table B.2-2 Derivation of Soil RBSLs for Industrial Land-Use Scenario, Noncarcinogenic Endpoints -- Organic Volatile Constituents

Constituent	H ($atm\text{-}m^3/mol$)	RfD (mg/kg/day)	RfC (mg/m^3)	SI-RBSL (ug/g)	DC-RBSL (ug/g)	INH-RBSL (ug/g)	Cumulative RBSL (ug/g)
Acetone	2.88E-05	1.0E-01	--	2.0E+05	3.5E+04	--	3.0E+04
PPDDT	5.37E-05	5.0E-04	--	1.0E+03	1.8E+02	--	1.5E+02
Methylene chloride	2.37E-03	6.0E-02	--	1.2E+05	2.1E+04	--	1.8E+04
Tetrachloroethane (1,1,2,2-)	3.72E-04	--	--	--	--	--	--
Toluene	6.14E-03	2.0E-01	4.0E-01	4.1E+05	7.0E+04	1.6E+03	1.6E+03
Xylenes	6.04E-03	2.0E+00	--	4.1E+06	7.0E+05	--	6.0E+05

-- Toxicity data not applicable.

Appendix Table B.2-2 Derivation of Soil RBSLs for Industrial Land-Use Scenario, Volatilization Factor (VF) and Soil Saturation Limit (SSL) Calculations

Volatilization Factor (VF) Equation

$VF = Q/C * (10^{-4}\ m^2/cm^2) * (3.14 * \alpha * T)^{1/2} / (2 * Dei * \theta_a * Kas)$

where:

$\alpha\ (cm^2/s) = (Dei * \theta_a) / (\theta_a + ((\rho s)(1 - \theta_a) / Kas))$

$Dei\ (cm^2/s) = Di * (\theta_a^{3.33} / n^2)$

$\theta_a = n - w\ \rho b = 0.28$ Lair/Lsoil

$n = 1 - (\rho b/\rho s) = 0.43$ Lpore/Lsoil

Soil Saturation Limit (Csat) Equation

$Csat = S/\rho b * [(Kd * \rho b) + \theta_w + (H' * \theta a)]$

where:		Units
S = solubility in water	= chemical-specific	mg/L-water
ρb = dry soil bulk density	= 1.5	kg/L
θ_w = water-filled soil porosity	= 0.15	L water / L soil
w = average soil moisture content	= 0.1 or 10 percent	unitless

VF Input Parameter Definitions

		Value	Units
VF = Volatilization Factor			m^3/kg
Q/C = inverse of the mean conc. at the center of a 30-acre-square source	=	35.1	g/m^2-s per kg/m^3
T = exposure interval	=	9.5E+08	s
Dei = effective diffusivity	=	$i * (\theta_a^{3.33}/n^2)$	cm^2/s
θ_a = air-filled soil porosity	=	0.28	Lair / Lsoil
Di = diffusivity in air	=	chemical-specific	cm^2/s
n = total soil porosity (for loam)	=	0.43	Lpore / Lsoil
w = average soil moisture content	=	0.10	cm3 water / g soil
ρb = dry soil bulk density		1.5	g/cm^3 or kg/L
ρs = soil particle density		2.65	g/cm^3
Kas = soil-air partition coefficient	=	H'/Kd	g-soil / cm^3-air
H = Henry's law constant	=	chemical-specific	atm-m^3/mol
H' = Henry's law constant, unitless	=	H * 41	unitless*
**41 is a units conversion factor (where units = mol/atm-m3)*			
Kd = soil-water partition coefficient	=	chemical-specific	cm^3/g or L/kg**
***or, in lieu of chem-specific data, Kd = Koc * foc*			
Koc = organic carbon partition coeffici	=	chem-specific	cm^3/g or L/kg
foc = organic carbon content of soil	=	0.006	g/g (0.6%)

Source of Equations and Default Input Parameters: ***EPA Draft Soil Screening Guidance (EPA/540/R-94/106, December 1994)***

Chemical-Specific Volatilization Factors (VFs) and Soil Saturation Limit (Csat) Derivations

Parameter	Di (cm^2/s)	Dei * (cm^2/s)	H (atm-m^3/mol)	H' (unitless)	Koc (cm^3/g)	Kd* (cm^3/g)	Kas * (g/cm3)	a * (cm^2/s)	S (mg/L-water)	VF (m^3/kg)	Csat (mg/kg)
Acetone	1.24E-01	9.7E-03	2.88E-05	1.2E-03	4.60E-01	2.76E-03	4.3E-01	5.7E-04	6.04E+05	2.0E+03	6.22E+04
PPDDT	1.37E-02	1.1E-03	5.37E-05	2.2E-03	2.37E+05	1.42E+03	1.5E-06	2.4E-10	3.41E-03	3.2E+06	4.85E+00
Methylene chloride	1.01E-01	7.9E-03	2.37E-03	9.7E-02	1.60E+01	9.60E-02	1.0E+00	1.0E-03	1.74E+04	1.4E+03	3.73E+03
Tetrachloroethane (1,1,2,2)	7.10E-02	5.5E-03	3.72E-04	1.5E-02	7.90E+01	4.74E-01	3.2E-02	2.6E-05	3.07E+03	9.8E+03	1.77E+03
Toluene	8.70E-02	6.8E-03	6.14E-03	2.5E-01	1.31E+02	7.86E-01	3.2E-01	3.0E-04	5.58E+02	2.7E+03	5.21E+02
Xylenes	7.20E-02	5.6E-03	6.04E-03	2.5E-01	2.60E+02	1.56E+00	1.6E-01	1.3E-04	1.86E+02	4.3E+03	3.17E+02

* refers to calculated value.

Where available, source of H values is EPA 1994, Table 5-5; otherwise taken from Table 2.6-1 of Interim Report

Source of Koc, Di, and S values is EPA 1994, Table 3-2, Chemical-Specific Properties Used in SSL Calculations

APPENDIX B.3

TOXICITY ASSESSMENT

SUPPORTING INFORMATION AND TOXICITY PROFILES

B.0 TOXICITY PROFILES

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B.2 ALUMINUM B.3-6

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B.1 ACETONE

General Background

Occurrence and Use

Acetone is an organic solvent with a wide variety of usages. It is one of the least hazardous chemical solvents. It is used as a nail polish remover; chemical intermediate for methyl methacrylate and methyl isobutyl ketone; manufacturing smokeless powder; and the production of modacrylic fibers.

Physical and Chemical Properties of Acetone

Molecular Weight	58.00[1]
Water Solubility, mg/l	1.0E+06[1]
Vapor Pressure, mm Hg	2.7E+02[2]
Henry Law Constant, atm-m^3/mole	2.1E-05[3]
Octanol-Water Partition Coefficient	5.8E-01[2]
Organic Carbon Partition Coefficient (L/g)	2.2E+00[1]
Bioaccumulation Factor for Fish	3.9E-01[1]
Bioaccumulation Factor for Shellfish	4.6E-02[1]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Verschueren, 1983
[3]ICF, 1987

Environmental Fate and Transport

Acetone is produced in large quantities and may be released to the environment as stack emissions, fugitive emissions, and in waste water in its production and use as a chemical intermediate and solvent. Most acetone used in solvents will be ultimately released into the air. Acetone is a product of the photooxidation of some alkanes and alkenes that are found in urban air and it is also released from volcanoes and forest fires. It is a metabolic product, released by plants and animals. If released on soil, acetone will both volatilize and leach into the ground and probably biodegrade. If released into water, acetone will probably biodegrade. It will also be lost due to volatilization (estimated half-life 20 hr from a model river). Bioconcentration in aquatic organisms and adsorption to sediment should not be significant. In the atmosphere, acetone will be lost by photolysis and reaction with photochemically produced

hydroxyl radicals. Half-life estimates from these combined processes average 22 days and are shorter in summer and longer in winter. It will also be washed out by rain (HSDB, 1993).

Pharmacokinetics: Routes of Exposure, Distribution, Absorption, Transport, and Degradation

Occupational exposure to acetone will be via dermal contact with solvents containing the chemical and via inhalation of the vapor. Acetone is one of the least hazardous industrial solvents, but it is highly volatile and is rapidly absorbed in the respiratory tract. Small quantities may also be absorbed through the skin (International Labor Office, 1983).

The general population is exposed to acetone in the atmosphere from sources such as auto exhaust, solvents, tobacco smoke, and fireplaces as well as from dermal contact with consumer products containing acetone such as solvents. In addition there will be exposure by ingestion of food that may naturally contain acetone or from contaminated drinking water (HSDB, 1993).

Because of its solubility in water, acetone is readily absorbed into the bloodstream and thus is transported rapidly throughout the body. Excretion is rapid for 8 hours after a single oral dose but was not complete in 24 hours. The ratio of excretion was approximately 40-70% in breath, 15-30% in urine, and 10% of the total through skin (Clayton and Clayton, 1981).

Non-Carcinogenic Health Effects

Acute Exposure

Humans: Human exposure to high concentrations of acetone produces central nervous system depression and unconsciousness. The toxic concentration of acetone in human blood is 200-300 μg/ml with the lethal concentration being 550 μg/ml (Winek, 1985). Inhalation of 2,000 ppm acetone is fatal upon brief exposure (Arena, 1974).

Laboratory Animals: Limited information is available on the health effects of acetone in animals. Acute inhalation exposure to rats may lead to damage to the nervous system. It is also known that corneal damage and edema are common symptoms following exposure to acetone in rabbits (Patty's 1982).

Chronic Exposure

Humans: Prolonged or repeated skin exposure can cause skin irritations or contact dermatitis (HSDB 1995). Chronic inhalation exposure at high concentrations may lead to a number of neurological and respiratory symptoms ranging from respiratory tract irritation, coughing, drowsiness, and headaches to loss of coordination, and, in severe cases, coma (Clement Associates 1985). Other common symptoms associated with repeated exposure to acetone include bronchitis, gastritis, phyarynagitis, and conjunctivitis (HSDB 1995).

Laboratory Animals: Long-term exposure of rats to low levels of acetone produces increases in liver and kidney weights (may be indicative of disease), as well as changes in red blood cell counts (HSDB 1995). Goldberg et al (1964) noted incoordination and difficulty climbing after rats were chronically exposed to acetone.

Carcinogenic Health Effects:

Acute Exposure

Humans: No information is available on the carcinogenic effects of acetone in humans.

Laboratory animals: Acetone has not been tested in a carcinogenicity bioassay, but gave negative results in a skin painting test (Clement Associates, 1985).

Chronic Exposure

Humans: No information is available on the carcinogenic effects of acetone in humans.

Laboratory animals: No information is available on the carcinogenic effects of acetone in animals.

Mutagenicity and Teratogenicity

Mutagenicity:

Little information is available on the mutagenic activity of acetone. Acetone was not mutagenic in the Ames assay (Clement Associates, 1985), or in sister chromatid exchange assays (HSDB, 1993).

Teratogenicity and other reproductive/developmental effects:

Acetone (0.10 ml/egg injected) significantly reduced the percentage of hatchability in developing chick embryos and caused a high embryonic mortality during the first week of incubation (Ameenuddin and Sunde, 1984). Kitchen and Ebron (1984) cultured 10.5-day-old rat embryos for two days in whole rat serum containing 0.1% acetone, noting no adverse toxic or teratogenic effects (HSDB, 1993).

EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

Acetone is classified as a group D carcinogen; not classifiable as to human carcinogenicity due to lack of evidence.

EPA Dose-Response Parameters (IRIS, 1993):

ORAL RFD : 1E-1 mg/kg/day

CRITICAL EFFECT/TARGET ORGAN: Increased liver and kidney weights and nephrotoxicity

ORAL RFD UNCERTAINTY : UF = 1000.

An uncertainty factor of 1000 is used; 100 for inter- and intraspecies extrapolation and 10 to extrapolate from subchronic to chronic exposure.

ORAL RFD MODIFYING FACTOR : MF = 1.

ORAL RFD CONFIDENCE :
Study: Medium
Data Base: Low
RfD: Low

Confidence in the principal study is rated medium, since a moderate number of animals/dose/sex and an extensive number of parameters were measured. The data base is rated low because a very limited number of studies are available and no pertinent supporting studies were located. The overall confidence rating for the RfD is low.

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B.2 ALUMINUM

I. Use and Occurrence

Aluminum is a metallic element that is a major constituent of the earth's crust typically in the +3 oxidation state, but does not exist naturally in the elemental form (USEPA, 1987). It is used in construction and consumer products, and its compounds are used as constituents in paint, and as coagulants in industrial processes. It is also used in food processes, cosmetics, and therapeutics. Aluminum commonly combines with oxygen, fluorine, and silicon to forma a variety of compounds (USEPA, 1987).

II. Physical and Chemical Properties of Aluminum

Molecular Weight	26.98
Water Solubility, mg/l	0.0E+00
Bioaccumulation Factor for Fish	1.0E+01
Bioaccumulation Factor for Shellfish	6.3E+01

Source: Multi-Media Exposure Assessment Manual, 1989

III. Environmental Fate and Transport

Naturally occurring levels of aluminum may show considerable variability as demonstrated by the wide range of concentrations (i.e., 700-10,000 mg/g) reported for soils of the eastern United States (Shackelette and Boerngen, 1984). Levels of elemental aluminum in the ambient air over the continental United States have been reported to range from 0.14-8.0 mg/m^3 (USEPA, 1985). Combustion emissions of aluminum consist primarily of aluminum oxide particles which are removed by settling, and precipitation washout (USEPA, 1987). The level of aluminum in natural waters varies geographically. In waters where the pH is less than 5.0, as with industrial wastes, mine runoff, acidic spring waters, mires and volcanic areas, the aluminum level can exceed 100 mg/l (USEPA, 1987). With more alkaline Ph levels, the solubility of aluminum decreases.

IV. Routes of Exposure, Distribution, Absorption, Transport and Degradation

There are significant differences in gastrointestinal absorption and bioavailability of aluminum, depending on its speciation (USEPA, 1992). There are sufficient data to demonstrate that aluminum is absorbed from the gastrointestinal tract, but bioavailability and mechanisms of absorption are not known (Wilhelm et al., 1990). The amount of aluminum that is absorbed is dependent on the chemical form, Ph of the intestine, concentration of

aluminum, and dietary factors. Yokel and McNamara (1988) administered single doses of 8 inorganic and organic aluminum compounds to rabbits. The compounds tested, in order of increasing absorption were aluminum borate, aluminum glycinate, aluminum hydroxide, aluminum chloride, sucralfate, aluminum lactate, aluminum nitrate, and aluminum citrate. Absorption ranged from 0.27 to 2.18%. A higher percent was absorbed from a large dose of aluminum than from a lower dose (van der Voet and de Wolff, 1986). Dietary factors such as phosphate, citrate, and fluoride with which aluminum can form complexes also influence absorption.

Aluminum accumulates in the lungs of exposed humans and contains relatively high levels compared to other tissues (USEPA, 1987). Studies have not conclusively determined whether aluminum is absorbed across the pulmonary lining since they failed to measure blood aluminum levels.

V. Acute Toxicity

Intraperitoneal injections and oral dosing of rats with high doses of aluminum hydroxide, aluminum chloride and aluminum sulfate caused lethargy, anorexia, and in several cases, death (Carson et al., 1986). Particles of aluminum metal deposited in the eye may cause necrosis (tissue destruction) of the cornea (Sittig, 1985). Salts of aluminum may cause dermatoses (skin disorders), eczema, conjunctivitis (eye irritation), and irritation of the mucosa membranes of the upper respiratory system (Sittig, 1985). Data suggest that pneumoconiosis (lung disease) might also be a possible outcome. In the majority of cases of pulmonary effects investigated, however, past exposures were to very high concentrations not limited to aluminum dust alone, but to a mixture of aluminum, silica fume, iron dusts, and other pulmonary irritating materials (Sittig, 1985). In general, aluminum is considered to possess low acute toxicity for the normal individual following oral exposure (USEPA, 1985). Oral LD_{50}s in several animal species ranged from 380 to 780 mg/kg (USEPA, 1985).

VI. Chronic Toxicity

The greatest health concern regarding chronic exposure to aluminum is its neurological effects. The first evidence for aluminum induced neurotoxicity in humans was demonstrated by Alfrey et al (1972) who discovered that patients receiving long term hemodialysis for chronic renal failure developed a degenerative neurological syndrome (Dialysis Dementia). This disease was attributed to the presence of aluminum in the dialysate. Aluminum has also been implicated as a contributing cause of other chronic diseases including Amyotrophic Lateral sclerosis (ALS), Parkinson's Disease (PD) and Alzheimer's Disease (AD). However, the link between AD and aluminum exposure is currently being disputed. Animals given intracerebral injections of aluminum phosphate or powder display a progressive encephalopathy (brain degeneration) and neurofibrillary tangles that are rich in aluminum and histologically resemble those changes observed in persons with Alzheimers disease (Goyer, 1986). Also, high brain aluminum levels have been associated with encephalopathy in elderly

and in Alzheimers patients (USEPA, 1987). However, this interaction is not resolved since it is not known whether aluminum in the neurofibrillary tangles are a direct cause or an effect, and also, why certain individuals are more susceptible to a ubiquitous metal has not been adequately addressed (Goyer, 1986).

A second target organ for aluminum in both humans and laboratory animals is bone. Several studies have shown that aluminum exposure may cause osteomalacia, a condition characterized by low bone formation. In addition to the direct deposition of aluminum is the bone tissue, aluminum may induce osteomalacia by forming insoluble complexes with phosphates in the GI tract. These complexes are no easily absorbed and long term exposure to aluminum may result in hypophosphatemia which intern leads to hypercalciuria and bone resorption. The decreased phosphate absorption may also affect other physiological systems involving phosphorylation (USEPA, 1992).

Aluminum has also been shown to produce adverse hematological effects by causing a decrease in heme synthesis by interfering with iron metabolism or the biosynthesis of protoporphyrin (USEPA, 1992).

Animal studies have indicated a symptomatic pattern typical of nuisance particulate of bronchopneumonia, edema, emphysema, and/or severe fibrosis from inhalation of powdered aluminum (Proctor et al., 1988). Chronic use of large oral doses of aluminum (i.e., antacids) in humans reduces phosphate levels and leads to phosphate depletion in the long term (WHO, 1984; Proctor et al., 1988). A chronic feeding study of rats fed aluminum phosphide-fumigated chow with an average concentration of 0.51 mg phosphide/kg for a 2-year period produced no differences between control and treated animals (USEPA, 1989a). Administration of elemental aluminum to rats at the rate of 2.5 mg/kg body weight per day over a six-month period resulted in minimal systematic toxicity (WHO, 1984).

VII. Mutagenicity, Carcinogenicity and Teratogenicity

Mutagenicity

Elemental aluminum was not mutagenic in the Ames assay using Salmonella typhimurium (strains TA98, TA1535, and TA1538) (USEPA, 1987). Aluminum chloride produced negative results in a DNA damage/repair assay using Bacillus subtillus, but it did produce chromatid breaks and gaps in mouse bone marrow cells in vitro (USEPA, 1987).

Carcinogenicity

Pertinent data could not be located regarding the carcinogenicity of elemental aluminum following oral exposure (USEPA, 1987). Occupational exposure (i.e., inhalation) to

aluminum has not been associated with pulmonary or systemic neoplastic alterations in humans (USEPA, 1987).

Several animal studies have been conducted evaluating the carcinogenicity of aluminum compounds. Aluminum potassium sulfate administered in the drinking water of rats and mice at concentrations of 5 mg/l (free base weight) for life resulted in a significant increase in total tumors (all sites; unspecified) in male rats, while female mice experienced a significantly increased incidence of lymphoma leukemia (USEPA, 1987). In an inhalation study, granulomas developed in the lungs of rats and guinea pigs that were exposed to 2.5 mg/m^3 aluminum chlorohydrate, 6 hours/day, 5 days/week for 12 months or 25 mg/m^3 for 6 months (USEPA, 1987). These responses cannot be attributed solely to aluminum because the actual structure of aluminum chlorohydrate is not known but is thought to be a complex aluminum chlorohydrate and propylene glycol, which may have been a contributing factor (USEPA, 1987). Based on limited evidence of carcinogenic activity in animals, the EPA has categorized aluminum as a possible human carcinogen (Group C) (USEPA, 1987).

Teratogenicity (and other reproductive effects)

Oral administration of aluminum chloride in drinking water to rats revealed no effects on reproductive capacity. Endpoints evaluated in the study included pregnancy rate, the number of implantation sites, corpora lutea and resorption sites, and the number of live and dead implants (USEPA, 1987). No evidence of fetal malformations has been observed in animals following oral exposure (USEPA, 1985). In another study, decreased spermatozoa counts and sperm motility occurred in rats fed 2.5 mg/kg/day of aluminum chloride for six months (USEPA, 1987). These effects were not observed at lower doses. Histological and histochemical alterations in the testes were also observed, however, aspects of this study are considered inadequate (USEPA, 1987). Low birth weights were observed in pregnant rats fed tap water mixed with Maalox (an antacid therapeutic) in a 1:4 ratio. However, water intake and aluminum intake were not measured. Body weights of pups cross-fostered from nonexposed females recovered. Reproductive data related to the inhalation of aluminum could not be found in the available literature (USEPA, 1987).

VIII. EPA Carcinogenic Classification and Dose-Response Parameters

The EPA has not a carcinogenic classification for aluminum, due to the fact that definitive conclusions can not be drawn from the available data.

EPA Dose-Response Parameters:

An interim Rfd has ben issued for aluminum by the USEPA's Environmental Criteria and Assessment Office. The RfD is based on a study conducted by Donald et al. (1989), who identified an LOAEL of 100 mg/kg/day for minimal neurotoxicity in the offspring of mice exposed to aluminum lactate in the diet during gestation and lactation. Application of an

uncertainty factor of 100 (3 for use of a minimal LOAEL, 10 for interspecies extrapolation, and 3 to protect sensitive individuals) results in a provisional Rfd of 1 mg/kg/day. Medium confidence is placed in the critical study. It identified a LOAEL for a sensitive effect, however, a small sample size was used. Confidence in the database is medium. It should be noted that large differences (>10 fold) in the bioavailability of aluminum exist. The aluminum form must be considered when using this RfD.

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B.3 ANTIMONY

I. Occurrence and Use

ntimony (Sb) is a naturally occurring metal found in a tri- or pentavalent state. Antimony is frequently associated with sulfide and sulfide ores (Carson et al., 1986). Antimony and compounds are primarily used in the production of lead, copper and other metal alloys, as well as commercially in fireproofing textiles, ceramics, glassware, pigments, and in antiparasitic drugs (Carson et al., 1986). Antimony is a common industrial air pollutant, but the general public is largely exposed to antimony from food (Goyer, 1986).

Soil antimony concentrations typically range from 0.1 to 10 mg/kg (dry weight) (Elinder and Friberg, 1986). Air concentrations in the Chicago area have ranged between 1.4 to 55 ng/m^3, with a mean of 32 ng/m^3.

II. Physical and Chemical Properties of Antimony

Molecular Weight	122.00
Water Solubility, mg/l	0.0E+00
Bioaccumulation Factor for Fish	1.0E+00
Bioaccumulation Factor for Shellfish	1.0E+01

Sources: Multi-Media Exposure Assessment Manual, 1989

III. Environmental Fate and Transport

Various forms of antimony found in the environment from natural and anthropogenic sources undergo a complex cycle of chemical interconversion and transfer between media. antimony in water may undergo either oxidation or reduction, depending on Ph and other ions present. Soluble forms of Sb (e.g., antimony potassium oxalate and antimony potassium tartrate) tend to by quite mobile in water, while less soluble species adsorb to clay or soil particles (Callahan et al., 1979).

Antimony in gaseous, vapor and particulate forms enters the atmosphere and is transported via air until it undergoes atmospheric fallout or washout and is deposited in oceans, estuaries, lakes, rivers, sediments and terrestrial systems. Antimony may enter the food chain via root uptake by terrestrial plants and via bioaccumulation in fish- and plant-eating mammals. Antimony deposited in sediment can also be released to the atmosphere through microbial activity under anaerobic conditions. Antimony may leach from municipal landfills, sewage

sludge, oil-fired plant incinerator ash and fertilizers to contaminate ground water, surface water and sediment (Callahan et al., 1979).

IV. Routes of Exposure, Distribution, Absorption, Transport and Degradation

According to U.S. EPA (1980), multimedia antimony exposures are essentially negligible by comparison to occupational exposures at which discrete clinical health effects have been observed. Myocardial effects are among the best-characterized human health effects associated with antimony exposure.

Quantitative estimates on the efficiency of pulmonary absorption of antimony are not available, but Elinder and Friberg (1986) state that trivalent antimony is absorbed from the lungs to a large extent. Absorption from the gastrointestinal (GI) trace is slow and it has been reported that at least 15 percent of a single oral dose of antimony potassium tartrate was absorbed by mice (Waitz et al., 1965). Once absorbed, the highest concentrations are found in the thyroid, adrenals, liver, heart and kidneys (Carson et al., 1986). Elimination of antimony is somewhat rapid depending on route and valence state but occurs via both feces and urine. The typical human daily intake of antimony, from all sources ranges between 10 and 1250 μg (Elinder and Friberg, 1986).

V. Acute Toxicity

The primary effect of acute antimony exposure is direct irritation of tissues (Sittig, 1985). Acute inhalation exposures elicited an irritative effect on the upper respiratory tract of workers exposed to 73 mg/m^3 antimony trichloride (Elinder and Friberg, 1986) Exposes to high levels of antimony fumes are capable of producing GI effects of abdominal cramps, diarrhea, and vomiting (Carson et al., 1986). In severe cases, pulmonary edema and even death have been seen in exposed workers. Other effects seen are rhinitis (nasal mucous irritation) and skin irritation, which may lead to lesions in moist exposed areas of the body (Sittig, 1985). Experimental animals administered an intravenous injection of antimony displayed circulatory and cardiac alterations (Carson et al., 1986).

VI. Chronic Toxicity

Antimony tends to accumulate in the lung following inhalation exposures where chronic respiratory tract symptoms of pharyngitis and tracheitis are seen (Goyer, 1986). If exposures persist, these irritation systems may progress to bronchitis, pneumoconiosis, obstructive pulmonary disease, and emphysema (Goyer, 1986). These pulmonary effects can be observed visually as changes in chest x-rays (characterized by densely distributed opacities (Elinder and Friberg, 1986). Chronic occupational exposures to antimony trioxide have been associated with heart disease with occasional fatalities (Carson et al., 1986). Pustular skin eruptions ("antimony spots") in exposed workers are sometimes seen in employees working with antimony compounds (Elinder and Friberg, 1986). For years antimony was used in

anti-parasitic therapy (principally for schistosomiasis) where some of the above side effects were noted as well as elevation of liver enzymes (GOT and GPOT) in some patients at the early stages of therapy (Elinder and Friberg, 1986).

VII. Mutagenicity, Carcinogenicity and Teratogenicity

Mutagenicity

Several antimony compounds were found to be mutagenic in Bacillus subtillus (Carson et al., 1986). Increased chromosomal defects were observed in human lymphocytes and Syrian hamster embryo cells incubated in antimony compounds (Paton and Allison, 1975; Casto et al., 1979).

Carcinogenicity

There is very little data on possible human carcinogenicity of antimony compounds. The American Conference of Governmental Industrial Hygienists (ACGIH) concluded in 1983 that antimony oxide should be regarded as a suspected carcinogen based on unpublished data obtained from a large antimony smelter in the U.K. which showed an increased incidence of mortality from lung cancer in heavily exposed workers (ACGIH, 1983). However, Elinder and Friberg (1986) state that in this study other chemical exposures occurred which make interpretation of this data from the U.K. difficult. In addition, a high frequency of lung neoplasias was observed in rats exposed to airborne antimony at a concentration of 4.2 mg/m^3 (Watt, 1983), while oral dosing of rats has not produced any excess of tumors (Goyer, 1986). Antimony has not been evaluated by the EPA for carcinogenicity, therefore a carcinogenic classification has not been determined (USEPA, 1989a).

Teratogenicity (and other reproductive effects)

A 1967 Russian study reported an 8 percent increase of spontaneous late abortions in female antimony smelter workers compared to an unexposed population control (Carson et al., 1986). Infant weights from exposed mothers were not birth differential but were significantly lower when measured at one year. Other studies have reported a slight increase in premature deliveries for female antimony workers exposed during pregnancy (Carson et al., 1986). Experimental animals have experienced uterine and ovarian disorders when exposed to antimony, but no cases of fetal malformation have been reported in pregnant rats exposed to 125 or 250 mg/kg antimony (route unknown) (Carson et al., 1986).

VIII. EPA Carcinogenic Classification and Dose-Response Parameters

The following dose-response parameters and discussions were extracted from IRIS, 1994.

EPA Carcinogenic Classification:

The EPA has not classified Antimony in terms of carcinogenicity, and no cancer dose-response parameters have been derived.

Dose Response Parameters (IRIS, 1994):

Carcinogenic Effects: No data

Noncarcinogenic Effects:

ORAL RFD SUMMARY :
RfD: 4E-04 mg/kg/day
STUDY USED TO DERIVE RfD:
Shroeder, H.A., M. Mitchner and A.P. Nasor. 1970. Zirconium, niobium, antimony, vanadium and lead in rats: Life term studies. J. Nutrition. 100:59-66.

ORAL RFD UNCERTAINTY:
UF = 1000. An uncertainty factor of 1000 (10 for interspecies conversion, 10 to protect sensitive individuals, and 10 because the effect level was a LOAEL and no NOEL was established) was applied to the LOAEL of 0.35 mg/kg bw/day.

ORAL RFD MODIFYING FACTOR:
MF = 1.

ORAL RFD CONFIDENCE:
Study: Low
Data Base: Low
RfD: Low

Confidence in the chosen study is rated as low because only one species was used, only one dose level was used, no NOEL was determined, and gross pathology and histopathology were not well described. Confidence in the data base is low due to lack of adequate oral exposure investigations. Low confidence in the RfD follows.

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B.4 ARSENIC

I. Occurrence and Use

Arsenic is a component of manufactured metal alloys, electrical devices, glass, wood preservatives, agricultural chemicals, and is also used as a therapeutic agent. The element is distributed widely in natural soils; typical concentrations is U.S. soils have been found to be between >1 and about 30 mg/kg (Kabata-Pendias and Pendias, 1987). Most arsenic releases to the environment occur as byproducts of metal smelting and refining activities.

II. Physical and Chemical Properties of Arsenic

Molecular Weight	75.00[1]
Water Solubility, mg/l	0.0E+00[2]
Vapor Pressure, mm HG	0.0E+00[3]
Bioaccumulation Factor for Fish	1.0E+00[4]
Bioaccumulation Factor for Shellfish	4.0E+00[5]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Weast, 1979
[3]EPA, 1981
[4]Spehar et al., 1980
[5]Strenge et al., 1986

III. Environmental Fate and Transport

In the natural environment arsenic has four different oxidation states;chemical speciation is important in determining arsenic's distribution and mobility. Interconversions of the +3 and +5 states as will as organic complexation do occur and can be mediated by microorganisms. Arsenic is generally quite mobile in the environment and is mainly transported by water (WHOO, 1981). In oxygenated water, arsenic usually occurs as arsenate, but under reducing conditions (i.e., deep will waters) arsenite predominates. In the aquatic environment, volatilization is important when biological activity or highly reducing conditions produce arsine of methyl-arsenics. Sedimentation of arsenic in association with iron and aluminum does occur frequently (SHO, 1981).

Significant sources of As in soils are related to industrial activities such as metal processing, coal combustion, geothermal power production, and to the use of arsenical herbicides. The reactions of As in soils are highly governed by its oxidized state. However, arsenate ions are

known to be readily fixed by such soil components as clays, phosphatic gels, humus, and calcium, and the most active in As retention are hydrated Fe and Al oxides. In oxygenated soil, inorganic arsenic is prevalent in the pentavalent (+5) form. Under reducing conditions, the trivalent form predominated (WHO, 1981). Hydroxy-Al on the external surfaces of micaceous minerals is reported to be especially significant in the retention of As. The mobility of As in soil was shown to be proportional to the As added and inversely proportional to time and to Fe and Al contents. The toxicity of As depends on the concentration of soluble As, therefore, sodium arsenate and arsenic trioxide, formerly used as herbicides, are the most toxic (Kabata-Pendias and Pendias, 1987).

IV. Routes of Exposure, Distribution, Absorption, Transport, and Degradation

Arsenic is readily absorbed via the oral and inhalation routes. Both inorganic and organic forms of arsenic are readily absorbed from the gastrointestinal tract with the more soluble forms being more readily absorbed than the insoluble forms (USEPA, 1984). Approximately 95 percent of soluble inorganic arsenic administered to rats is absorbed from the gastrointestinal tract (Coulson et al., 1935; Ray-Bettley and O'Shea, 1975). The USEPA (1984) assumes that, on the average, 70-80 percent of arsenic is absorbed in the respiratory tract. Dermal absorption is not significant (USEPA, 1984).

V. Acute Toxicity

Acute exposure of humans to high levels (71 mg/kg) of metalloid arsenic has been associated with gastrointestinal effects, hemolysis, and neuropathy.

VI. Chronic Toxicity

Chronic exposure of humans to arsenic can produce toxic effects on both the peripheral and central nervous systems, keratosis, hyperpigmentation, precancerous dermal lesions and cardiovascular damage (USEPA, 1984).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity:

Current existing evidence suggests that arsenic is mutagenic. Although bioassays of laboratory animals have not as yet revealed an ability to arsenic to cause chromosome aberrations, some studies suggest that humans exposed to arsenic exhibit elevated frequencies of sister chromatid exchanges and chromosome aberrations. Arsenic may be substituted for phosphorus in the DNA backbone, and may inhibit DNA repair mechanisms (ATSDR, 1989)

Carcinogenicity:

Arsenic is a known human carcinogen. Epidemiological studies of workers in smelters and in plants manufacturing arsenical pesticides have shown that inhalation of arsenic is strongly associated with lung cancer and perhaps with hepatic angiosarcoma (USEPA, 1984). Ingestion of arsenic has been linked to a form of skin cancer and more recently to bladder, liver and lung cancers (Tseng et al., 1968; Chen et al., 1986).

Teratogenicity (and other reproductive effects):

Arsenic is embryotoxic, fetotoxic, and teratogenic in several animal species (USEPA, 1984).

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

The following dose-response parameters and discussions were extracted from IRIS, 1994.

EPA Carcinogenic Classification:

Arsenic is classified as a known human carcinogen (Group A). This classification is based on observation of increased lung cancer mortality in populations exposed primarily through inhalation and on increased skin cancer incidence in several populations consuming drinking water with high arsenic concentrations.

Dose-Response Parameters (IRIS, 1994):

Carcinogenic Effects:

INHALATION UNIT RISK: 4.3E-3/μg/cu.m

INGESTION UNIT RISK: 1.75 mg/kg/day

Noncarcinogenic Effects:

ORAL RFD SUMMARY :
RfD: 3E-4 mg/kg/day

Critical effect: keratosis, hyperpigmentation, and possible vascular complications.

NOTE:There was not a clear consensus among Agency scientists on the oral RfD. Applying the Agency's RfD methodology, strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently recommended RfD value, i.e., 0.1 to 0.8 μg/kg/day. It should be noted, however, that the RfD methodology, by definition, yields a number with inherent uncertainty spanning perhaps an order of magnitude. New data that possibly impact

on the recommended RfD for arsenic will be evaluated by the Work Group as it becomes available. Risk managers should recognize the considerable flexibility afforded them in formulating regulatory decisions when uncertainty and lack of clear consensus are taken into account.

Conversion Factors: NOAEL was based on an arithmetic mean of 0.009 mg/L in a range of arsenic concentration of 0.001 to 0.017 mg/L. This NOAEL also included estimation of arsenic from food. Since experimental data were missing, arsenic concentrations in sweet potatoes and rice were estimated as 0.002 mg/day. Other assumptions included consumption of 4.5 L water/day and 55 kg bw (Abernathy et al., 1989). NOAEL = [(0.009 mg/L x 4.5 L/day) + 0.002 mg/day] / 55 kg = 0.0008 mg/kg/day. The LOAEL dose was estimated using the same assumptions as the NOAEL starting with an arithmetic mean water concentration from Tseng (1977) of 0.17 mg/L. LOAEL = [(0.17 mg/L x 4.5 L/day) + 0.002 mg/day] / 55 kg = 0.014 mg/kg/day.

ORAL RFD UNCERTAINTY :
UF = 3. The UF of 3 is to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals.

ORAL RFD MODIFYING FACTOR :
MF = 1.

ORAL RFD CONFIDENCE :
Study: Medium
Data Base: Medium
RfD: Medium

Confidence in the chosen study is considered medium. An extremely large number of people were included in the assessment (>40,000) but the doses were not well-characterized and other contaminants were present. The supporting human toxicity data base is extensive but somewhat flawed. Problems exist with all of the epidemiological studies. For example, the Tseng studies do not look at potential exposure from food or other source. A similar criticism can be made of the Cebrian et al. (1983) study. The U.S. studies are too small in number to resolve several issues. However, the data base does support the choice of NOAEL. It garners medium confidence. Medium confidence in the RfD follows.

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B.5 BARIUM

I. Occurrence and Use

Barium is a relatively abundant naturally occurring metallic element which constitutes about 0.04% of the earth's crust, with the greatest occurrence in rock formations. Naturally occurring concentrations in soils may vary considerably, as indicated by one report of a 10-1500 ppm range in background concentrations. Barium releases to the environment occur as the result of many anthropogenic activities, such as drilling for oil and gas, and the burning of fossil fuels. Barium is also used in the manufacture of fillers for automotive paints, and specialty compounds used in bricks, tiles and jet fuels (ATSDR, 1989). which accumulates in plants and animals.

II. Physical and Chemical Properties of Barium

Molecular Weight	137.00[1]
Water Solubility, mg/l	0.0E+00[2]
Bioaccumulation Factor for Fish	4.0E+00[3]
Bioaccumulation Factor for Shellfish	2.0E-01[4]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Weast, 1979
[3]Napier et al., 1980
[4]Guthrie et al., 1979

III. Environmental Fate and Transport

Barium is emitted into the atmosphere mainly by the industrial processes involved in mining, refining, and production of barium and barium based chemicals and as a result of combustion of coal and oil (HSDB, 1992).

Current evidence suggests that barium has the ability to accumulate in both plants and animals. Barium has been found in essentially all biological materials assayed. Marine animals have been reported to concentrate the element 7-100 times, and marine plants up to 1000 times the concentration in seawater in which they grow. Barium content of edible crops has been reported to range from 10 μg/g detected in wheat and corn to 3-4 mg/g detected in brazil nuts. Typical barium concentrations in milk have been reported to be 45-136 μg/g, in wheat flour 1,300 μg/g, and in oatmeal 2,320-8290 μg/g (ATSDR, 1989).

IV. Routes of Exposure, Distribution, Absorption, Transport, and Degradation

Soluble compounds of barium are known to be gastrointestinally absorbed in the human body, and their toxicities are correlated with their solubility. The highest concentration of normal levels of barium in the human body accumulate in the skeleton (2 μg/g; 90% of the total barium body burden), with lesser amounts accumulating in other organs such as the eye, lungs, connective tissue, skin, and adipose tissue. Mean concentrations have been reported to be in the range of 0.10 ppm in the kidneys; 0.08 ppm in the spleen; 0.05 ppm in muscle, including cardiac muscle tissues; 0.05 ppm in the brain; and 0.03 ppm in the liver (HSDB, 1992).

V. Acute Toxicity

Initial symptoms of barium poisoning are gastrointestinal disorders, including nausea, vomiting, colic, and diarrhea, followed by myocardial (heart muscle) stimulation and general muscular stimulation with tingling in the extremities. Severe cases progress to a loss of tendon reflexes, general muscular paralysis, and death from respiratory arrest or ventricular fibrillation (irregular heartbeat). (Proctor et al., 1988). The barium ion is a physical antagonist of potassium in vivo, and symptoms of barium poisoning are attributable to hypokalemia (lack of potassium in the blood). The effect is probably due to a transfer of potassium from extracellular to intracellular compartments rather than to urinary or gastrointestinal losses.

VI. Chronic Toxicity

Studies attempting to demonstrate a link between consumption of barium-contaminated drinking water and the incidence of hypertension are inconclusive (ATSDR, 1989). No other chronic effects have been reported.

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity:

Inadequate studies exist to evaluate the potential mutagenic effects of barium. (USEPA, 1985; Klaassen et al., 1986).

Carcinogenicity:

Current evidence suggests that barium is not a carcinogen. Statistically significant increases in total tumors are reported to be absent (ATSDR, 1989).

Teratogenicity:

Inadequate studies exist to evaluate the potential teratogenic/reproductive effects of barium. (USEPA, 1985; Klaassen et al., 1986).

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification (IRIS, 1994):

Barium is classified as a member of Group D (not classifiable as to human carcinogenicity), based on perceived inadequacy of data derived from animal studies.

EPA Dose-Response Parameters:

Carcinogenic Effects:

No carcinogenic dose-response parameters have been promulgated for barium.

Noncarcinogenic Effects (IRIS, 1994):

ORAL RFD SUMMARY :
RfD: 0.07 mg/kg/day

Critical effect: Increased blood pressure

ORAL RFD UNCERTAINTY:
UF = 3. According to U.S. EPA guidelines, an uncertainty factor of 10 is applied when a NOAEL from a subchronic human study is employed. However, data are available from chronic human studies which support this NOAEL, as well as several oral chronic animal studies. Therefore, this UF is not considered necessary. In addition, another factor of 10 is used with a human study to protect sensitive individuals. However, the data base supports the finding that the critical effect is hypertension which results from long exposure durations, and that the population most at risk is the adult male. Furthermore, the chosen study is a careful observation of this critical effect in adult males. Because of both the critical study's unique focus and the supporting studies, a 3-fold UF, instead of a 10-fold UF, was chosen as most appropriate to protect for sensitive individuals within that population.

ORAL RFD MODIFYING FACTOR :
MF = 1.

ORAL RFD CONFIDENCE :
Study: Medium
Data Base: Medium
RfD: Medium

As previously stated, EPA does not believe that any single study, considered alone, is adequate to calculate an RfD for barium. However, EPA believes that medium confidence can be placed in the total data base used to determine the RfD.

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B.6 BENZO(A)PYRENE, CHRYSENE, AND PYRENE (POLYCYCLIC AROMATIC HYDROCARBONS)

I. General Background

II. Physical/Chemical Properties:

Polyclic aromatic hydrocarbons (PAHs) are a class of compounds that are formed during the incomplete combustion or pyrolysis of organic materials containing carbon and hydrogen. Several hundred different PAHs have been identified from combustion and pyrolysis sources (Grimmer, 1983). In this discussion, only 15 PAHs will be considered. These include the following: acenaphthene, acenaphthylene, anthracene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, fluoranthene, fluorene, indeno(1,2,3-cd)pyrene, phenanthrene and pyrene.

The molecular weight of the aforementioned PAHs range from 154.2 to 278.35. They exist as solids, plates, needles, tablets or prisms having a melting point of 92 °C to 273 °C and a boiling point of 96.2 °C to 495 °C. Their water solubilities vary from being insoluble to 3.93 mg/l in water. Their octanol-water partition coefficients vary from 5.3×10^{3} to 6.9×10^{6}. Their vapor pressures vary from 1×10^{-10}mm Hg (20 °C) to 10 mm Hg (146 °C) and from 10^{-11} torr (20 °C) to 9.6×10^{-4} torr (25 °C). The Henry's law constant varies from 7.3×10^{-8} to 1.45×10^{-3} (HHS, 1990).

Environmental Fate Characteristics:

In surface water, PAHs can volatilize, photodegrade, oxidize, biodegrade, bind to particulates, or accumulate in aquatic organisms. In sediments, PAHs can biodegrade or accumulate in aquatic organisms. PAHs in soil can biodegrade or accumulate in plants; PAHs can enter groundwater and be transported within an aquifer.

Transport and partitioning of PAHs in the environment are determined to a large extent by physical/chemical properties such as water solubility, vapor pressure, Henry's law constant, octanol-water partition coefficient (K_{ow}). In general, PAHs have low water solubilities.

PAHs are present in the atmosphere in the gaseous phase or sorbed to particulates. PAHs in urban air are primarily associated with submicron diameter soot particles that have residence times of weeks and are subject to long-range transport (Butler and Crossley 1981). PAHs are removed from the atmosphere by both wet and dry deposition; the relative importance of each process varies with the individual PAH. For example, Perwak, et al (1982) estimated that a total of 23% of benzo(a)pyrene released to the atmosphere is deposited on soil and water

surfaces. Dry deposition of benzo(a)pyrene adsorbed onto atmospheric aerosols accounts for most of the removal; wet deposition is less significant by a factor of 3 to 5.

PAH compounds tend to be removed from the water column by volatilization to the atmosphere, binding to particulates or sediments or by being accumulated by or sorbed onto aquatic biota. Compounds with Henry's law constants in the range of 10^{-3} to 10^{-5}, are associated with significant volatilization, while compounds with values less than 10^{-5} volatilize from water only to a limited extent (Lyman, et al 1982).

Because of their low solubility, PAHs in aquatic systems are primarily found sorbed to particles that either have settled to the bottom or are suspended in the water column. In an estuary, volatilization and adsorption to suspended sediments with subsequent deposition are the primary removal processes for medium and high molecular weight PAHs, whereas volatilization and microbial degradation are the major removal processes for low molecular weight compounds (Readman, et al 1982). In an enclosed marine ecosystem study, less than 1% of the original amount of radiolabelled benz(a)anthracene added to the system remained in the water column after 30 days; losses were attributed to adsorption to settling particles and to a lesser extent to photodegradation (Hinga and Pilson 1987).

High molecular weight PAHs having K_{oc} values in the range of 10^5 to 10^6 indicate stronger tendencies to adsorb to organic carbon. Sorption of PAHs to soil and sediments increases with increasing organic carbon content and is also directly dependent on particle size. Karickhoff, et al. (1979) reported partition coefficients (Kp) for sorption of pyrene to sediments as follows: sand 9.4 to 68; silt 1,500 to 3,600; and clay 1,400 to 3,800. Gardner, et al. (1979) found that three to four times more anthracene and about twice as much fluoranthene, benz(a)anthracene and benzo(a)pyrene were retained by marsh sediments than by sand. PAHs may also volatilize from soil. Volatilization of acenaphthene, anthracene, fluorene and phenanthrene (low molecular weight PAHs) from soil may be substantial (Coover and Sims 1987). Lower molecular weight compounds may also volatilize from sediments whereas this process is not significant for the higher molecular weight compounds (Southworth 1979).

PAHs have been detected in groundwater either as a result of migration directly from contaminated surface waters or through the soil (Ehrlich, et al 1982; Wilson, et al. 1986). Fluorene from an abandoned creosoting pit migrated through sand and clay into groundwater (Wilson, et al 1986).

PAHs can be accumulated in aquatic organisms from water, sediments and food. In general, bioconcentration was greater for the higher molecular weight compounds than for the lower molecular weight compounds. In fish, biotransformation of the parent compound can occur. Biotransformation by the mixed function oxidase system in fish liver can result in the formation of carcinogenic and mutagenic intermediates; exposure to PAHs has been linked to the development of tumors in fish (Eisler 1987). The ability of fish to metabolize PAHs may

explain why benzo(a)pyrene is frequently not detected or found at only very low levels in fish from environments heavily contaminated with PAHs (Varanasi and Gmur 1980, 1981). Varanasi, et al. (1985) ranked the amount of benzo(a)pyrene metabolized by aquatic organisms as follows: fish > shrimp > amphibod crustaceans > clams. Half lives for elimination of PAHs in fish ranged from less than 2 days to 9 days (Niimi 1987).

Sediment-associated PAHs can be accumulated by bottom-dwelling invertebrates and fish (Eisler 1987). Varanasi, et al. (1985) found that benzo(a)pyrene was accumulated in fish, amphibod crustaceans, shrimp, and clams when estuarine sediment was the source of the compound. Approximate tissue to sediment ratios were 0.6-1.2 for amphibods, 0.1 for clams and 0.05 for fish and shrimp.

PAHs can accumulate in terrestrial plants. Some terrestrial plants can take up PAHs via the roots or foliate; uptake rates are dependent on the concentration, solubility and molecular weight of the PAH and on the plant species (Edwards 1983). Ratios of PAH concentrations in vegetation to those in soil ranged from 0.001 to 0.18 for total PAHs and from 0.002 to 0.33 for benzo(a)pyrene. About 30%-70% of atmospheric PAHs (indeno(1,2,3-cd)pyrene, fluoranthene, and benzo(a)pyrene) deposited on a forest were sorbed onto tree foliage (i.e., leaves and needles) and then deposited as litterfall (Matzner 1984).

PAHs may accumulate in terrestrial animals through the food chain or by ingestion of soil (Gile, et al, 1982).

PAHs can undergo photooxidation and can react in the atmosphere with pollutants such as ozone, nitrogen oxides, sulfur dioxide and peroxyacetylnitrate (NRC 1983). Atmospheric half lives are generally less than 30 days. Some PAHs are degraded by oxidation reactions that have been measured in the dark (to eliminate the possibility of photodegradation). Korfmacher, et al. (1980) found that, while fluorene was completely oxidized, fluoranthene and phenanthrene were not oxidized and benzo(a)pyrene and anthracene underwent minimal oxidation. These compounds were tested adsorbed to coal fly ash; the investigators stated that the form of the compound (adsorbed or pure) and the nature of the adsorbent greatly affect the rate and extent of the process.

The most important processes contributing to the degradation of PAHs in water are photooxidation, chemical oxidation, and biodegradation by aquatic microorganisms (Neff 1979). Hydrolysis is not considered to be an important degradation process for PAHs (Radding, et al 1976). In natural aquatic systems, photooxidation and biodegradation can significantly contribute to the degradation of PAHs, depending on environmental conditions.

In general, PAHs can be significantly metabolized by microbes in water under oxygenated conditions. However, under anoxic conditions, degradation will be extremely slow (Neff 1979). Some PAHs are partially or completely degraded by some species of aquatic bacteria and fungi.

In soil, microbial metabolism is the major process for degradation of PAHs in soil environments. Photolysis, hydrolysis and oxidation are not considered important processes for the degradation of PAHs in soil (Sims and Overcash 1983). The rate and extent of degradation of PAHs in soil are affected by environmental factors, characteristics of the microbial population and the physical and chemical properties of the PAHs. Environmental factors that may influence the rate of PAH degradation in soil include temperature, pH, oxygen concentration, PAH concentrations and contamination history of soil, soil type, mixture, nutrients and other substances that may act as substrate co-metabolites (Sims and Overcash 1983).

Anthracene and fluoranthene showed slightly higher biodegradation rates than benz(a)anthracene or benzo(a)pyrene in a study with fine and medium sands and marsh sediments (Gardner, et al 1979). Degradation rates expressed as percentage of the mass removed per week for the four compounds were anthracene 2.0%-3.0%, fluoranthene 1.9%-2.4%, benz(a)anthracene 1.4%-1.8% and benzo(a)pyrene 0.84%-1.4%. The rate of biodegradation may be altered by the degree of contamination (Ya Khesina, et al 1969).

Absorption, Transport and Degradation:

No studies were located regarding the absorption of PAHs in humans following inhalation exposure. However, absorption of PAHs following inhalation may be inferred from the presence of urinary metabolites of PAHs in workers exposed to these compounds in an aluminum plant (Becher and Bjorseth 1983). The high concentration of PAHs in the occupational setting did not correspond to the amount of PAHs deposited, metabolized and excreted in the urine in this study. Researchers suggested that PAHs adsorbed to airborne particulate matter may not be bioavailable and that the dose-uptake relationship may not be linear over the entire PAH concentration range.

In animal studies following inhalation exposure, Sun, et al (1982) administered radioactive benzo(a)pyrene (0.6 μg/l) adsorbed on Ga_2O_3 particles as an aerosol. After 30 minutes of exposure, the fraction of the total amount of aerosol particles deposited in the lung was approximately 20% for Ga_2O_3 and approximately 10% for the pure hydrocarbon aerosol. After two weeks, complete absorption of the initially instilled hydrocarbon had occurred. The association of benzo(a)pyrene with the particles increased the absorption of the compound.

The size of the particles on which benzo(a)pyrene is absorbed affects the pulmonary absorption of the chemical (Cresia, et al 1976).

Intratracheal administration of radioactive benzo(a)pyrene to rats resulted in its rapid absorption. Radioactivity in the liver reached a maximum of 21% of the dose within 10 minutes of instillation (Weyand and Bevan 1986, 1988). Similar results were also seen in guinea pigs and hamsters following intratracheal exposure (Weyand and Bevan 1986, 1987b, 1988). In monkeys and dogs, there was either little or very slow direct transfer of

benzo(a)pyrene or its metabolites into the blood (Petridou-Fischer, et al 1988). Monkeys and dogs received nasal instillation of radioactive benzo(a)pyrene at doses of 0.16-0.21 mg/kg. Radiolabeled metabolites were detected in the nasal cavity, but little or no activity was detected in the blood and excreta of either species during the 48 hrs after exposure.

Via the oral route, indirect evidence suggests that benzo(a)pyrene may not be readily absorbed following exposure in humans. In human volunteers who ingested broiled meat that contained approximately 9 μg of benzo(a)pyrene, Hecht, et al 1979, detected less than 0.1 μg/person (ie., below the detection limit) of benzo(a)pyrene in the feces of these individuals.

Via the dermal route of exposure, application of 2% crude coal tar to the skin of humans for 8-hour periods on 2 consecutive days yielded evidence of PAH absorption (Storer, et al 1984). Phenanthrene, anthracene, pyrene and fluoranthene were detected in the blood, but benzo(a)pyrene was not detected; thus absorption of

PAHs in crude coal tar was variable. This variability was attributed to differences in percutaneous absorption, rapid tissue deposition after absorption or metabolic conjugation with rapid urinary excretion. An in vitro study using human skin revealed that the extent of permeation after 24 hours was established as 3% of the applied dose of radioactive benzo(a)pyrene (10 $\mu g/cm^2$) (Kao, et al 1985).

In conclusion, via the dermal route, PAHs are absorbed through the skin of humans. Therefore, skin contact with soil and water contaminated with PAHs at hazardous waste sites can result in exposure to these compounds. Systemic absorption of PAHs from the skin is variable among these compounds.

No studies were located regarding the distribution of PAHs in humans following oral, inhalation and dermal routes of exposure. However, rat studies indicate that benzo(a)pyrene is distributed to the lung, liver, kidney, gastrointestinal tract and carcass after inhalation exposure (Weyand and Bevan 1986, 1987a, 1988) and to the liver, lung and kidneys after oral exposure (Yamazaki, et al 1987). In animal studies via the dermal route, PAHs can readily penetrate the skin but very little is distributed to tissues. Only 1.3% of the applied dose of radioactive anthracene (9.3 $\mu g/cm^2$) was detected in tissues of rats at six days after administration (Yang, et al 1986).

The metabolism of PAHs alters these chemicals both chemically and structurally, rendering them more water-soluble and more excretable. Benzo(a)pyrene is metabolized to several arene oxides. Once formed, these arene oxides may rearrange spontaneously to phenols, undergo hydration to the corresponding trans-dihydrodiols or react covalently with glutathione. Phenols may also be formed by direct oxygen insertion, although unequivocal proof for this mechanism is lacking. 6-Hydroxybenzo(a)pyrene is further oxidized either spontaneously or metabolically to the 1,6-, 3,6-, or 6,12-quinones. Evidence exists for the further oxidative metabolism to two additional phenols. The phenols, quinones and

dihydrodiols can all be conjugated to glucuronides and sulfate esters; the quinones also form glutathione conjugates (IARC 1983).

II. Non Carcinogenic Health Effects

Acute Toxicity:

Humans: No studies were located regarding immunological effects in humans following exposure to the PAHs covered in this profile. Benzo(a)pyrene is immunogenic when applied dermally to the skin of animals. In mice, acute application of benzo(a)pyrene elicited an allergic contact hypersensitivity in mice (Klemme, et al 1987). Contact hypersensitivity was also observed in guinea pigs following two dermal applications of benzo(a)pyrene given over a period of 2-3 weeks. (Old, et al 1963).

Laboratory animals: Mice acutely administered benz(a)anthracene by oral gavage for 2 days exhibited increased incidences of hepatomas and pulmonary adenomas (Klein 1963). No malignant tumors were observed in this study. Other acute-duration studies reporting oral exposure to benzo(a)pyrene and increased incidence of benign and malignant tumors of the forestomach in animals include Hartwell 1951, Shubik and Hartwell 1957, Thompson 1971, Tracor/Jitco 1973a and b. After acute oral exposure to benzo(a)pyrene, Wattenberg and Bueding 1986 reported forestomach papillomas in animals. Neal and Rigdon 1967 reported that mice fed benzo(a)pyrene for 2 or more days exhibited gastric neoplasms. Chu and Malmgrem 1965 reported papillomas and carcinomas of the alimentary tract in hamsters fed benzo(a)pyrene. Mammary tumors were observed in rats administered a single oral dose of benzo(a)pyrene (McCormick, 1981).

Chronic Toxicity:

Humans: No chronic studies of PAHs were reported in the available literature.

Laboratory animals: No chronic studies of PAHs were reported in the available literature.

III. Carcinogenic Health Effects:

Acute Exposure

Humans: No studies were located regarding cancer in humans following inhalation exposure to any of the 15 PAHs discussed in this profile. However, epidemiologic studies have shown increased mortality due to lung cancer in humans exposed to coke-oven emissions (Lloyd 1971; Mazumdar, et al 1975; Redmond, et al 1976), roofing-tar emissions (Hammond, et al 1976), and cigarette smoke (Maclure and MacMahon 1980; Wynder and Hoffmann 1967). Each of these mixtures contains benzo(a)pyrene, chrysene, benz(a)anthracene, benzo(b)fluoranthene, dibenz(a,h)anthracene as well as other potentially carcinogenic PAHs and other carcinogenic

and potentially carcinogenic nitrosamines, coal tar pitch and creosote. It is thus impossible to evaluate the contribution of any individual PAH to the total carcinogenicity of these mixtures in humans because of the complexity of the mixtures and the presence of other carcinogens.

No studies were located that gave evidence of a direct association between human dermal exposure to individual PAHs and cancer induction. However, reports of skin tumors among individuals exposed to mixtures containing PAHs lend some qualitative support to their potential for carcinogenicity in humans. Pott (1775) reported scrotal cancer among chimney sweeps. Skin cancer among those dermally exposed to shale oils has been reported (Purde and Etlin 1980). These reports provide only qualitative suggestions pertaining to the human carcinogenic potential of compounds found in chimneys and shale oils, such as benzo(a)pyrene, chrysene, dibenz (a,h) anthracene, benz(a)anthracene or benz(b)fluoranthene.

Laboratory animals: Studies in laboratory animals have demonstrated the ability of benz(a)anthracene, benzo(b)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene to induce skin tumors following dermal exposure. In a long-term chronic bioassay using mice receiving benzo(b)fluoranthene throughout their lifetime, malignant tumors appeared as early as 4 mos in the high dose group. The lowest dose at which benzo(b)fluoranthene elicited malignant tumors was 0.1%, which is approximately equal to a dose of 2.9 mg/kg received 3x weekly or an average daily dose of 1.2 mg/kg (Wynder and Hoffmann 1959b). Mice topically administered 2 μg benzo(a)pyrene throughout their lifetime developed increased skin tumor incidences of papillomas and carcinomas (Habs, et al 1984). Mice administered 12.5 μg benzo(a)pyrene for 99 weeks exhibited malignant skin tumors (Warshawsky and Barkley 1987). Incidences of tumors in these dermal studies was related to the dosage of the compound.

Other studies reporting the carcinogenicity of selected PAHs in laboratory animals include the following: Bingham and Falk 1979, Cook, et al 1933, Cook 1933, Horton and Christian 1974, Wynder and Hoffmann 1959a, Van Duuren, et al 1967, Lijinsky, et al 1965, Ranadine and Karande 1963, Hoffmann and Wynder 1966, Horton and Christian 1974).

Chronic Exposure

Humans: No information is available.

Animals: Chronic inhalation studies of animals given benzo(a)pyrene reported a dose-response relationship between inhaled benzo(a)pyrene particles and respiratory tract tumorigenesis. Tumors were formed in the nasal cavity, pharynx, larynx, trachea, esophagus and forestomach of hamsters (Thyssen, et al 1981). Other studies reporting the association between inhalation exposure to benzo(a)pyrene and other atmospheric irritants and tumors in animals are Heinrich, et al 1986 and Laskin, et al 1970.

IV. Mutagenicity and Teratogenicity

Mutagenicity: No studies were located regarding genotoxic effects in humans following exposure to PAHs. Oral exposure to benzo(a)pyrene produced gene mutations in mice in the mouse coal color spot test (Davidson and Dawson 1976 and 1977). Gene mutations were produced in bacteria that were injected intraperitoneally into mice after they were exposed to benz(a)anthracene (Simmon, et al 1979).

Teratogenicity (Developmental Toxicity)/Reproductive Studies: No studies were located regarding developmental effects in humans following oral exposure to PAHs. Prenatal exposure to benzo(a)pyrene produced reduced viability of litters at parturition, reduced mean pup weight during postnatal development and a high incidence of sterility in the progeny of mice with associated alterations in gonadal morphology and germ-cell development (Mackenzie and Angevine 1981). In mice, increased incidence of stillborns, resorptions and malformations were observed in offspring of animals exposed to benzo(a)pyrene (Legraverend, et al 1984).

No studies were located regarding reproductive effects in humans following oral exposure to PAH compounds. Mackenzie and Angevine 1981 reported that benzo(a)pyrene administered by gavage to pregnant mice decreased the percentage of pregnant females at parturition and produced a high incidence of sterility in the progeny. Rigdon and Rennels 1964 found that benzo(a)pyrene administered in the diet reduced the incidence of pregnancy in female rats.

V. IARC Carcinogenicity Classification

The IARC (1983, 1984) has classified PAHs as carcinogenic generally according to the weight-of-evidence categories they developed, as shown in Table 1. Carcinogenic PAHs were defined as those for which IARC has stated there is either "sufficient" or "limited" evidence of carcinogenicity. In addition, several PAHs for which IARC has stated that there is "inadequate" evidence of carcinogenicity are also classified as carcinogenic on the basis of their structural similarity to known carcinogenic PAHs. Finally, PAHs were classified as carcinogenic if they were reported in the analytic data to be simple methyl derivatives of known or suspected carcinogenic PAHs where the position of methylation was unspecified, e.g., "methylbenzo(a)pyrene."

All PAHs for which IARC has found "inadequate" data to assess carcinogenicity and do not have a structural similarity to a suspected carcinogen were classified as noncarcinogenic. Those for which the available data do not provide any evidence of carcinogenicity were classified as noncarcinogenic.

Table 1

CARCINOGENICITY OF PAHs

Chemicals for which there is sufficient evidence that they are carcinogenic in animals:

Benzo(a)anthracene
Benzo(b)fluoranthene
Benzo(j)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Dibenzo(a,h)acridine
Dibenzo(a,j)acridine
Dibenzo(a,h)anthracene
7H-Dibenzo(c,g)carbazole
Dibenzo(a,e)pyrene
Dibenzo(a,h)pyrene
Dibenzo(a,i)pyrene
Dibenzo(a,l)pyrene
Indeno(1,2,3-c,d)pyrene
5-Methylchrysene

Chemicals for which there is limited evidence that they are carcinogenic in animals:

Anthanthrene
Benzo(c)acridine
Carbazole
Chrysene
2- and 3-methylfluoranthene
Dibenzo(a,c)anthracene
Dibenzo(a,j)anthracene
Dibenzo(a,e)fluoranthene
2-,3-,4-,and 6-methylchrysene
Cyclopenta(c,d)pyrene

Chemicals for which the evidence is inadequate to assess their carcinogenicity:

Benzo(a)acridine
Benzo(g,h,i)fluoranthene
Benzo(a)fluorene
Benzo(b)fluorene
Benzo(c)fluorene
Benzo(g,h,i)perylene
Benzo(c)phenanthrene
Benzo(e)pyrene
Coronene
1,4-Dimethylphenanthrene
Fluorene
1-Methylchrysene
1-Methylphenanthrene
Perylene
Phenanthrene
Triphenylene

Chemicals for which the available data provide no evidence that they are carcinogenic:

Anthracene
Fluoranthene
Pyrene

Source: IARC 1983, 1984.

VI. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

No carcinogenic classification was reported in the available literature.

EPA Dose Response Parameters:

Carcinogenic Effects: The EPA derived an oral cancer potency factor of 11.5(mg/kg/day)$^{-1}$ based on the study of Neal and Rigdon (1967) (EPA, 1991a). In this study, benzo(a)pyrene administered orally to mice for 2 or more days induced gastric tumors. An inhalation cancer potency factor of 6.1 (mg/kg/day)$^{-1}$ was derived by the EPA based on the study of Thyssen, et al 1990 (EPA, 1991a). In this study, chronic inhalation exposure to benzo(a)pyrene induced respiratory tract tumors in hamsters.

Noncarcinogenic Effects: A dose-response estimate is not available for the noncarcinogenic effects of benzo(a)pyrene (EPA 1991a).

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B.7 BERYLLIUM

I. Use and Occurrence:

The primary source of beryllium is the open-pit mining of bertrandite ore, which is processed into beryllium hydroxide, and further processed into beryllium metal, alloys and oxide. Beryllium alloys and metal have a wide variety of applications in electrical components, tools, structural components for aircraft, missiles, and satellites, and other metal-fabricating uses (ATSDR, 1988). Natural levels of beryllium in soil range from approximately 0.6 mg/kg to 3.5 mg/kg (Kabata-Pendias and Pendias, 1984).

II. Physical and Chemical Properties of Beryllium

Molecular Weight	9.0[1]
Water Solubility, mg/l	0.0E+00[2]
Vapor Pressure, mm Hg	0.0E+00[3]
Bioaccumulation Factor for Fish	1.9E+01[4]
Bioaccumulation Factor for Shellfish	1.0E+01[5]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Weast, 1979
[3]USEPA, 1981
[4]USEPA, 1980
[5]Napier et al., 1980

III. Fate and Transport:

Although beryllium is a naturally occurring substance, the major source of its emission to the environment is the combustion of coal and fuel oil, which releases particulates and fly ash containing beryllium into the atmosphere. Beryllium released to the atmosphere from coal combustion is likely to be in the form of beryllium oxide. Atmospheric beryllium particulates will eventually settle to the earth's surface by dry deposition or may be removed from the atmosphere by wet deposition (i.e., rainfall). Upon reaching soil and sediment, beryllium will probably be retained in an insoluble form and be generally immobile (ATSDR, 1988).

IV. Routes of Exposure, Absorption, Distribution, Transport, and Degradation:

The principle routes of human exposure are inhalation, and ingestion of Be salts. Transport of this metal through human tissue is accomplished via the bloodstream. Occupational

exposure to beryllium results in high levels in the lungs and bone and lower levels in the liver and kidney (Tepper et al., 1961; Meehan and Smyth, 1967). Respiratory toxicity is the main effect of inhalation exposure to beryllium.

V. Acute Toxicity:

Acute inhalation exposure (at levels of 28 μg/kg lung tissue dry weight) has been found to result in acute chemical pneumonitis (EPA, 1987, Health Effects Document for Beryllium, EPA/600/8-84/026F). Dermal exposure to soluble beryllium compounds can cause contact dermatitis (EPA, 1980 as cited in EPA, 1987). Skin contact may also result in a delayed allergic reaction, which is characterized by large skin lesions that may not easily heal.

VI. Chronic Toxicity:

Chronic beryllium pneumonitis (berylliosis) is characterized by the development of granulomatous lesions of the lung. Inhalation of beryllium causes inflammation of the entire respiratory tract and berylliosis (chronic lung disease), which may occur several months or even years after exposure (EPA, 1987 same as above).

VII. Mutagenicity, Carcinogenicity and Teratogenicity:

Mutagenicity:

The mutagenicity of various beryllium compounds is not clear; a positive or negative response depends on the type of bacterial strain and the type of assay system that is used. Beryllium sulfate was generally negative in Ames assays, but induced chromosome aberrations and sister chromatid exchanges in mammalian systems (ATSDR, 1988).

Carcinogenicity:

Beryllium is carcinogenic via inhalation and intratracheal routes in experimental animals resulting primarily in lung and/or bone tumors (EPA, 1986 as cited in EPA, 1987). Several epidemiological studies have suggested that occupational exposure to beryllium may result in an increased risk of lung cancer although the data are inconclusive (EPA, 1987).

Human epidemiological studies indicate a possible relationship between inhalation of beryllium and the incidence of lung cancer in exposed workers. Animal studies have demonstrated the induction of tumors by a variety of beryllium compounds. An increase in lung cancer was observed in rats following both chronic oral and inhaled dosages of Be, with inhalation being the more dangerous route of exposure (i.e. producing a higher incidence of cancer at lower concentrations). Bone cancer has been induced in rabbits and mice following chronic intravenous injection of various Be salts (IRIS, 1994).

Teratogenicity:

No data available on the teratogenicity of this chemical.

VIII. EPA Carcinogenic Classification and Dose-Response

Parameters:

EPA Carcinogenic Classification:

EPA (1994) has classified beryllium as a group B2 Agent - Probable Human Carcinogen based on sufficient evidence of carcinogenicity in animals but inadequate evidence of carcinogenicity in humans.

Dose-Response Parameters (IRIS, 1994):

Carcinogenic effects:

CANCER SLOPE FACTOR (ORAL) : 4.3 per(mg/kg)/day
DISCUSSION OF CONFIDENCE :
The estimate is derived from a study which did not show a significant increase in tumorigenic response. While this study is limited by use of only one non-zero dose group, the occurrence of high mortality and unspecified type and site of the tumors, it was used as the basis of the quantitative estimate because exposure occurred via the most relevant route. Oral risk estimates derived by extrapolation from studies in other species/strains for the intravenous and inhalation routes (also highly uncertain) are within an order of magnitude.

INHALATION UNIT RISK : 2.4E-3 per (μg/cu.m)
DISCUSSION OF CONFIDENCE :

The estimate of risk for inhalation exposure was based upon an epidemiologic study having several confounding variables. The estimates of exposure levels and duration were also somewhat uncertain. While a quantitative assessment based on several animal studies resulted in a similar estimate of risk (which increases the confidence somewhat), the quality of the available studies was poor (that is, they were conducted at single dose levels or lacked control groups).

Noncarcinogenic effects:

ORAL RFD: 5E-3 mg/kg/day
CRITICAL EFFECT/TARGET ORGAN: No adverse effects

ORAL RFD UNCERTAINTY:

UF = 100. The uncertainty factor of 100 reflects a factor of 10 each for interspecies conversion and for the protection of sensitive human subpopulations.

ORAL RFD CONFIDENCE:
Study: Low
Data Base: Low
RfD: Low

Confidence in the study is rated as low because only one dose level was administered. Although numerous inhalation investigations and a supporting chronic oral bioassay in mice exist, along with the work by Cox et al. (1975) which indicates that a higher dose level might be a NOEL, these studies are considered as low to medium quality; thus, the data base is given a low confidence rating. The overall confidence in the RfD is low, reflecting the need for more toxicity data by the oral route.

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B.8 BIS(2-ETHYLHEXYL)PHTHALATE

I. General Background

Occurrence and Use

Bis(2-ethylhexyl)phthalate (BEHP) also known as diethylhexyl phthalate (DEHP). This compound is a plasticizer for plastics and polymers used in every major product category including construction, automotive, household products, apparel, toys, packaging and medical products. Thus, it is an almost ubiquitous environmental contaminant. It is also a frequent laboratory contaminant of environmental samples.

Physical and Chemical Properties of Bis(2-ethylhexyl)phthalate

Molecular Weight	391.00
Water Solubility, mg/l	4.0E-01
Vapor Pressure, mm Hg	2.0E-07
Henry's Law Constant, atm-m^3/mole	4.4E-07
Octanol-Water Partition Coefficient	4.1E+09
Bioaccumulation Factor for Fish	1.6E+02
Bioaccumulation Factor for Shellfish	1.6E+07

Source: Multi-Media Exposure Assessment Manual, 1989

Environmental Fate and Transport

Bis-(2-ethylhexyl) phthalate (DEHP) is used as a plasticizer for polyvinylchloride (PVC) and other polymers in large quantities and is likely to be released to air and water during production and waste disposal of these plastic products. DEHP in water will biodegrade (half-life 2-3 wk), adsorb to sediments and bioconcentrate in aquatic organisms. Atmospheric DEHP will be carried long distances and be removed by rain. The compound will strongly bind to soil and sediments, and will neither evaporate or leach into groundwater. DEHP released to water systems will biodegrade fairly rapidly (half-life 2-3 weeks) following a period of acclimation. Evaporation and hydrolysis are not significant aquatic processes. DEHP will also bioaccumulate in aquatic organisms (HSDB, 1992).

Routes of Exposure, Absorption, Distribution, Transport, and Degradation

Human exposure will occur in occupational settings and from air, from consumption of drinking water, food (especially fish etc, where bioconcentration can occur) and food wrapped in PVC, as well as during blood transfusions from PVC blood bags (HSDB, 1992). BEHP is readily absorbed following oral or inhalation exposure (EPA, 1980). Dermal absorption has not been studied to any great extent, but based on its high lipophilicity, it is expected that DEHP will also be absorbed efficiently through the skin.

II. Non-Carcinogenic Health Effects

Acute Exposure

Humans: No information is provided in the available literature.

Laboratory animals: No information is provided in the available literature.

Chronic Exposure

Humans: No information is provided in the available literature.

Laboratory animals: Chronic exposure to relatively high concentrations of BEHP in the diet (i.e., 0.5% of total diet) can cause retardation of growth and increased liver and kidney weights in laboratory animals (Yanagita et al., Biochemical Pharmacology 27 (19), 1978, p. 2283). Reduced fetal weight and an increased number of resorptions have been observed in rats exposed orally to BEHP (EPA, 1980).

III. Carcinogenic Health Effects

Acute Exposure

Humans: Cancer induction in humans has not been documented through epidemiologic studies.

Laboratory animals: BEHP is reported to be carcinogenic in rats and mice, causing increased incidences of hepatocellular carcinomas or neoplastic nodules following oral administration (NTP, 1982).

Chronic Exposure

Humans: No information is available.

Laboratory animals: No information is available.

IV. Mutagenicity and Teratogenicity

Mutagenicity:

DEHP shows mutagenic activity to Salmonella typhimurium TA-100 with and without S-9 mixture (HSDB, 1992).

Teratogenicity:

Embryolethal and teratogenic effects in Sprague-Dawley rats have been demonstrated, with IP injections of 5 or 10 g/kg body weight on days 5, 10 and day 15 of gestation (HSDB, 1992).

V. EPA Carcinogenic Classification and EPA Dose-Response Parameters (IRIS, 1992):

EPA Carcinogenic Classification:

BEHP is classified as a probable human carcinogen (Class B2).

EPA Dose-Response Parameters:

Carcinogenic Effects:

CANCER SLOPE FACTOR (ORAL) : 1.4E-2/mg/kg/day
DISCUSSION OF CONFIDENCE :

An adequate number of animals was observed and a statistically significant increase in incidence of liver tumors was seen in both sexes and were dose dependent in both sexes of mice and female rats. A potential source of variability in the NTP study is the possibility of feed scattering. The above calculations are based on standard food consumption rates for mice (13% of body weight) and rats (5% of body weight).

No inhalation unit risk dose-parameter has been reported.

Noncarcinogenic effects:

ORAL RFD: 2E-2 mg/kg/day
CRITICAL EFFECT/TARGET ORGAN: Increased relative liver weight
ORAL RFD UNCERTAINTY :

UF = 1000. Factors of 10 each were used for interspecie /ariation and for protection of sensitive human subpopulations. An additional factor of 10 was used since the guinea pig exposure was longer than subchronic but less than lifetime, and because, while the RfD is set on a LOAEL, the effect observed was considered to be minimally adverse.

ORAL RFD CONFIDENCE :
Study: Medium
Data Base: Medium
RfD: Medium

The study by Carpenter et al. (1953) utilized sufficient numbers of guinea pigs and measured multiple endpoints. The fact that there were only two concentrations of DEHP tested precludes a rating higher than medium. Since there are corroborating chronic animal bioassays, the data base is likewise rated medium. Medium confidence in the RfD follows.

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B.9 CADMIUM

I. Occurrence and Use

Cadmium (Cd) is a noncorrosive metal used in a wide variety of industrial processes, such as electroplating and galvanizing, and is a byproduct of zinc and lead mining. It is also used as a color pigment for paints and plastics, and cathode material for nickel-cadmium batteries. The combustion of fossil fuels and tobacco also produce anthropogenic releases of cadmium into the environment (HSDB, 1993).

Background Cd levels in soils should not exceed 0.5 ppm, and all higher values reflect anthropogenic impact (Kabata-Pendias, and Pendias, 1984).

II. Physical and Chemical Properties of Cadmium

Molecular Weight	112.00[1]
Water Solubility, mg/l	0.0E+00[2]
Vapor Pressure, mm Hg	0.0E+00[3]
Bioaccumulation Factor for Fish	2.0E+02[4]
Bioaccumulation Factor for Shellfish	2.0E+03[4]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Weast, 1979
[3]USEPA, 1981
[4]Napier, 1980

III. Environmental Fate and Transport

In a U.S. air sampling study, most ambient air levels of cadmium were <10 ng/m^3, which is very near the detection limit (Carson et al., 1987). Cadmium can enter surface water due to a variety of manufacturing operations, such as electroplating and discarding of spent electroplating solutions (HSDB, 1993). Several studies have concluded that adsorption, rather than precipitation, control CD concentrations in soil solutions until a threshold pH value (i.e., pH 7.5) is exceeded. Cd is most mobile in acidic soils within the range of pH 4.5 to 5.5, whereas in alkaline soils, Cd is rather immobile (Kabata-Pendias and Pendias, 1984).

IV. Routes of Exposure, Distribution, Absorption, Transport, and Degradation

Cd is an airborne workplace contaminant, but exposure is of greater concern to the general population. It is found in food stuffs such as grains, meat, fish and fruit, in contaminated air, water, and soil, as well as in cigarette smoke. Humans are exposed to cadmium via inhalation and ingestion, at which time the metal can be transported through the bloodstream to vital organs (Clayton and Clayton, 1981). In the blood, a small molecular weight plasma protein known as metallothionien binds cadmium. The elimination half-life of cadmium is long (16-33 yrs), but decreases under conditions of acute exposures.

Gastrointestinal absorption of cadmium in humans ranges from 5-6% (USEPA, 1985a) Pulmonary absorption of cadmium in humans is reported to range from 10% to 50% (CDHS, 1986). Cadmium bioaccumulates in humans, particularly in the kidney and liver (USEPA, 1985a,b).

V. Acute Toxicity

Acute toxic effects associated with cadmium compounds can occur in humans under unusually intense exposure scenarios, such as intentional or accidental poisoning. Symptoms of acute non-fatal toxicity resulting from consumption of drinks contaminated with an estimated 16 mg/l of cadmium included nausea, vomiting, and abdominal pain. Inhalation of cadmium fumes may result in acute chemical pneumonitis and pulmonary edema (ATSDR, 1989).

VI. Chronic Toxicity

Chronic oral or inhalation exposure of humans to cadmium has been associated with renal dysfunction, itai-itai disease (bone damage), hypertension, anemia, endocrine alternations, immunosuppression, and irreversible lung damage in the form of chronic bronchitis and emphysema (Clayton and Clayton, 1981).

Progressive accumulation of Cd in soft tissues, particularly the kidney, poses a serious human health risk. A higher incidence of kidney damage reported for certain regions of Japan has been linked to a high intake of dietary cadmium. Renal toxicity occurs in humans at a renal cortex concentration of cadmium of 200 about μg/g (USEPA, 1985b).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity:

Numerous assays of cadmium's genotoxic potential have been conducted, yielding mixed results. Bacterial gene mutation assays have been conducted using Salmonella strains, and three of four were reported to be negative, although some positive or weak positive responses were indicated in one study. Tests for chromosomal aberrations involving human and other mammalian cells have also yielded mixed results. In vitro tests of human blood lymphocytes were positive in one case, weakly positive in a second case, and negative in two cases.

Human W138 and MCR5 cells also yielded negative results. Results were positive using chinese hamster cells, but negative using mouse mammary carcinoma cells (ATSDR, 1989).

Carcinogenicity:

Epidemiological studies have demonstrated a strong association between inhalation exposure to cadmium and cancers of the lungs, kidneys, and prostate (USEPA, 1985b). In experimental animals, cadmium induces injection-site sarcomas and testicular tumors. When administrated by inhalation, cadmium chloride is a potent pulmonary carcinogen in rats. Cadmium is a well-documented animal teratogen (USEPA, 1985b). Several animal studies support this data. Chronic inhalation exposure of rats to cadmium produce lung tumors in Wistar rats, and tumors at various sites (including mammary tumors in females) in Fischer rats (IRIS, 1994).

Teratogenicity:

Teratogenic effects of cadmium administered at high doses in bioassays is abundantly documented, but little evidence directly addresses the question of whether lower, environmentally realistic doses might exert such effects (ATSDR, 1989).

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

Cadmium is classified as a probable human carcinogen (Class B1). This classification applies to agents for which there is limited evidence of carcinogenicity in humans from epidemiologic studies but significant evidence in animals.

This designation is based on a higher incidence of lung cancer in cadmium smelter workers, and increased incidence of prostrate cancer in battery workers.

EPA Dose-Response Parameters (IRIS, 1994):

Carcinogenic effects:

Limited evidence from occupational epidemiologic studies of cadmium exposure is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection. Seven studies in rats and mice where in cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.

There is no oral Cancer Slope Factor for cadmium.

INHALATION UNIT RISK: 1.8E-3 per (ug/cu.m)

Noncarcinogenic Effects:

ORAL RfD and SUMMARY:
5E-4 mg/kg/day (water)
1E-3 mg/kg/day (food)

CRITICAL EFFECT/TARGET ORGAN: significant proteinuria in human subjects

ORAL RFD UNCERTAINTY :
UF = 10. This uncertainty factor is used to account for intrahuman variability to the toxicity of this chemical in the absence of specific data on sensitive individuals.

ORAL RFD MODIFYING FACTOR:
MF = 1.

ORAL RFD CONFIDENCE:
Study: Not applicable
Data Base: High
RfD: High
INHALATION RFD SUMMARY:

A risk assessment for this substance/agent is under review by an EPA work group.

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B.10 CHLOROFORM

I. Background

Chloroform ($CHCl_3$) is a volatile and relatively soluble liquid (in water and fat) that is used principally as an industrial solvent and as a chemical intermediate (Fawell and Hunt, 1988). Chloroform at trace levels is ubiquitous in the environment with average urban ambient atmospheric concentrations less than 1000 ppt (parts per trillion) and rural air concentrations less than 10 ppt (U.S. Environmental Protection Agency, 1985). A drinking water survey in the United States indicated a range of concentrations from 0.1 to 311 mg/liter with a median concentration of 21 mg/liter (WHO, 1984). Inhalation is believed to be the primary route of exposure under normal circumstances, followed by ingestion of chloroform contaminated drinking water (U.S. Environmental Protection Agency, 1985). Respiratory absorption efficiency ranges from 49 to 77 percent (U.S. Environmental Protection Agency, 1980), which is influenced by body weight, fat content, and solubility of chloroform in blood (U.S. Environmental Protection Agency, 1985). Absorption of chloroform from the gastrointestinal tract in humans is complete (Fry and others, 1972). Once absorbed, chloroform is readily distributed to all tissues, especially those with a high lipid content. Tissues with the highest chloroform concentrations (in descending order) are adipose tissue, brain, liver, kidney, and blood (McConnell and others, 1975).

II. Acute Toxicity

In humans and experimental animals, the principal effects of acute chloroform exposure are central nervous system (CNS) depression, hepatotoxicity (liver toxicity), and to a lesser extent, kidney and cardiac effects (U.S. Environmental Protection Agency, 1985). Chloroform is of low acute toxicity with an oral LD_{50} in male and female rats of 908 mg/kg and 1117 mg/kg, respectively (Chu and others, 1980).

The depressive effect of chloroform on the CNS is focused on the cortex, higher brain centers, medulla and spinal cord (Wood-Smith and Stewart, 1964). Brain centers responsible for respiration, vomiting, temperature regulation, vasomotor and vagal activity are all depressed (Adriani, 1970; U.S. Environmental Protection Agency, 1985). Hepatotoxicity is characterized by rapid glycogen depletion, fatty degeneration and centrilobular necrosis (tissue destruction) with corresponding symptoms of progressive weakness, prolonged vomiting, coma and even death, usually by the fourth or fifth day in severe cases (U.S. Environmental Protection Agency, 1985). Clinical evidence of altered liver function include increased serum bilirubin, bile in urine, increased nitrogen excretion and reduced creatine clearance (U.S. Environmental Protection Agency, 1985).

Other organs adversely affected by acute exposures to chloroform include the respiratory tract (increased respiratory rate), cardiovascular system (arrhythmias and reduced blood pressure), urinary tract (decreased urine flow), and hematologic effects (increased number of red blood

cells, leukocytes, and polymorphonuclear cells) (U.S. Environmental Protection Agency, 1985). The systemic effects of chloroform are independent of route of exposure.

III. Chronic Toxicity

Most of the data on the chronic toxicity of chloroform has been obtained from carcinogenicity studies. In humans the most common toxic effects of chronic chloroform exposure is on the liver and CNS. Liver effects have been observed for experimental animals and occupationally exposed human populations. Symptoms observed in workers exposed to chloroform (77 to 237 ppm) include thirst, irritability, lassitude, mental sluggishness, and loss of appetite (NIOSH, 1974; Challen and others, 1958). Also, no liver damage was observed. Several researchers observed jaundice, enlarged livers, and increased incidence of viral hepatitis in occupationally exposed workers (Bomski and others, 1967; Phoon and others, 1983). Daily consumption of chloroform by humans in toothpaste and mouthwash was studied over a five-year period (WHO, 1984). No liver toxicity was observed at daily consumption rates estimated at 0.34 - 0.96 mg/kg (WHO, 1984). Chronic animal exposure studies have also indicated kidney damage and CNS depression in chloroform treated animals (ATSDR, 1987).

Carcinogenicity

There are no epidemiological studies on the carcinogenicity of chloroform. There are several epidemiological studies of the effects of chlorinated drinking water studies which indicate a small significant increase in the incidence of bladder or colon cancer (U.S. Environmental Protection Agency, 1990). These studies are not conclusive since many other carcinogenic compounds were also present.

There is sufficient evidence of animal carcinogenicity of chloroform, specifically in the kidneys and livers of exposed rats and mice (ATSDR, 1987). In a drinking water study designed to examine the carcinogenicity of chloroform at low doses, rats and mice received up to 160 mg/kg/day and 263 mg/kg/day, respectively for 104 weeks (Jorgenson and others, 1985). A significant and dose-related increase in the incidence of renal (kidney) tumors was observed (U.S. Environmental Protection Agency, 1990). In another carcinogenic study, rats and mice were fed chloroform at varying doses for 78 weeks (NCI, 1976). Upon examination, a significant increase in kidney epithelial tumors in male rats and hepatocellular (liver) carcinomas in mice was observed (NCI, 1976). The USEPA has reviewed the available data on carcinogenicity and has categorized chloroform as a probable human carcinogen (Group B_2) based on an increased incidence of several tumor types in rats and three strains of mice (U.S. Environmental Protection Agency, 1990).

Mutagenicity

Most mutagenic tests have produced negative in vitro and in vivo results for chloroform. Specifically, negative results have been obtained in tests examining covalent binding to DNA,

Ames Salmonella histidine reversion, DNA damage, and chromosomal aberrations (U.S. Environmental Protection Agency, 1990). In contrast, chloroform caused mitotic recombination in bacteria and sister chromatid exchange in human lymphocytes and mouse bone marrow cells after in vivo exposure (U.S. Environmental Protection Agency, 1990). The carcinogenicity of chloroform may be the result of metabolism to phosgene, which is known to cause DNA cross-links (U.S. Environmental Protection Agency, 1990). As supporting evidence of this theory, urine extracts from chloroform-treated mice were found to be mutagenic (Agustin and Lim-Syliance, 1978).

Reproductive/Developmental

There is very little data on the reproductive effects of chloroform in animals and no human data are available. Inhalation of chloroform (400 to 800 ppm), for 4 hrs/day for 5 days resulted in an increase in the rate of abnormal sperm in exposed mice (Land and others, 1981). This effect was not seen following intraperitoneal injection. Additionally, gonadal atrophy was seen in rats orally treated with chloroform for 13 weeks up to 410 mg/kg/day (Palmer and others, 1979).

No data was available on the developmental effects of chloroform in humans. Chloroform was shown to readily cross the placenta and accumulate in the amniotic fluid (Danielsson and others, 1986). Associated developmental effects of high doses of chloroform in animals include fetal resorptions, decreased fetal weight, increased incidence of cleft palate and incomplete development of skull bones (ATSDR, 1987).

Sensitive Populations

The laboratory studies showed a distinctive strain difference in toxic effects to the kidney (for example, strains that more effectively metabolize chloroform to phosgene are more sensitive) (ATSDR, 1987). Also, a sex related difference in susceptibility to kidney effects was observed where male mice are much more sensitive to chloroform than are females. This differential sensitivity is thought to be linked to higher testosterone levels in males. Human males may also have an increased chloroform sensitivity. Dietary factors (in other words, ethanol exposure and starvation) are known to potentiate the effects of chloroform.

Chemical Interactions

Chloroform toxicity is affected by any factor which alters its metabolism (U.S. Environmental Protection Agency, 1985). Metabolism of chloroform to more reactive compounds is well documented; therefore co-exposure to compounds that enhance metabolism would increase the toxicity of chloroform (ATSDR, 1987).

Many compounds are known to induce liver microsomal enzyme activity and thereby potentially enhance chloroform toxicity, such as ethanol, polybrominated biphenyls,

polychlorinated biphenyls, and ketones such as chlordecone (ATSDR, 1987; U.S. Environmental Protection Agency, 1985). In contrast, disulfiram, diethyldithiocarbamate and carbon disulfide inhibit metabolizing enzymes and protect against chloroform hepatotoxicity (ATSDR, 1987).

Dose-Response Parameter Estimates

The dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by the USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects:

Chloroform is classified as a probable human carcinogen (Group B2) by the USEPA. The Cancer Assessment Group (CAG) of USEPA has computed an oral cancer potency estimate of 6.1×10^{-3} $(mg/kg/day)^{-1}$ (U.S. Environmental Protection Agency, 1990). This estimate was based on several studies involving rats and mice which experienced increased incidences of hepatocellular carcinomas, and renal and liver tumors (NCI, 1976; Jorgenson and others, 1985). An inhalation cancer potency factor of 8.1×10^{-3} $(mg/kg/day)^{-1}$ was derived by the USEPA using a root-to-root extrapolation of the oral cancer potency factor as stated above (U.S. Environmental Protection Agency, 1990).

Oral Cancer Potency Estimate: 6.1×10^{-3} $(mg/kg/day)^{-1}$ (U.S. Environmental Protection Agency, 1990).

Inhalation Cancer Potency Estimate: 8.1×10^{-2} $(mg/Kg/day)^{-1}$ (U.S. Environmental Protection Agency 1990).

Noncarcinogenic Effects:

The Office of Health and Environmental Assessment has derived a chronic oral reference dose of 1×10^{-2} mg/kg/day (U.S. Environmental Protection Agency, 1990) based on an increased levels of SGPT and SGOT in high-dose animals (beagle dogs administered doses of 30 mg/kg/day for 6 days/week for 7.5 years) (Heywood and others, 1979). Uncertainty factors of 10 each were applied to the LOAEL of 12.9 mg/kg/day to account for the interspecies conversion, protection of sensitive human subpopulations, and concern that the effects seen was a LOAEL and not a NOAEL (U.S. Environmental Protection Agency, 1990). An inhalation RfD is not currently available.

Oral RfD: 1×10^{-2} mg/kg/day (U.S. Environmental Protection Agency, 1990).

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B.11 CHROMIUM

I. Occurrence and Use

Chromium is a naturally occurring element that is found in soil and in volcanic dust and gases. It is found in the environment in three major states: chromium (0), chromium (III), and chromium (VI). It is only found in nature as in combined oxidation states, and rarely in the zero valence state (IARC 1980). This profile focuses on the two most common forms of the metal, chromium (III) and chromium (IV).

Chromium (III) occurs naturally in the environment. With the exception of acetate and nitrate salts, the trivalent chromium compounds are generally insoluble in water. In most biological systems, chromium is present in the trivalent form. It is an essential nutrient required in trace quantities for normal glucose metabolism (Anderson 1981).

Chromium (VI) rarely occurs in nature apart from anthropogenic sources because it is readily reduced in the presence of oxidizable organic matter. With the exception of a few compounds, hexavalent chromium exists only as oxo species that are strong oxidizing agents. The oxidizing potential of chromate ions depends on pH. Chromate and dichromate compounds are the most common forms of hexavalent chrome. They are soluble and stable in natural waters because of the low concentration of reducing matter (EPA 1984). The ammonium and alkali metal salts of hexavalent chromium are also generally water soluble, whereas the alkaline metal salts (eg. calcium, strontium) are less soluble in water.

Chromium (VI) and Chromium (0) are produced by industrial processes. The metal Chromium (0) is a steel-gray solid with a high melting point. Chromium is used mainly for making steel and other alloys. In the form of the mineral chromite, it is used by the refractory industry to make bricks for metallurgical furnaces. Chromium compounds produced by the chemical industry are used for chrome plating, the manufacture of pigments, leather tanning, wood treatment, and water treatment (ATSDR, 1989).

II. Physical and Chemical Properties of Chromium III and Chromium VI

Molecular Weight	52.00[1]
Water Solubility, mg/l	0.0E+00[2]
Vapor Pressure, mm Hg	0.0E+00[3]
Bioaccumulation Factor for Fish	2.0E+01[4]
Bioaccumulation Factor for Shellfish	2.0E+03[4]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Weast, 1979
[3]USEPA, 1981
[4]Napier et al., 1980

III. Environmental Fate and Transport

Chromium occurs naturally in the earth's crust. Soil chromium levels were reported as follows: 37 ppm geometric mean in the USA (Shacklette and Boerngen 1984) and 5-3,000 ppm with 0.5 -10,000 ppm extreme limits (Dragun 1988). In soil, chromium probably occurs as insoluble Cr (III) oxide (Cr_2O_3-nH_2O), since the organic matter in soil tends primarily to convert soluble chromate (chromium (VI)) to insoluble Cr_2O_3 . Chromium in soil may be transported to the atmosphere in the form of aerosol, while runoff and leaching may transport chromium from soil to surface waters and groundwaters. Flooding of soils and the subsequent anaerobic decomposition of plant matters may increase mobilization of chromium in soils due to formation of soluble complexes with humic substances.

Chromium is primarily removed from the atmosphere by fallout and precipitation. Atmospheric chromium removed by physical processes enters surface water or soil predominantly; however, prior to their removal, chromium particles of aerodynamic diameter (less than 20 um) may remain airborne for long periods and may be transported long distances. In the atmosphere, chromium (VI) may be reduced to chromium (III) at a significant rate by vanadium (V^{2+}, V^{3+} , and VO_2^+), Fe^{2+} , HSO_3^- and AS^{3+} (EPA 1987).

Because there are no known chromium compounds that can volatilize from water, transport of chromium from water to the atmosphere is not likely other than by transport by windblown sea sprays. In surface waters, chromium may be transported in five forms, as follows: 1) in solution and organic complexes, 2) adsorbed 3) precipitated and co-precipitated 4) in organic solids and 5) in sediments (Towill, et al., 1978). The exact chemical forms of chromium in surface waters are not well defined. Although most of the soluble chromium in surface water may be present as Cr (VI) (Towill, et al., 1978), a small amount may be present as Cr (III) organic complexes (DeGroot and Allersma, 1978; Fukai, 1967). Most of the chromium (III) in surface water is eventually expected to precipitate in sediments. Small amounts of chromium (III) may remain in solution as soluble complexes. Chromium (VI) will predominantly be present in soluble form. Chromium (VI) will eventually be reduced to chromium (III) by organic matter present in water. The residence time of chromium in lake water was estimated to be between 4.6 to 18 years.

The oxidation of chromium (III) to chromium (VI) by solid MnO_2 in water remained unaffected by dissolved oxygen, and the process was very slow in slightly acidic (pH 6) and basic solutions (pH 11) because of the low solubility, the $Cr(OH)^3$ that is formed at these pHs (Eary and Rai 1987). Therefore, this oxidation process would not be significant in most natural waters where the pH range is usually between 6 and 9 because this process is very

slow in slightly acidic water. Similar oxidation of chromium (III) to chromium (VI) in the atmosphere is unlikely (EPA 1987).

IV. Routes of Exposure, Absorption, Distribution, Transport, and Degradation

The general population is exposed to small amounts of chromium results by breathing air, and ingesting drinking water and food containing chromium. Much higher exposure to chromium results from working in certain chromium industries and to people who smoke cigarettes. The two largest sources of chromium emissions in the atmosphere are from the chemical manufacturing industry and the combustion of natural gas, oil, and coal. A detailed list of other sources of exposure to chromium can be found in the ATSDR Toxicologic Profile (1989).

Chromium can enter the body via oral, inhalation and dermal exposure. For the general population, the gastrointestinal tract is the primary route of entry, although entry through the airways can be significant near industrial sources. Following occupational exposure, the airways and skin are the primary routes of uptake. Inhalation studies conducted using animals indicate that 53% to about 85% of the chromium from chromium (VI) compounds is cleared from the lungs after intratracheal injection compared to 5 to 30% clearance of the chromium from chromium (III) compounds (Baetjer, et al., 1959, Visek, et al., 1953, Wiegand, et al., 1984).

Via the oral route, Donaldson and Barreras (1966) found that about 0.4% of the radioactive chromium from an oral dose of a labelled compound containing chromium (III) was absorbed and about 10.6% of the labelled compound containing chromium (VI) were absorbed in humans. Anderson, et al., (1983) confirmed minimal (about 0.4%) gastrointestinal absorption of dietary and supplemental chromium in humans. The supplemental chromium was provided as a tablet containing 200 μg chromium (III) as chromic chloride.

In the dermal studies, using volunteers, Mali (1963) found that potassium dichromate (VI) but not chromic (III) sulfate penetrated intact epidermis. Samitz and Shrager (1966) found that absorption of chromic sulfate was negligible, with slightly larger amounts of chromium (III) nitrate absorbed. The absorption of chromic (III) chloride was similar to potassium dichromate. Randall and Gibson (1987), and Lindberg and Vesterberg (1983a), indicate some absorption of chromium (III) and (VI) via the skin.

Following inhalation exposure to chromite (III) dust for 28 days, chromium is absorbed and distributed in animals to the kidneys, lungs and spleen (Kamiya, et al., 1981). Once absorbed, chromium (VI) is reduced to chromium (III) (Kitagawa, et al., 1982, Levis, et al., 1978). During reduction of chromium (VI) in the plasma, chromium protein complexes are formed; these complexes are excreted by the kidneys. Formation of these complexes can be harmful if they occur at high enough levels. In addition, chromium (VI) crosses cell membranes easily and is reduced inside cells, forming chromium protein complexes during reduction. Once

complexes with protein, chromium cannot leave the cell. Chromium (III) crosses cell membranes less readily, does not readily bind to intracellular protein and can diffuse out. Chromium (VI) can be reduced to chromium (III) in vitro by gastric juice, but whether intragastric reduction occurs in vivo is not known.

The toxicity of chromium is attributed primarily to the hexavalent form. In humans and experimental animals, gastrointestinal absorption of inorganic salts of chromium III is low (from 0.5% to 3%). However, organic complexes of chromium III are more readily absorbed (approximately 10% to 25%). The spleen and kidneys of rats were shown to have the highest concentration of chromium when chromium chloride intravenous doses (Hopkins, 1965) or chromic chloride in drinking water (Mackenzie et al., 1958) were administered. (EPA, 1985; Casarett and Doull, 1986).

V. Acute Toxicity

A wide variety of acute effects have been observed in humans, including contact dermatitis, skin ulcerations, kidney failure, nasal irritation, nosebleeds, respiratory congestion, teeth erosion and discoloration, stomachaches and kidney failure. These effects are reported to be much more severe for Chromium VI exposure than for Chromium III exposure.

Langard and Norseth (1986) indicated that oral doses of 2-5 g of unspecified chromate compounds (chromium (VI)) are fatal to humans. Acute poisoning symptoms included gastrointestinal bleeding, massive fluid loss and death in some individuals following cardiovascular shock. These effects tended to occur within 12 hr of ingestion. When the ingested dose was reduced to less than, or equal to, 2 g, tubular renal necrosis and diffuse liver necrosis developed and contributed to the cause of death in fatal cases. Liver and kidney effects developed 1 to 4 days after ingestion.

Via the dermal route, patients died after antiscabies ointment containing chromium (VI) was applied to the skin (Brieger 1920). Symptoms included necrosis at the application site, nausea, vomiting, shock and coma. Autopsies revealed tubular necrosis and hyperemia of the kidney. Other reviews of death after dermal exposure to chromium compounds include Major (1922) and Fritz, et al., (1959). It is important to note that these cases involved damaged rather than intact skin.

VI. Chronic Toxicity

Long-term oral exposure of animals to relatively low levels of chromium compounds has not resulted in systemic toxic effects. The effects of chromium (VI) on the nasal mucosa and lung function in humans may be the most sensitive noncancer end point for chronic inhalation exposure to chromium (VI) compounds. Other effects observed following chromium (VI) exposure include effects on the immune system, nervous system and liver. Dermal exposure to both chromium (III) and chromium (VI) can result in chromium sensitization.

In a Russian study (Kuperman 1964), 10 normal individuals were exposed to chromium (VI) aerosols of unspecified composition at 0.0015 to 0.04 mg/m^3. Concentrations of 0.01-0.024 mg/m^3 chromium (VI) sharply irritated the nose when inhaled for short periods. The most sensitive individual responded at a concentration of 0.0025 to 0.004 mg/m^3 chromium (VI). It was not known if this was a reaction to chromium (VI) or to the acidity of the aerosol. Many cases of nasal mucosal injury (inflamed mucosa, ulcerated or perforated septum) in workers exposed to CrO_3 have been reported (Bloomfield and Blum 1928, Gresh 1944, Zvaifler 1944, Klienfeld and Russo 1965, Vigliani and Zurlo 1955). Effects occurred at chromium (VI) concentrations ranging from 0.06 to 0.72 mg/m^3. The length of exposure to these cases was highly variable. Cohen and Kramkowski (1973) and Cohen, et al., (1974) found that 12/37 workers employed by a chrome-plating plant developed nasal ulceration or perforation within 1 year of being employed. Airborne chromium (VI) concentrations ranged from less than 0.71 to 9.12 $\mu g/m^3$. Other reported cases of nasal and lung effects due to chromium exposure are (Hanslian, et al., 1967, Markel and Lucas 1973 and Lindberg and Hedenstierna 1983).

Other respiratory effects have been reported in workers exposed to chromium compounds. Alwen and Jonas (1938), Fischer-Wasels (1938), Koelsch (1938), Lehmann (1932), Mancuso (1951) reported that workers exposed chronically to chromate (VI) dust resulted in chronic irritation of the respiratory tract, congestion and hyperemia, chronic rhinitis, congestion of the larynx, polyps of the upper respiratory tract, chronic inflammation of the lungs, emphysema, tracheitis, chronic bronchitis, chronic pharyngitis, and perivascular lung markings, enlargement of hilar region lymph nodes and adhesions of the diaphragm.

Although immune effects have not been reported in humans following exposure to chromium compounds, immune effects have been reported in animals. Inhalation exposure to chromium (VI) and chromium (III) compounds at concentrations of 0.2 - 0.9 mg/m^3 resulted in depression of some indices of immune system function in animals, whereas chromium (VI) at concentrations of less than 0.1 mg/m^3 chromium resulted in stimulation. For a review, see Steven, et al., (1976), Camner, et al., (1974) and Waters, et al., (1975).

Sensitization can occur after exposure of humans and guinea pigs to chromium via the dermal route (Maloof 1955, Milner 1980, Avnstorp and Menne 1982, Husain 1977, Gross, et al., 1968, Schwartz-Speck and Grundsman 1972, Jansen and Berrens 1968, Siegenthaler, et al., 1983). Although reactions to chromium (VI) are more common, reactions to chromium (III) can also occur. Inhalation exposure of workers to chromium compounds may also result in sensitization (Moller, et al., 1986). Because the development of hypersensitivity is highly variable between individuals, it is not possible to develop a generalizable dose-response relationship for this effect.

Chromium may have central nervous system effects (Diaz-Mayans, et al., 1986 and Mathur, et al., 1977). Mathur, et al., 1977 reported changes in brains of rabbits given daily intraperitoneal doses of chromium (III) nitrate or potassium dichromate (VI) at 2 mg/kg chromium for 3 or 6 weeks. These changes included neuronal degeneration in the cerebral cortex, marked

chromatolysis, nuclear changes in neurons, neuronal degeneration in the cerebral cortex accompanied by neuronophagia, neuroglial proliferation and meningeal congestion. Abnormal deposits of chromium in the brains of patients with encephalopathies treated with radiological contrast substances containing chromium (Duckett 1986) provide limited evidence that the brain may also be a target organ for chromium toxicity in humans.

Chromium is a nephrotoxin producing tubular necrosis, with low doses acting specifically at the proximal convoluted tubule of the kidney. Human and animal studies do not clearly define the doses that produce adverse effects. Powers, et al., (1986) reported marked acute proteinuria, glycosuria, phosphaturia, enzymuria, severe electrolytic imbalance, increased kidney weight and morphological changes in the kidneys of rats given a single subcutaneous injection of sodium dichromate (chromium (VI)) at a dose of 20 mg/kg. Animal studies documenting kidney effects include Kirschbaum, et al., 1981, Baines 1965, Evan and Dail 1974, Powers, et al., 1986, Laborda, et al., 1986, Berndt 1976 and Srivastava, et al., 1985.

Liver effects have occurred in humans and animals following inhalation and parenteral exposure to chromium compounds. Hepatic changes observed in animals exposed daily for 3-6 weeks to chromic compounds (2 mg/kg of chromic (III) nitrate or potassium dichromate (VI)) included congestion and dilation of the central veins and sinusoids, discrete foci of necrosis and hemorrhage in liver parenchyma, nuclear pleomorphism, multinucleated cells in the lobules and bile duct proliferation. Studies indicate chromium (VI) caused more damage than chromium (III).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity:

Results of in vivo mutagenicity studies have consistently shown positive results for chromium (VI) compounds (Rasmuson 1985, Knudsen 1980, Paschin, et al., 1982, Newton and Lilly 1986, Bigaliev, et al., 1978, Wild 1978, DiPaolo and Castro 1979, Bigaliev, et al., 1977 and Sarto, et al., 1982) and negative results for chromium (III) compounds (Wild 1978) in standard tests. Other in vitro studies reviewing mutagenicity of chromium include Bianchi and Levis 1985, EPA 1984a, Bonati, et al., 1976 and Singh 1983. These results support the carcinogenicity findings in animal studies for chromium. Chromium (III) has tested positive in only isolated nuclei and purified DNA, in studies at high concentrations and in cells with phagocytic activity. The difference in activity of the two valence states of chromium is a result of differences in ability to permeate cell membranes.

Carcinogenicity:

Epidemiological studies reviewed in IARC (1980, 1982, 1987) and EPA (1984, 1986c) clearly indicate an increased respiratory cancer risk in chromate production workers (Baetjer 1950, Alderson, et al., 1981, Hayes, et al., 1979, Machle and Gregorius 1948, Mancuso and Heuper

1951, Mancuso 1975, Ohsaki, et al., 1978 and Taylor 1966). Increased risks of respiratory cancer have also been found in some studies of chrome pigment workers (Langard and Norseth 1975 and Davies 1984), chrome-plating workers (Franchini, et al., 1983, Sorahan, et al., 1987) and ferrochromium workers (Langard, et al., 1980). Mancuso (1975) found that the lung cancer mortality was dose-related to total chromium exposure and with a latency period of 27-36 years after initial exposures. The epidemiological studies do not clearly implicate specific compounds, but do implicate chromium (VI), as opposed to Cr (III), as the carcinogenic form. Based on the epidemiological evidence, the EPA (1986c) and IARC (1987) have concluded that exposure to chromium (VI) compounds via the inhalation route is carcinogenic to humans.

Teratogenicity:

There are no human or animal studies of developmental toxicity following inhalation, oral or dermal exposure to chromium. Exposure to chromium (III) or chromium (VI) compounds may result in developmental effects via the parenteral route, however. In studies by Gale (1978) and Gale and Bunch (1979), increased fetal death and an increase in external abnormalities were observed in hamsters treated by intravenous injection with CrO_3 (chromium (VI)) on a single day of gestation. Matsumoto, et al., 1976 observed fetal weight depression and an increase in external abnormalities in the fetuses of mice treated intraperitoneally with $CrCl_3$ at 14.64-24.4 mg/kg chromium (III) on the eighth day of gestation. No effects were observed at 9.76 mg/kg.

Some studies indicate that chromium (III) and chromium (VI) compounds may affect reproduction. Behari, et al., 1978 observed testicular effects in rabbits injected intraperitoneally with chromium (III) nitrate or potassium dichromate (VI) at 2 mg/kg/day for 3 or 6 weeks. Microscopic examination of the testes showed thickening of the tunica albuginea, congestion of blood vessels and degenerative changes of the seminiferous epithelium in chromium (III)-treated rats. Chromium (VI) treatment resulted in mild edema of the interstitial tissue and congestion of the blood vessels; at 6 weeks the tubules were devoid of spermatocytes.

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

EPA (1994) has classified inhaled chromium VI in Group A - Human Carcinogen by inhalation. Chromium (III) is not considered to be a carcinogen.

Carcinogenic Effects of Chromium III:

The EPA (1994) has not evaluated chromium (III) for human carcinogenic potential.

Carcinogenic Effects of Chromium VI:

INHALA[illegible] UNIT RISK: 1.2E-2 per (ug/cu.m) or
$4.1 \times 10^{+1}$ $(mg/kg/day)^{-1}$

DISCUSSION OF CONFIDENCE :

The inhalation cancer potency factor was derived from a study based on the occupational exposure of workers to chromium and deaths from lung cancer (Mancuso 1975). Results of studies of chromium exposure are consistent across investigators and countries. A dose-relationship for lung tumors has been established. The assumption that the ratio of Cr III to Cr VI is 6:1 may lead to a 7-fold underestimation of risk. The use of 1949 hygiene data, which may underestimate worker exposure, may result in an overestimation of risk. Further overestimation of risk may be due to the implicit assumption that the smoking habits of chromate workers were similar to those of the general white male population, since it is generally accepted that the proportion of smokers is higher for industrial workers than for the general population.

Because there is no evidence for the carcinogenicity of chromium compounds by the oral route of administration, the EPA has not derived an oral cancer potency factor (EPA 1994, 1993) for chromium (VI).

Noncarcinogenic Effects of Chromium III:

ORAL RFD: 1E+0 mg/kg/day (as an insoluble salt)
CRITICAL EFFECT/TARGET ORGAN: No effects observed
ORAL RFD UNCERTAINTY :
UF = 100. The factor of 100 represents two 10-fold decreases in mg/kg bw/day dose that account for both the expected interhuman and interspecies variability to the toxicity of the chemical in lieu of specific data.

ORAL RFD MODIFYING FACTOR :
MF = 10. The additional modifying factor of 10 is adopted to reflect uncertainty in the NOEL because: 1) the effects observed in the 90-day study were not explicitly addressed in the 2-year study and, thus, the highest NOAEL in the 2-year study may be a LOAEL; 2) the absorption of chromium is low (<1%) and is influenced by a number of factors; thus, a considerable potential variation in absorption exists; and 3) animals were allowed to die naturally after feeding stopped (2 years) and only then was histology performed.

ORAL RFD CONFIDENCE :
Study: Low
Data Base: Low
RfD: Low

The principal study is rated low because of the lack of explicit detail on study protocol and results. Low confidence in the data base reflects the lack of high-dose supporting data. The low confidence in the RfD reflects the foregoing, but also reflects the lack of an observed effect level. Thus, the RfD, as given, should be considered conservative, since the MF addresses only those factors which might lower the RfD.

Noncarcinogenic Effects of Chromium VI:

ORAL RFD: 5E-3 mg/kg/day
CRITICAL EFFECT/TARGET ORGAN: No effects reported
ORAL RFD UNCERTAINTY: UF = 500. The uncertainty factor of 500 represents two 10-fold decreases in dose to account for both the expected interhuman and interspecies variability in the toxicity of the chemical in lieu of specific data, and an additional factor of 5 to compensate for the less-than-lifetime exposure duration of the principal study.

ORAL RFD CONFIDENCE:
Study: Low
Data Base: Low

Confidence in the chosen study is low because of the small number of animals tested, the small number of parameters measured and the lack of toxic effect at the highest dose tested. Confidence in the data base is low because the supporting studies are of equally low quality, and teratogenic and reproductive endpoints are not well studied. Low confidence in the RfD follows.

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B.12 COPPER

I. Occurrence and Use

Copper is ubiquitous in the earth's crust, primarily found as sulfides and oxides. Natural levels in soil range from 8 to 90 mg/kg (Kabata-Pendias and Pendias, 1984).

About half of copper production is used as a conductor in electrical equipment due to its high conductivity. It is used in many alloys: beryllium-copper, brass, bronze, gunmetal, bell metal, german silver, etc. These are used in plumbing, electronics, and the manufacture of various parts and goods. Copper compounds are used in pesticides, antifouling paints, algicides, fungicides, and insecticides. Some compounds are used as pigments in paints and ceramics (Carson et al., 1987).

II. Physical and Chemical Properties of Copper

Molecular Weight	63.55[1]
Water Solubility, mg/l	0.0E+00[2]
Vapor Pressure, mm Hg	0.0E+00[3]
Bioaccumulation Factor for Fish	5.0E+01[4]
Bioaccumulation Factor for Shellfish	4.0E+02[4]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Weast, 1979
[3]Lyman et al., 1982
[4]Napier et al., 1980

III. Environmental Fate and Transport

Factors affecting deposition of copper in soil include the degree of weathering, the nature and intensity of the soil formation, drainage, Ph, oxidation-reduction potential, and the amount of organic matter in the soil. Since copper is likely to be more mobile under acidic than alkaline conditions, the relation of pH to copper in the environment has been of great concern to agriculturalists and biologists. Alkaline conditions in the soil and surface water favor precipitation of copper. Acid conditions promote solubility of copper, increase the concentration ionic copper, and thereby change the microorganism and other aquatic animal populations, depending on tolerance for various levels of copper in solution. In soils exposed to atmospheric deposition, high levels of copper and other metals may occur that can be

directly toxic to certain soil microorganisms and can disrupt important microbial processes in soil, such a nutrient cycling (HSDB, 1992).

IV. Routes of Exposure, Distribution, Absorption, Transport, and Degradation

The principal route of exposure to copper is by ingestion of contaminated food and water, although inhalation exposure can occur in the workplace. Copper is absorbed in the stomach and duodenum, and typically about half of a dose will be absorbed. The main sites of deposition are the liver brain and muscles, and a 70-kg adult stores 70 to 120 mg. The major excretion route is the bile (Carson et al., 1987). Copper is an essential nutrient, but is toxic to humans at high levels.

V. Acute Toxicity

Exposure to metallic copper dust by inhalation can cause a short-term illness similar to metal fume fever that is characterized by chills, fever, aching muscles, dryness of the mouth and throat, and headache. Exposure to copper fumes can produce upper respiratory tract irritation, a metallic or sweet taste, nausea, and occasional discoloration of skin and hair. Individuals exposed to dusts and mists of copper salts may exhibit congestion of nasal mucous membranes, sometimes congestion of the pharynx, and occasionally ulceration with perforation of the nasal septum. If sufficient concentrations of copper salts reach the gastrointestinal tract, they act as irritants and can produce salivation, nausea, vomiting, gastritis and diarrhea. The elimination of ingested ionic copper by vomiting and diarrhea generally protects the patient from more serious systemic toxic effects, which can include hemolysis, hepatic necrosis, gastrointestinal bleeding, oliguria, azotemia, hemoglobinuria, hematuria, proteinuria, hypotension, tachycardia, convulsions and death. Copper salts act as skin irritants upon dermal exposure, producing an itching eczema. Conjunctivitis or even ulceration and turbidity of the cornea may result from the direct contact of ionic copper with the eye (Clement Associates, 1985).

VI. Chronic Toxicity

Chronic exposure via inhalation may produce "metal fume fever," which is an influenza-like syndrome, with attacks lasting a day or so. Long-term exposure may also produce nasal ulcerations and bleeding. Anemia has also been observed as a symptom of exposure among workers exposed to copper in the air at levels at of below the TLV (Carson et al., 1987). Chronic exposure of rats and swine via ingestion of copper or its compounds at 2-40 mg/kg/day has resulted in pathological changes of the liver, kidneys, blood, gastrointestinal tract, and in a variety of tissues (ATSDR, 1989).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity:

Copper appears to increase the mutagenic activity of triose reductase and ascorbic acid in bacterial test systems. However, copper itself does not appear to have mutagenic or teratogenic effects in animals or humans (Clement Associates, 1985).

Carcinogenicity:

Available data relating to copper carcinogenicity are presently inadequate. Thus, according to the guidelines of the U.S. EPA, copper is not classifiable as to human carcinogenicity. A long term study of copper hydroxyquinoline administered to mice in their diet yielded negative results in both strains used ($B6C3F_1$ and $B5AKF_1$). However, subcutaneous exposure of male $B6C3F_1$ mice yielded a highly significant elevation in the incidence of reticulum cell sarcomas, although elevated incidences of tumors were not observed in treated $B5AKF_1$ mice or treated female mice of either strain. Studies involving Wistar rats are also presently inconclusive (ATSDR, 1989).

Teratogenicity:

Numerous studies have documented the teratogenicity of copper compounds. Bioassay animals to which copper compounds have been shown to be teratogenic include C57BL and DBA strain mice (copper sulfate at 25.9 and 51.7 mg/kg/day), and hamsters (copper sulfate at 2.13 mg/kg and copper citrate a 0.25-1.5 mg/kg)(ATSDR, 1989).

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

Copper is not classified as a human carcinogen (Group D), based on inadequate animal data from assays of copper compounds, equivocal mutagenicity data and lack of any human data.

EPA Dose-Response Parameters (IRIS, 1994):

No carcinogenic or noncarcinogenic dose-response parameters are reported in IRIS for copper. HEAST reports a current drinking water standard of 1.3 mg/l. This can be used to obtain an RfD of 0.0371 mg/kg/day, assuming a 70 kg body weight and water consumption of 2 L/day.

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B.13 PPDDT

I. Occurrence and Use

DDT, Dichlorodiphenyltrichloroethane, was one of the most widely used pesticides agricultural crops, and for controlling insects that carry diseases such as malaria and typhus. Technical DDT is primarily a mixture of three forms (p,p'-DDT, o,p'-DDT, and o,o'-DDT); it also contains its degradative breakdown products (i.e., metabolites), DDE (p,p'-dichlorodiphenyldichloroethylene) and DDD (p,p'-dichlorodiphenyldichloroethane). These compounds are among the most persistent environmental contaminants, and have the ability to bioaccumulate in biological tissues. DDD was also used exclusively as a pesticide, and as a medicinal to treat cancer of the adrenal gland (ATSDR, 1989).

DDT, DDE and DDD do not occur naturally in the environment; their presence in the water, land and air is generally the result of past production and use. Their use as insecticides was banned in the US in 1972 because of their high degree environmental persistence except in cases of public health emergencies.

II. Physical and Chemical Properties

DDT

Molecular Weight	354.49
Water Solubility, mg/l	5.0E-03
Vapor Pressure, mm Hg	5.5E-06
Henry's Law Constant, atm-m^3/mole	5.1E-04
Octanol-Water Partition Coefficient	1.6E+06
Bioaccumulation Factor for Fish	3.0E+04
Bioaccumulation Factor for Shellfish	8.4E+03

Source: Multi-Media Exposure Assessment Manual, 1989

DDE

Molecular Weight	328.03
Water Solubility, mg/l	0.12

Vapor Pressure, mm Hg	6.5E-06
Henry's Law Constant, atm-m^3/mole	6.8E-05
Octanol-Water Partition Coefficient	7.00
Bioaccumulation Factor for Fish	NA[1]
Bioaccumulation Factor for Shellfish	NA

Source: ATSDR, 1989
[1]NA = Not available

DDD

Molecular Weight	320.05
Water Solubility, mg/l	0.160
Vapor Pressure, mm Hg	10.2E-07
Henry's Law Constant, atm-m^3/mole	ND[1]
Octanol-Water Partition Coefficient	6.2E+00
Bioaccumulation Factor for Fish	NA[2]
Bioaccumulation Factor for Shellfish	NA[2]

Source: ATSDR, 1989
[1]ND = No data
[2]NA = Not available

III. Environmental Fate and Transport

Historically, DDT was released to the environment during its formulation and extensive use as a pesticide in agricultural and vector control applications. Although it was banned for use in this country in 1972, it is still being used in several areas of the world. DDT and its environmental degradation products preferentially bind to soil and sediment, where they may be subject to photodegradation on the surface and biodegradation in the subsurface. However, under certain conditions, DDT may persist for long periods of time or may be converted to DDE, which persists even longer. DDT, DDE, and DDD in the atmosphere are subject to photodegradation or redeposition by rain or dry deposition. DDT, DDE, and DDD in water

are subject to sedimentation, volatilization, photodegradation, and uptake into the food chain. Both DDT and DDE bioaccumulate in organisms, and levels are subject to increase as they advance up the food chain. DDT and its metabolites have been detected in virtually all media (ATSDR, 1989).

DDT and its metabolites may be transported from one medium to another by the processes of solubilization, adsorption, bioaccumulation, or volatilization. It appears however, that the dominant fate of these compounds is adsorption to soil and sediments. Studies of DDT transformations in soils indicate prolonged persistence. During these extended periods of time, DDT, DDE, and DDD undergo extensive adsorption to soil particles, as predicted by their organic carbon partition coefficients (with K_{oc} values of 2.4E+05, 4.4E+06, and 7.7E+05 for DDT, DDE, and DDD, respectively), and by their low solubilities in water. Therefore, loss of these compounds in runoff is primarily due to transport of particulates to which these compounds are bound. Since they are bound strongly to soil, they are not easily displaced from their site of application, nor do they tend to leach to groundwater (ATSDR, 1989).

Although atmospheric levels of DDT and its metabolites are now negligible due to their usage ban, atmospheric deposition is possible by two other mechanisms. DDT and DDE may volatilize from water and soil surfaces, as predicted by their Henrys Law Constants and vapor pressures, and by the organic content of the soil under investigation. The tendency of DDD to volatilize is approximately three-fold less than that of DDT or DDE (ATSDR, 1989). Small particles which carry DDT or its degradation products may also be distributed through the atmosphere. Residues are removed from the atmosphere to the greatest extent by precipitation, but also by diffusion into large bodies of water, and by chemical transformation (ATSDR, 1989).

Because of their low water solubility and lipophilic nature, DDT, DDE, and DDD in water is partitioned, transported, or converted in several ways, including adsorption to sediments, bioconcentration in aquatic organisms, volatilization, photodegradation, and biodegradation (ATSDR, 1992).

IV. Routes of Exposure, Absorption, Distribution, Transport, and Degradation

The most important route of human exposure is nonoccupational, resulting from the fallout of DDT aerosols and its metabolites in air, water and soil, and sediments.

DDT, DDE, and DDD is absorbed most readily in the human body through the GI tract, when a person eats food or accidentally ingests soil contaminated with these compounds. Absorption through the lungs is only considered a minor pathway, because inhaled DDT particles are generally too large to pass through the lungs into the body, and are more likely coughed-up and ingested. These compounds are very poorly absorbed through the skin.

Once inside the body, DDT and its metabolites are distributed to all adipose tissues in proportion to their respective lipid content. Metabolism of these compounds is very slow, and tissue concentrations may remain constant over time, or bioaccumulate with continued exposure. However, amounts of DDT in the body will decrease with decreasing exposure. The route of excretion is primarily via urine, but it has also been demonstrated that these contaminants may be eliminated in the female body in breast milk (Takei et al., 1983, and ATSDR, 1989).

Non-Carcinogenic Health Effects

V. Acute Expsoure

Humans: Symptoms of acute and subacute exposures to high doses of DDT in humans include paresthesia of the tongue, lips and face, apprehension, hypersusceptibility to stimuli, irritability, dizziness, disturbed equilibrium, tremor and tonic and clonic convulsions. Motor unrest and fine tremors associated with voluntary movements progress to coarse tremors without interruption in moderate to severe poisoning.

Laboratory animals: Animals may develop motor coordination disabilities, which can progress to tremors, following acute exposure to DDD; after fatal doses, death occurs in 24 to 72 hours (ATSDR, 1989).

VI. Chronic Exposure

Humans: No clinical or laboratory evidence of injury to man from occupational (repeated) exposures to DDT has been reported although the amount stored in body fat was higher than that found in the general population.

Laboratory Animals: The primary effect appears to be malfunctions of the central nervous system. A 2-year feeding study in rats caused severe body tremors, as well as morphological changes in the liver (ATSDR, 1989). Chronic oral toxicity exposures to experimental animals resulted in tremors, increased mortality and increased liver, kidney and spleen weights with degenerative changes occurring in the liver.

Carcinogenic Health Effects

VII. Acute Exposure

Humans: No information is available.

Laboratory animals: DDT is one of the most widely studies pesticides in animals, and data are available on a number of carcinogenicity studies in several species. Carcinogenic effects

occur exclusively via the oral route; there is no evidence that DDT, DDE or DDD induce cancer via inhalation or dermal exposure (ATSDR, 1989).

VIII. Chronic Exposure

Humans: No information is available.

Laboratory animals: Chronic oral exposure of DDT in the diet causes cancer in mice, rats or hamsters. Mice develop liver hepatomas following exposure to 32.5 mg DDT/kg/day for 15 to 30 weeks, and observed for 50 to 105 weeks following cessation of treatment (Tomatis et al., 1974). Chronic exposure (>1 year) to DDT also causes cancer in rats and hamsters. Rats maintained on diets containing DDT for more than 2 years or at doses higher than 25 mg DDT/kg/day developed liver tumors, primarily in female rats (Cabral et al., 1982, and Rossi et al., 1977). Rossi et al. (1983) reported increases in adrenal gland tumor in hamsters exposed to 83 mg DDT/kg/day.

Similar carcinogenic effects were observed in studies employing oral exposure to DDE and DDD. There is no evidence to indicate that DDT, DDE, and DDD are carcinogenic in dogs of nonhuman primates (ATSDR, 1989).

Mutagenicity and Teratogenicity

Mutagenicity:

The mutagenic activity of DDT and its metabolites is relatively weak (ATSDR, 1992).

Teratogenicity:

Although there have been no reports of teratogenic effects from DDT, with the possible exception of ringtail in rats (Ottoboni 1969), DDT has consistently resulted in decreased fertility in mice (Keplingler et al.,. 1968, McLachlan and Dixon 1972, Schmidt 1973); however, conflicting results have been reported in rats (Fitzhugh and Nelson 1947, Fitzhugh 1948, Treon and Cleveland 1955, Clement and Okey 1974, Jonsson et al.,. 1975, Ware and Good 1967 and Dubey et al.,. 1971) and dogs (Deichmann et al.,. 1971, Deichmann and MacDonald 1971 and Ottoboni et al.,. 1977). (EPA 1980, 1984, Klaassen et al.,. 1986 and Clayton and Clayton 1981).

EPA Carcinogenic Classification and EPA Dose-Response Parameters

X. EPA Carcinogenic Classification:

DDT is classified as a B2 carcinogen by the EPA, based on the observation of tumors (generally of the liver) in seven studies in mice and three studies in rats.

DDE is classified as a B2 carcinogen by the EPA, based on increased incidence of liver tumors including carcinomas in two strains of mice and in hamsters, and of thyroid tumors in female rats by diet.

DDD is classified as a B2 carcinogen by the EPA, based on increases incidence of lung tumors in male and female mice, lever tumors in male mice, and thyroid tumors in male rats.

EPA Dose-Response Parameters for DDT:

ORAL RFD: 5E-4 mg/kg/day
CRITICAL EFFECT/TARGET ORGAN: Liver lesions
ORAL RFD UNCERTAINTY :
UF = 100. A factor of 10 each was applied for the uncertainty of interspecies conversion and to protect sensitive human subpopulations. An uncertainty factor for subchronic to chronic conversion was not included because of the corroborating chronic study in the data base.

ORAL RFD CONFIDENCE :
Study: Medium
Data Base: Medium
RfD: Medium

The principal study appears to be adequate, but of shorter duration than that desired; therefore, confidence in the study can be considered medium to low. The data base is only moderately supportive of both the critical effect and the magnitude, and lacks a clear NOEL for reproductive effects; therefore, confidence in the data base can also be considered medium to low. Medium to low confidence in the RfD follows.

CANCER SLOPE FACTOR (ORAL) : 3.4E-1 per (mg/kg)/day
DISCUSSION OF CONFIDENCE :

Ten slope factors derived from six studies were within a 13-fold range. The slope factor derived from the mouse data alone was 4.8E-1 while that derived from the rat data alone was 1.5E-1. There was no apparent difference in slope factor as a function of sex of the animals. The geometric mean of the slope factors from the mouse and rat data combined was identical for the same tumor site as that for DDE [3.4E-1 per (mg/kg)/day], a structural analog.

INHALATION UNIT RISK : 9.7E-5 (ug/cu.m)
DISCUSSION OF CONFIDENCE :

This inhalation risk estimate was calculated from the oral data presented in CARO.

EPA Dose-Response Parameters for DDE:

CANCER SLOPE FACTOR (ORAL) : 3.4E-1/mg/kg/day
DISCUSSION OF CONFIDENCE :

An adequate number of animals was observed. The geometric mean obtained using the slope factors from the mouse studies alone is 7.8E-1/mg/kg/day. This is within a factor of 2 of that derived from the mouse and hamster studies combined. In addition, the slope factor for DDE was within a factor of 2 of the slope factors for liver tumors for three structurally similar compounds: DDT, 3.4E-1/mg/kg/day; DDD, 2.4E-1/mg/kg/day; and Dicofol, 4.4E-1/mg/kg/day.

EPA Dose-Response Parameters for DDD

CANCER SLOPE FACTOR (ORAL) : 2.4E-1/mg/kg/day
DISCUSSION OF CONFIDENCE

An adequate number of animals was tested. The slope factor was calculated using tumor incidence data from only one dose. The slope factor was similar to, and within a factor of 2, of the slope factors for this same site of three other structurally similar compounds: DDT, 3.4E-1/mg/kg/day; DDE, 3.4E-1/mg/kg/day; and dicofol, 4.4E-1/mg/kg/day.

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B.14 LEAD

I. Occurrence and Use

Lead (Pb) is a major environmental contaminant and one of the most common pollutants at hazardous waste sites. Combustion of gasoline is the major source of lead, as well as being a component of automotive batteries and paint.

Air emissions from combustion sources and lead paint are the primary anthropogenic sources of environmental lead.

II: Physical and Chemical Properties of Lead

Lead is a gray-white metal of silvery luster that has a low melting point (327.5°C) and a boiling point of 1740°C. The metal is soft, malleable and ductile, a poor electrical conductor and highly impervious to corrosion. A listing of the solubilities and physical properties of the more common compounds of lead is given in Weast 1982 and EPA 1986a. Most inorganic lead salts are sparingly soluble (eg., PBF_2, $PbCl_2$) or virtually insoluble ($PbSO_4$, $PbCrO_4$) in water; the notable exceptions are lead nitrate, $PB(NO_3)_2$ and lead acetate, $Pb(OCOCH_3)_2$. Inorganic lead (II) salts are, for the most part, relatively high-melting-point solids with correspondingly low vapor pressures at room temperatures. The vapor pressures of the most commonly encountered lead salts are also tabulated in EPA 1986a.

Molecular Weight	207.00[1]
Water Solubility, mg/l	0.0E+00[2]
Bioaccumulation Factor for Fish	1.0E+02[3]
Bioaccumulation Factor for Shellfish	1.0E+02[3]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Weast, 1979
[3]Napier et al., 1980

III. Environmental Fate and Transport

Although the chief source of environmental exposure is the atmosphere, Pb enters the soil and water as fallout. Lead deposited on soils can bind to a number of other naturally occurring materials, including other dusts, clays, hydrous oxides and humic and fulvic acids. Once it pollutes the soil, opportunities are enhanced for lead to be absorbed and recycled into the human food chain through grazing animals, home gardening and general agricultural activity.

The greatest concentrations of Pb have been found close to heavily travelled roads. Lead enters the human body via inhalation and ingestion.

At one time, automobile exhaust accounted for about 90% of all air emissions in the United States; the recent phase-down of lead content of gasoline and reductions in usage of leaded gasoline have, and will continue to, substantially decrease the contribution of automobile exhaust to air lead (EPA 1986b). Lead in automobile exhaust originates from the combustion of gasoline containing organic lead additives, primarily tetraethyl and tetramethyl lead. Lead is emitted from vehicles primarily as particles of inorganic lead, with a small percentage as volatile lead alkyls.

Lead released to the air deposits on terrestrial surfaces and enters the soil, where it can have several possible fates. Lead can be retained in organic complexes near the soil surface. For example, insoluble lead species may be free or adsorbed on solid inorganic or organic matrices. Studies of lead/soil interactions show that soil fixation of lead is mainly affected by pH, cation exchange capacity and organic matter content of soil. Lead appears most strongly associated with soil organic carbon fraction. If little or no organic material is in the soil, other components can regulate lead fixation. These include hydrous manganese oxide (Forstner, et al., 1981) and hydrous ferric oxide (Swallow, et al., 1980). Levels of lead in rural soils, away from industrial emissions and roadbeds, range from 5-30 μg lead/g soil (see Table 1). Levels of lead near roadbeds can be much larger (30-2000 μg/g) and will vary with past and present traffic density and vehicle speed (Page and Gange 1970; Quarles, et al., 1974; Wheeler and Rolfe 1979). Much higher levels (greater than 30,000 μg/g) can occur in the immediate vicinity of industrial sources (Yankel, et al., 1977; EPA 1986b). Lead bound to organic constituents in soil can remain in soil for long periods of time. As a result, elevated levels can persist long after sources of deposition have been reduced (Prpic-Majic, et al., 1984).

Dust is an important source of oral lead intake in infants and children. The term "dust" refers to house and outdoor dust; house dust is dust in the interior of buildings and includes such things as material from fabrics (carpet) and paint and soil tracked or blown into the house. Outdoor dust includes anthropogenic materials deposited on outside surfaces, referred to as "street dust," and the mobile uppermost layer of natural soil, referred to as "soil dust" (EPA 1986b). Outdoor dusts can be transported by wind and rain runoff (Laxen and Harrison 1977). Lead persistence in dust depends on the amount and size of particles; big particles tend to persist in air longer than smaller ones. Levels in outdoor dust near point sources have been shown to decline within 1-2 years after atmospheric emissions decreased (Morse, et al., 1979; Prpic-Majic, et al., 1984).

Lead can enter ambient water from atmospheric deposition and surface runoff, where it tends to form insoluble salts and precipitates. Concentrations of lead in US ambient waters are typically low. Mean values tend to be less than 3-28 μg/l (NAS 1980; EPA 1986b). In contrast to ambient water, levels in drinking water can be much higher (10-1,000 μg/l) because

of leaching of lead from lead pipe and leaded solder joints. Lead concentrations in drinking water vary with the amount of lead in the household plumbing and corrosiveness of the water. Soft or acidic waters tend to be more corrosive and promote higher concentrations of dissolved lead in the drinking water (Worth, et al., 1981). Drinking water can be a major source of lead intake for infants and young children who consume large amounts of infant formula prepared with household water.

IV. Routes of Exposure, Absorption, Distribution, Transport, and Degradation

Oral intake, rather than inhalation, is generally the predominant route of intake for humans. Intake occurs through ingestion of food and beverages, and in infants and children, through ingestion of dust and soil.

Ingestion of lead-based paint is one of the most frequent causes of severe lead intoxication in children (Chisolm 1984). Although the US Consumer Product Safety Commission banned the use of household paints containing greater than 0.06% lead in 1977, the hazard persists in homes and apartments constructed before the ban. Infants and children can be exposed to lead in paint from ingesting and inhaling house dust contaminated with paint dust and from intentionally ingesting paint chips (paint pica). Exposure can occur outside the house from ingestion of street and soil dust.

Absorption of ingested lead is quantitatively the most significant route of uptake of inorganic lead in most human populations; the exception is occupational exposures in which inhalation of airborne lead results in significant uptake. Percutaneous absorption (ie., dermal uptake) is not considered a significant route of absorption of inorganic lead.

Alkyl lead compounds (e.g., triethyl, trimethyl, tetraethyl and tetramethyl lead) are more highly lipophilic than inorganic lead and are readily absorbed from the lung and skin.

For inhalation, amounts and patterns of deposition of particulate aerosols in the respiratory tract are affected by the size of the inhaled particles, age-related factors that determine breathing patterns (e.g., nose breathing vs. mouth breathing), airway geometry and airstream velocity within the respiratory tract. Estimates for fractional absorption of large particles (greater than 2.5 um) deposited in the upper respiratory tract range from 40-50% (Kehoe, 196la,b,c; Chamberlain and Heard, 1981).

Chamberlain, et al., (1978) exposed adult human subjects to radioactive lead in engine exhaust, lead oxide or lead nitrate (less than 1 um particle size) and observed that 90% of the deposited lead was cleared from the lung within 14 days. Morrow, et al., (1980) reported 50% absorption of deposited lead inhaled as lead chloride or lead hydroxide within 14 hours. An analysis of the radioisotopic dilution studies of Rabinowitz, et al., (1977) in which adult human subjects were exposed daily to ambient air lead indicated that about 90% of the deposited lead was absorbed daily (EPA 1986b).

Quantitative analyses of the relationship between aerosol particle size and deposition in the human respiratory tract have been combined with information on size distributions of ambient air lead aerosols to estimate deposition and absorption efficiencies for inhaled lead in adults and children (EPA 1986b; Cohen 1987). It was estimated that 38% of the inhaled lead in adults living in the vicinity of an industrial source is absorbed. For some urban and rural atmospheres, where submicron particles dominate the airborne lead mass, the estimated fractional absorption is 15-30% (Cohen, 1987).

The retention and absorption of gaseous tetraethyl and tetramethyl lead has been examined in volunteers who inhaled radioactively-labelled tetraalkyl lead (Heard, et al., 1979). Initial lung retention was 37 and 51% for tetraethyl and tetramethyl lead, respectively. Of these amounts, 40% of tetraethyl lead and 20% of tetramethyl lead was exhaled within 48 hours; the remaining fraction (tetraethyl, 60%; tetramethyl, 80%) was absorbed.

The gastrointestinal tract is the primary site of absorption of lead in children and most adult populations, with the exception of those subject to occupational exposure (EPA 1986b). Gastrointestinal absorption of lead varies with age, diet and nutritional status as well as the chemical species and particle size of the ingested lead. Dietary balance studies have yielded estimates ranging from 7-15% for gastrointestinal absorption in adults (Kehoe, 1961 a,b,c; Chamberlain, et al., 1978; Rabinowitz, et al., 1980). Gastrointestinal absorption of dietary lead is greater in infants and children than in adults. A mass balance study in infants aged 2 weeks to 2 years yielded estimates of 42% for children with dietary intakes of greater than or equal to 5 μg PB/kg body weight. Lower dietary intakes were associated with highly variable absorption (Ziegler, et al., 1978). A study conducted with infants and children aged 2 months to 8 years (daily intake, 10 μg Pb/kg body weight) yielded estimates of 53% for gastrointestinal absorption (Alexander, et al., 1973). Individuals with poor nutritional status may absorb more lead from environmental sources (EPA, 1986b).

Inorganic lead is not readily absorbed through the skin (percutaneous absorption). Values of 0-0.3% of the administered dose were reported for humans exposed to dermal applications of cosmetic preparations containing lead acetate.

Mineralized tissues (eg., bone and teeth) are the single largest pool for absorbed lead, accounting for about 95% of the total lead burden in adults and slightly less in children (Barry 1975, 1981). Lead not contained in mineralized tissue is distributed in soft tissues, primarily blood, liver and kidneys. Small amounts accumulated in other soft tissues such as brain, although not quantitatively significant to the overall distribution of the body burden, are of considerable toxicological importance. Lead readily transfers across the placenta and distributes to fetal tissues (Horiuchi, et al., 1959; Barltrop, 1959; Lauwerys, et al., 1978; Kovar, et al., 1984; Tsuchiya, et al., 1984; Korpela, et al., 1986). Lead distributes to a variety of tissues after exposure to lead alkyls. Levels of lead in humans that have been exposed to tetraethyl and tetramethyl lead are highest in liver followed by kidney and brain (Bolanowska, et al., 1967; Grandjean and Nielsen 1979).

Metabolism of inorganic lead consists primarily of reversible ligand reactions including the formation of complexes with amino acids and nonprotein thiols and binding to various complexes with amino acids and nonprotein thiols and binding of various cellular proteins (Bruenger, et al., 1973; Raghavan and Gonick, 1977; Everson and Patterson 1980; Ong and Lee 1980; DeSilva 1981). Tetraethyl and tetramethyl lead undergo oxidative dealkylation to the corresponding trialkyl derivatives that are thought to be the neurotoxic forms of these compounds. Dealkylation of tetralkyl lead occurs in a variety of species, including humans (EPA 1986b).

V. Acute Toxicity

Acute lead-induced nephrotoxicity is characterized by proximal tubular nephropathy of the kidney. Characteristic lesions described in both humans and animals include nuclear inclusion bodies and mitochondrial changes in the epithelial cells of the pars recta of the proximal tubule and impaired solute reabsorption (eg., glucose, amino acids, phosphate) of the kidney. Acute nephrotoxicity has been observed in children with lead encephalopathy and is associated with relatively high blood lead levels (ie., greater than 80 μg/dl) (Chisolm, et al., 1955; Chisolm 1962, 1968; Pueschel, et al., 1972; EPA 1986b).

VI. Chronic Toxicity

Lead Neurotoxicity in adults: Severe lead neurotoxicity is characterized by overt symptoms of irritability, shortening of attention span, headache, muscular tremor, peripheral neuropathy, abdominal pain, loss of memory and hallucinations. Delirium, convulsions, paralysis and death can also occur. In adults, some of these overt symptoms may become apparent at blood lead levels in the range of 40-60 μg/dl (EPA 1986b). Nonovert symptoms of neurotoxicity associated with lead exposure in adults include impaired performance on psychomotor tests, decreased nerve conduction velocity and impaired cognitive function. Blood lead levels associated with these effects range upwards from 30 μg/dl (EPA 1986b).

Lead Neurotoxicity in Children: Symptoms of overt neurotoxicity in children are similar to those observed in adults. Nonovert symptoms of neurotoxicity that have been reported in children include impairments or abnormalities in psychomotor and cognitive function. Severe psychomotor and cognitive deficits appear to be associated with blood lead levels at the range of greater than or equal to 40-60 μg/l in "high-risk" populations of children (EPA 1986b). High risk populations include children with previous histories of lead encephalopathy or paint pica and children with possible occupational exposure (eg., lead pottery manufacture).

Several large-scale studies (EPA 1986b) reported effects on mental development and cognitive ability associated with blood lead levels greater than or equal to 10-15 μg/dl.

An inverse linear association between Stanford-Binet IQ scores and contemporary blood lead levels was seen over the entire range of 6-47 μg/dl in a study of uniformly low

socioeconomic status black children, 3-7 years old (Hawk, et al., 1986; Schroeder and Hawk, 1987). A study of 6-9-year old children in Edinburgh, Scotland, also indicated a negative linear correlation between blood lead and scores on tests of cognitive ability (Fulton, et al., 1987). The correlation extended across a range of 5-22 μg/dl mean blood lead levels.

A nerve conduction velocity study in children (aged 5-9 years) living in the vicinity of a lead smelter (Schwartz, et al., 1988) indicated a threshold for decreased maximal nerve conduction and estimated it to be within the range of 20-30 μg/dl.

Effects of Lead on Heme Biosynthesis and Erythropoiesis: Lead interferes with heme biosynthesis by decreasing the activity of enzymes in this pathway (EPA 1986b). Significant impairment of hemoglobin synthesis occurs in adults only at relatively high blood levels. The threshold for a decrease in blood hemoglobin in adults and children is achieved at a blood lead level of 50 μg/dl (Meredith, et al., 1977; Fischbein, 1977; Alvares, et al., 1975). Frank anemia in adults has been associated with levels greater than 80 μg/dl (Tola, et al., 1973; Grandjean 1979; Lilis, et al., 1978; Wada, et al., 1973; Baker, et al., 1979). Available information indicates the potential for undesirable effects on heme biosynthesis and erythroblast pyrimidine metabolism in children with blood lead levels greater than 10-15 μg/dl and possibly at lower levels (EPA 1990).

Effects of Lead on the Kidney: Chronic toxicity in the kidney is characterized by interstitial fibrosis and decreased glomerular filtration rate (Goyer 1982; EPA 1986b, ATSDR/EPA 1988). Chronic nephropathy, indicated by nuclear inclusion bodies, mitochondrial changes, interstitial fibrosis and glomerular changes, have been associated with prolonged (greater than 10 years) occupational exposures and blood lead levels greater than 40-60 μg/dl (Lilis, et al., 1968; Cramer, et al., 1974; Biagini, et al., 1977; Wedeen, et al., 1975, 1979; Buchet, et al., 1980; Hong, et al., 1980).

Effects of Lead on Blood Pressure: The relationship between concurrent blood lead levels and blood pressure in adults has been examined in several epidemiological studies (Pocock, et al.,1984, 1985, 1988; Harlan, et al., 1985; Pirkle, et al., 1985; Landis and Flegal 1987; Elwood, et al., 1988 a, b; Neri, et al., 1988; Sharp, et al., 1988; Weiss, et al., 1988; Moreau, et al., 1988). The weight of evidence provided by the several large- and small-scale epidemiology studies supports the existence of a positive correlation between blood lead level and blood pressure. In addition, the results of numerous animal studies support a dose-response relationship between lead exposure and elevated blood pressure. Chronic exposure to inorganic lead increases blood pressure in laboratory animals (Victery 1988). The correlation between blood lead levels and blood pressure in humans appears to extend to blood lead levels less than 20 μg/dl and possibly to as low as 7 μg/dl. This suggests that as blood lead level increases greater than 7 μg/dl to levels greater than or equal to 20 μg/dl, the risk for increased blood pressure increases.

Effects of Lead on Serum Vitamin D Levels: Serum levels of 1,25-dihydroxycholecalciferol are inversely correlated with blood lead in children (Rosen, et al., 1980; Mahaffey, et al., 1982). The correlation persists when examined across a range of blood lead levels extending from 12-60 μg/dl; however, the dose-effect relationship has not been characterized. Based on a linear regression analysis of data on serum 1,25-dihydroxycholecalciferol and blood lead levels in children as well as data on 1,25-dihydroxycholecalciferol levels in other vitamin D related clinical disorders in children, it has been predicted that increasing the blood lead levels from 12-60 μg/dl will lower serum 1,25-dihydroxycholecalciferol to clinically adverse levels (Mahaffey, et al., 1982). Chronic depression of 1,25-dihydroxycholecalciferol levels of a much smaller magnitude than that associated with frank clinical disorders of calcium and phosphate metabolism have the potential to alter bone development and growth in children; therefore, blood lead levels greater than 12 μg/dl should be considered potentially undesirable with respect to changes in 1,25-dihydroxycholecalciferol levels in children. 1,25-25 dihydroxy-cholecalciferol, the active form of vitamin D, is a hormone that plays an important role in the regulation of gastrointestinal absorption and renal excretion of calcium and phosphorus and in the mineralization of bone. Deficiencies in 1,25-dihydroxy-cholecalciferol are associated with decreased bone mineralization and clinical syndrome of rickets in children. 1,25-dihydroxy-cholecalciferol may also stimulate gastrointestinal absorption of lead (Smith, et al., 1978).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity:

Structural chromosomal aberrations and increased sister chromatid exchanges in peripheral lymphocytes have been associated with chronic exposure to lead resulting in blood lead levels in the range of 24-89 μg/dl, although effects were not observed over this range of blood levels in numerous studies (EPA 1986b). Studies reviewed by EPA (1989b) demonstrated that lead compounds induce cell transformation in mouse cells and rat embryo cells infected with the Rauscher murine leukemia virus.

Carcinogenicity:

Epidemiological studies of industrial workers, where the potential for lead exposure is usually greater than for a "normal population," have been conducted to evaluate the role of lead in the induction of human neoplasia (Cooper 1976, 1981; Cooper and Gaffey 1975; Chrusciel 1975; Dingwall-Fordyce and Lane 1963; Lane 1964, McMichael and Johnson 1982; Neal, et al., 1941; Nelson, et al., 1982). Two studies (Dingwall-Fordyce and Lane 1963 and Nelson, et al., 1982) did not find any association between exposure and cancer mortality. Selevan, et al., (1984), in their retrospective cohort mortality study of primary lead smelter workers, found a slight decrease in the total cancer mortality compared to controls. Apparent excesses were observed for respiratory cancer and kidney cancer. Cooper and Gaffey (1975) and Cooper (1985 update) performed a cohort mortality study of battery plant workers and lead smelter workers. They found statistically significant excesses for total cancer mortality, stomach cancer and lung

cancer in the battery plant workers. Although similar excesses were observed in the smelter workers, they were not statistically significant. Cooper and Gaffey (1975) felt it was possible that individual subjects were monitored primarily on the basis of obvious signs of lead exposure, while others who showed no symptoms of lead poisoning were not monitored.

In general, these studies made no attempt to consider types of lead compounds to which workers were exposed or to determine probable routes of exposure as well as information on the possible contribution from smoking. All studies also included exposures to other metals such as arsenic, cadmium (both known carcinogens) and zinc for which no adjustment was done. The cancer excesses observed in the lung and stomach were relatively small. There was no consistency of site among the various studies and no study showed any dose-response relationship. Thus, the available human evidence is considered to be inadequate to refute or demonstrate any potential carcinogenicity for humans from lead exposure.

In animals, the carcinogenic potential of lead salts (primarily phosphates and acetates) administered via the oral route or by injection has been demonstrated in rats and mice by more than 10 investigators (Zollinger 1953; Boyland, et al., 1962; Van Esch, et al., 1962; Baldwin, et al., 1964; Balo, et al., 1965; Hass, et al., 1967; Mao and Molnar 1967; Epstein and Mantel 1968; Zawirska and Medras 1968; Van Esch and Kroes 1969; Zawirska and Medras 1972; Azar, et al., 1973; Furst, et al., 1976; Koller, et al., 1985). The most characteristic cancer response is bilateral renal carcinoma. Rats given lead acetate or subacetate orally have developed gliomas and lead subacetate also produced lung adenomas in mice after intraperitoneal administration. Most of these investigations found a carcinogenic response only at the highest tolerated doses. The lead compounds tested in animals are almost all soluble salts. Metallic lead, lead oxide and lead tetralkyls have not been tested adequately. Studies of inhalation exposure have not been located in the literature (EPA 1993).

Teratogenicity (and other reproductive effects):

Severe occupational exposure to lead has been associated with increased incidence of spontaneous abortion (EPA 1986b) in exposed women. In the Port Pirie cohort study, pregnancy outcome in populations near and distant from a lead smelter indicated that the risk for pre-term delivery was positively related to maternal blood lead, over a range of 8-32 μg/dl (McMichael, et al., 1986). Depressed sperm production and development has been associated with occupational exposure to lead. Based on studies by Lancranjan, et al., 1975 and Wildt, et al., 1983, the EPA concluded that undesirable effects on sperm or testes may occur in men as a result of chronic exposures leading to blood lead levels of 40-50 μg/dl (EPA 1986b).

The effects of prenatal and neonatal lead exposure on perinatal status and postnatal mental and motor development have been examined in several epidemiologic studies. Four prospective studies initiated in the cities of Boston, Cincinnati, Cleveland and Port Pirie, Australia, are notable (Bellinger, et al., 1987 a,b, 1989; Dietrich, et al., 1987, 1989; Ernhart, et al., 1986; McMichael, et al., 1986; Vimpani, et al.,1985; Baghurst, et al., 1987). Based on an

extensive evaluation of these studies, the EPA concluded that "all of these studies taken together suggest that neurobehavioral deficits, including declines in Bayley Mental Development Index (MDI) scores and other assessments of neurobehavioral function, are associated with prenatal blood lead exposure levels on the order of 10-15 μg/dl, and possibly even lower, as indexed by maternal or cord blood lead concentrations" (EPA 1986b).

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

The EPA has classified lead as a probable human carcinogen (Class B2). This classification is based on the observation of increased kidney cancer in rats and mice, and on increases in tumors in rats.

EPA Dose-Response Parameters (IRIS, 1994):

Carcinogenic Effects:

The EPA has not derived an oral and inhalation cancer potency factors for lead (EPA 1993).

Noncarcinogenic Effects:

Dose-response estimates for oral and inhalation exposures are not available for the noncarcinogenic effects of lead (IRIS, 1994). An old RfD for non-cancer effects is available for the non-cancer impacts of lead exposure on humans. It is 1.0E-03 mg/kg-day. However, the EPA has determined that an RfD would not be appropriate to protect children from adverse developmental impacts of lead exposure due to the complex relationship between lead exposures by various routes, blood-lead levels, and the occurrence of adverse effects. Instead, EPA had developed a biokinetic model for assessing the impacts of multi-route lead exposures on childrens' blood-lead levels (EPA 1990b) and recommends that it be used to evaluate the health significance of lead exposures, using a target blood lead level of 10 μg/dl as an indicator of potential adverse effects. This model applies to infants and young children (0 to 6 years old).

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B.15 MANGANESE

I. Occurrence and Use

Manganese is an essential element present in all living organisms. Background levels of manganese in soil range from approximately 300 to 950 mg/kg (Kabata and Kabata-Pendias, 1987).

Manganese is used in the manufacture of ceramics, matches, glass dyes, welding rods, and is a component of steel, steel alloys, cast iron, and super- and nonferris alloys. The metal is also used as a chemical intermediate in the production of high purity salts, and acts as a purifying and scavenging agent in metal production (HSDB, 1993).

II. Physical and Chemical Properties of Manganese

Molecular Weight	55.00[1]
Water Solubility, mg/l	0.0E+00[2]
Bioaccumulation Factor for Fish	4.0E+02[3]
Bioaccumulation Factor for Shellfish	9.0E+04[3]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Weast, 1979
[3]Napier et al., 1980

III. Environmental Fate and Transport

In the atmosphere, manganese solubilizes in rainwater at acidic pHs (85% solubilization at pH 2). The release of dissolved labile forms of manganese, and probably other metals, through such dissolution reactions is potentially and important step in the geochemical cycles of these elements. Studies of sediment core samples suggest that manganese compounds in sediments concentrate in the upper 2 cm of the core, and do not produce a vertical gradient of concentrations. This suggests that manganese compounds are bioavailable to aquatic organisms (HSDB, 1993).

IV. Routes of Exposure, Distribution, Absorption, Transport, and Degradation

Exposure to anthropogenic sources of manganese primarily occurs in the workplace via inhalation. Manganese releases to the ambient air are contributed by coal burning, fuel oil burning steelmaking, and battery manufacture. Natural concentrations in food account for oral manganese intakes ranging from 2 to 9 mg/day. The efficiencies with which manganese and

its compounds are absorbed following exposure via the inhalation, ingestion, and dermal routes have been reported to equal approximately 70.0, 3.0, and 0.0 percent respectively. The principal tissues and organs to which manganese and its compounds are distributed have been reported to include mitochondria of the pancreas, liver kidneys, intestine, and brain. The half-life of manganese in the human body is about 37 days, although manganese crossing the blood-brain barrier exhibits a longer half-time within the brain. The principal route of elimination is in the feces (ATSDR, 1989. Toxicological Profile for Manganese).

V. Acute Toxicity

Acute toxic effects associated with manganese compounds can occur in humans under unusual, and unusually intense exposure scenarios, such as intentional or accidental poisoning, which differs qualitatively and quantitatively from human environmental exposures. Acute toxic effects attributable to manganese may follow inhalation of its dusts, usually in mining or manufacturing facilities. The resulting condition, which is typically reversible, is known as manganese pneumonitis, characterized by epithelial necrosis followed by mononuclear cell proliferation. In humans, acute manganese poisoning is rare. Manganese, among other metals, may also cause a reversible condition known as metal fume fever following exposure to freshly formed metal oxide fumes of respirable particle size. Symptoms include fever, chills, sweating, nausea and coughing (ATSDR, 1989).

VI. Chronic Toxicity

Chronic effects occurring from inhalation exposure, generally over a period of 2 years, include central nervous system (CNS) toxicity. Those who develop manganese poisoning (manganism) exhibit a psychiatric disorder characterized by irritability, difficulty in walking, speech disturbances, and compulsive behavior which may include running, fighting, and/or singing. If the condition persists, a mask-like face, retropulsion or propulsion and a Parkinson-like syndrome develop (Mena et al., 1967). The major effect of manganese encephalopathy has been classified as severe selective damage to the subthalamic nucleus and palladium (Pentschew et al., 1963). In addition to these CNS effects, liver cirrhosis often occurs. Victims of chronic manganese toxicity tend to recover slowly, even after cessation of exposure (Klaassen et al., 1986).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity:

Available information relating to the potential mutagenicity of manganese is highly limited. Manganese has been reported to be mutagenic at high concentrations in some but not all in vitro bioassays by which it has been tested (ATSDR, 1989).

Carcinogenicity:

It is uncertain whether manganese is or is not carcinogenic. In one study, manganese chloride administration was associated with lymphosarcomas in 67 percent of treated mice, compared with 24 percent of controls. In another study, intraperitoneal injections of manganous sulfate at 660 mg/kg into mice over a period of 30 weeks resulted in a slight elevation of ling tumor incidence. Based upon presently available evidence, it is inappropriate to evaluate manganese as a human carcinogen (ATSDR, 1989).

Teratogenicity (and other reproductive effects):

Although deficiencies of manganese may result in developmental defects, it is uncertain whether excessive manganese can exert teratogenic effects (ATSDR, 1989).

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

Manganese is not classifiable as to human carcinogenicity (Group D), based on insufficient data for both human and animal studies.

EPA Dose-Response Parameters (IRIS, 1994):

Carcinogenic Effects:

No carcinogenic dose-response parameters have been promulgated for manganese.

Noncarcinogenic Effects:

ORAL RFD SUMMARY:

ORAL RFD (WATER): 5E-3 mg/kg-day
CRITICAL EFFECT/TARGET ORGAN: CNS effects

ORAL RFD (FOOD): 1.4E-1 mg/kg-day
CRITICAL EFFECT/TARGET ORGAN: No observed adverse effects

*Conversion Factors:
Assuming a water consumption of 2 L/day and a body weight of 70 kg, is equivalent to 0.005 mg/kg-day. The water RfD assumes a separate dietary intake of manganese, as this essential element is found in varying amounts in all diets.

ORAL RFD UNCERTAINTY : UF = 1

The information used to determine the RfD for manganese in food was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects. Humans exert an efficient homeostatic control over manganese such that body burdens are kept constant with variations in diet. It is recognized that manganese is an essential element, being required for normal human growth and maintenance of health. It has also been suggested that children are less susceptible to manganese intoxication and may require slightly higher levels of manganese than do adults. The available information providing a chronic NOAEL in many cross-sections of human populations, taken in conjunction with the essentiality of manganese warrants an uncertainty factor of 1.

ORAL RFD MODIFYING FACTOR : MF = 1

The modifying factors are 1 for both the dietary RfD and the drinking water RfD.

ORAL RFD CONFIDENCE :
Study -- Not applicable
Data Base -- medium
RfD -- medium

Many studies have reported similar findings with regard to the normal dietary intake of manganese by humans. These data are considered to be superior to any data obtained from animal toxicity studies, especially as the physiologic requirements for manganese vary quite a bit among different species, with man requiring less than rodents (Schroeder et al., 1966). There is no one study used to derive the dietary RfD for manganese. While several studies have determined average levels of manganese in various diets, no information is available to indicate toxic levels of manganese in the diet of humans. Because of the homeostatic control humans maintain over manganese, it is not considered to be very toxic when ingested with the diet. Confidence in the data base is medium and confidence in the dietary RfD for manganese is also medium.

It is again emphasized that this oral RfD is based on a total dietary intake of manganese and is not intended to be applied directly to drinking water situation. Because of the greater bioavailability of manganese from water, a separate RfD for water is proposed. This is based on the Greek epidemiologic study by Kondakis et al. (1989). This study has several strengths in that it examined a sensitive subpopulation of humans exposed to varying concentrations of manganese in the drinking water for a lifetime. Although the actual manganese content in the diet was not measured in the study, the author did indicate that the people in the three areas were age-matched, had similar social, economic and educational backgrounds and the food consumed by these subjects were purchased at the marketplace and were not expected to vary much in manganese content. The confidence in the critical study can be considered low-to-medium. Confidence in the data base can also be considered medium-to-low. The Greek study is supported by the more severe effects occurring at higher

levels in the Japanese study (Kawamura et al., 1941) and the study in rhesus monkeys (Gupta et al., 1980). Overall confidence in the drinking water RfD can be considered medium-to-low.

INHALATION RFD SUMMARY:
INHALATION RfD: 5E-5 mg/cu.m

CRITICAL EFFECT/TARGET ORGAN: Impairment of neurobehavioral function.

INHALATION RFD UNCERTAINTY:

An uncertainty factor of 1000 reflects 10 to protect sensitive individuals, 10 for use of a LOAEL, and 10 for database limitations reflecting both the less-than-chronic periods of exposure and the lack of developmental data, as well as potential but unquantified differences in the toxicity of different forms of Mn.

INHALATION RFD MODIFYING:
MF -- None

INHALATION RFD CONFIDENCE:
Study -- Medium
Data Base -- Medium
RfC -- Medium

Confidence in the principal studies (Roels et al., 1987, 1992) is medium. Neither of the principal studies identified a NOAEL for neurobehavioral effects, nor did either study directly measure particle size or provide information on the particle size distribution. The 1992 study by Roels et al. did provide respirable and total dust measurements, but the 1987 study measured only total dust. These limitations of the studies are mitigated by the fact that the principal studies found similar indications of neurobehavioral dysfunction, and these findings were consistent with the results of other human studies (Mergler et al., 1993; Iregren, 1990; Wennberg et al., 1991, 1992; as well as various clinical studies). In addition, the exposure history of the workers in the 1992 study by Roels et al. was well characterized and essentially had not changed over the preceding 15 years, thereby allowing calculation of integrated exposure levels for individual workers. However, individual integrated exposures were not established in the 1987 study of Roels et al. data base is medium. The duration of exposure was relatively limited in all of the principal and supporting studies, ranging from means of 5.3 and 7.1 years in the co-principal studies by Roels et al. (1992 and 1987, respectively) to a maximum of 16.7 years in the study by Mergler et al. (1993). Moreover, the workers were relatively young, ranging from means of 31.3 and 34.3 years in the co-principal studies (Roels et al., 1992 and 1987, respectively) to a maximum of 46.4 years (Iregren, 1990). These temporal limitations raise concerns that longer durations of exposure and/or interactions with aging might result in the detection of effects at lower concentrations, as suggested by results from studies involving longer exposure durations and lower concentrations (Mergler et al.,

1993; Iregren, 1990). In addition, except for the 1992 study by Roels et al., in which Mn exposure was limited to MnO2, the other principal and supporting studies did not specify the species of Mn and the proportions of the different compounds of Mn to which workers were exposed. It is not clear whether certain compounds or oxidation states of Mn are more toxic than others. Thus, it is not possible to distinguish the relative toxicity of different Mn compounds in these studies, despite some indications in the literature regarding the differential toxicity of various oxidation states of Mn. Although the primary neurotoxicological effects of exposure to airborne Mn have been qualitatively well characterized by the general consistency of effects across studies, the exposure-effect relationship remains to be well quantified, and a no-effect level for neurotoxicity has not been identified in any of these studies thus far. Finally, the effects of Mn on development and reproduction have not been studied adequately. Insufficient information on the developmental toxicity of Mn by inhalation exposure exists, and the same is true for information on female reproductive function. The study of the reproductive toxicity of inhaled Mn in males also needs to be characterized more fully. Reflecting medium confidence in the principal studies and medium confidence in the data base, confidence in the inhalation RfC is medium.

IX. References

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B.16 MERCURY

I. Occurrence and Use

Mercury (Hg) heavy metal which can exist in three forms: elemental, inorganic and organic compounds There are both natural and anthropogenic sources of mercury, including normal degassing of the earth's crust, mining, smelting, industrial discharge, paper pulp industries, pesticides and the burning of fossil fuels. As much as one third of atmospheric mercury may be due to industrial release of organic and inorganic forms.

II. Physical and Chemical Properties of Elemental Mercury

Molecular Weight	201.00[1]
Water Solubility, mg/l	0.0E+00[2]
Vapor Pressure, mm Hg	2.0E-03[3]
Bioaccumulation Factor for Fish	2.0E+05[4]
Bioaccumulation Factor for Shellfish	2.0E+05[4]

Source: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Weast, 1979
[3]EPA, 1981
[4]Napier et al., 1980

III. Environmental Fate and Transport

Methyl mercury which is formed in sediments following industrial discharge of mercury has been shown to cause serious deleterious effects to both human health and the environment because of its ability to bioaccumulate. Methyl mercury is stable in the aquatic environment and is taken up by fish in the food chain, which may eventually result in exposure to humans. A very famous outbreak of methyl mercury poisoning occurred in the Minamata Bay area of Japan, where mercury containing effluent from a vinyl chloride production process emptied into the bay from a nearby factory, causing contamination of fish and shellfish. Humans and animals eating fish experienced central nervous system disorders characterized by degeneration and death of nerves in the focal areas of the cerebral cortex (i.e. the largest part of the brain), loss of vision, speech impairment, and deafness. In the United States, there is concern over contamination of Mercury in the region of the Great lakes.

IV. Routes of Exposure, Absorption, Distribution, Transport, and Degradation

Exposure from soil and water may either be to elemental, inorganic, or organic mercury compounds. For exposure to biota or sediments under reducing (oxygen deficient) conditions, exposure to the more toxic organic species are generally predominant. Principle routes of exposure include atmospheric via the inhalation of elemental mercury vapor, and ingestion of methyl mercury in water and food. In humans, elemental and inorganic mercury compounds are efficiently absorbed following inhalation exposure but poorly absorbed following oral exposure (EPA, 1984). Once absorbed, mercury is generally distributed about the body, binding to the sulfhydryl groups of many proteins. Mercury is excreted in the urine and feces. Small quantities go into the hair and other routes, including the exhaling of some elemental mercury (Carson et al., 1987).

V. Acute Toxicity

Acute mercury poisoning is usually caused by the soluble inorganic salts. Early signs and symptoms include pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhea, and shock. Later swelling of the salivary glands, stomatitis, loosening of the teeth, nephritis, anuria, and hepatitis occur. Death results from the effects of the gastrointestinal tract (ulcerations, bleeding, shock) and/or kidney (Carson et al., 1987).

VI. Chronic Toxicity

The occupational exposure of workers to elemental mercury vapors (0.1 to 0.2 mg/m^3) has been associated with mental disturbances, tremors and gingivitis (EPA, 1984). The central nervous system is a major target for organic mercury compounds. Adverse effects in humans from exposure to organic mercury compounds have included the destruction of cortical cerebral neurons, damage to Purkinje cells and lesions of the cerebellum. Clinical symptoms following exposure to organic mercury compounds have included paresthesia, loss of sensation in extremities, ataxia, and hearing and visual impairment (WHO, 1976). A primary target organ for inorganic mercurials is the kidney. Human exposure to inorganic mercury compounds has been associated with anuria, polyuria, proteinuria and renal lesions (Hammond and Beliles, 1980).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity:

Both organic and inorganic compounds are reported to be mutagenic in eukaryotic systems (Leonard et al., 1984).

Carcinogenicity:

There is no definitive evidence reported in the literature indicating that either organic or inorganic mercury is carcinogenic by the ingestion, inhalation, or dermal pathways (ATSDR,

1989, Toxicological Profile for Mercury, PB90-181256). One positive study involving the ingestion pathway has been reported. Dietary exposure of mice to 15 ppm of methyl mercury in their diet resulted in renal tumors in 13 of 16 males, but in no females, surviving after 53 weeks. Eleven of the tumors in males were classified as adenocarcinomas and two as adenomas (EPA, 1985, Drinking Water Criteria Document for Mercury. Cincinnati, Environmental Criteria and Assessment Office, ECAO-CIN-025, EPA-600/X-84-178-1).

Teratogenicity

Embryotoxic and teratogenic effects, including malformations of the skeletal and genitourinary systems, have been observed in the offspring of animals exposed to organic mercury (EPA, 1984).

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

Mercury is not classified as a human carcinogen (Group D), based on insufficient data in animals, and no human data.

EPA Dose-Response Parameters:

No carcinogenic or noncarcinogenic dose-response parameters are reported in IRIS for Mercury. HEAST (1993) reports an oral chronic and subchronic RfD of 3.0E-04 mg/kg/day, and an inhalation chronic and subchronic RfC of 3.0E-04 mg/cm^3.

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B.17 METHYLENE CHLORIDE

I. Background

Methylene chloride (dichloromethane; DCM) is a multipurpose solvent used in industry for a variety of purposes including paint removal, manufacture of photographic equipment, as an aerosol propellant, in the manufacture of insecticides and fumigants, and as solvents and cleaners (Proctor and others, 1988). Methylene chloride is also produced in chlorinated drinking water treatment (NAS, 1977). DCM is a volatile liquid with high lipid (fat) solubility and moderate water solubility. Background ambient air concentrations of methylene chloride in the United States generally fall between 30 to 50 ppt (parts per trillion) (Brodzinsky and Singh, 1983; ATSDR, 1987), while the highest ambient air levels were measured in New Jersey at 360 ppb (Pellizzari and Bunch, 1979). Concentrations in drinking water average around 1 µg/l up to a maximum of 3 µg/l (U.S. Environmental Protection Agency, 1985).

DCM is readily absorbed from the respiratory and gastrointestinal tracts and more slowly across the skin (ATSDR, 1987). Due to the volatile nature of DCM, the principal route of exposure for humans is via inhalation. The absorption efficiency following inhalation is estimated to be 70 to 75 percent (DiVincenzo and Kaplan, 1981). It does tend to bioaccumulate, but the uptake is saturable, with establishment of a steady-state condition where intake and elimination are equal (ATSDR, 1987). Elimination from the body occurs primarily through exhaled air as unchanged DCM, carbon dioxide or carbon monoxide (ATSDR, 1987).

II. Acute Toxicity

Acute exposures to high concentrations of methylene chloride via oral and inhalation routes can cause severe toxic effects to a variety of organs. The principal target organs of DCM exposure are the liver and central nervous system (CNS). Impaired CNS function in humans from acute exposures to DCM (300-800 ppm for 5 hours) produced visual and auditory dysfunctions and decreased ability in various psychomotor tasks (Fodor and Winneke, 1971). Several cases of accidental human fatalities have occurred from acute exposures to high concentrations of methylene chloride (Bonventre and others, 1977). In animal studies, liver toxicity was evident following high doses of DCM (>4,000 ppm). Specific liver alterations included fatty infiltration, glycogen depletion, altered cytochrome P-450 activity, and increased liver weights (ATSDR, 1987). In addition, kidney damage and cardiovascular effects have also been observed in animals following acute exposures.

III. Chronic Toxicity

As in acute exposures, the central nervous system, the cardiovascular system, and the liver are the principal target organs affected. Animal studies have shown an aging effect in liver cells,

fatty proliferation in the liver, and altered liver enzyme levels (ATSDR, 1987). However, liver toxicity has not been reported in human epidemiological studies. The greatest effect of chronic exposure in humans appears to be on the cardiovascular and the central nervous system (ATSDR, 1987). Cardiovascular symptoms of chronic DCM exposure include chest pain (burning sensations around the heart), feelings of chest pressure and palpitations (ATSDR, 1987). Neurotoxic symptoms following DCM exposure include impaired short-term memory, insomnia, narcosis, and auditory and visual hallucinations (Weiss, 1967; NIOSH, 1976). Other organs suspected to be affected include the kidney and respiratory tract, but evidence is lacking to confirm the occurrence of these effects in humans (ATSDR, 1987).

IV. Carcinogenicity

Two human epidemiological studies have been conducted on the carcinogenicity of methylene chloride. Friedlander and others (1978) examined the proportionate mortality of 344 workers exposed to air concentrations of DCM ranging between 30 and 125 ppm (time-weighted average) for up to 30 years. No significant increase in the cancer mortality rate of the exposed populations were observed relative to an unexposed control population. In another study of workers exposed to 26 ppm DCM for 22 years, no significant excess deaths from malignant neoplasms, respiratory cancer, or liver cancer were detected (Hearne and others, 1987; ATSDR, 1987). However, there was an increased incidence of deaths due to pancreatic cancer, although this was not statistically significant.

There is sufficient evidence that methylene chloride is carcinogenic in several animal species. In a two-year study conducted by the National Coffee Association (NCA, 1982 and 1983), rats and mice were administered up to 250 mg/kg DCM in drinking water. Female rats and male mice exhibited a statistically significant increase in the incidence of neoplastic nodules or hepatocellular (liver) carcinomas, while these measures were not significant in female mice and male rats (U.S. Environmental Protection Agency, 1989). Burek and others (1980 and 1984) reported an increased incidence of mammary and salivary gland tumors in both male and female rats exposed to high concentrations of DCM. In another study, rats exposed to airborne DCM (up to 4,000 ppm) had an increased incidence of mammary adenomas, while male mice were found to have an excess of hepatocellular (liver) and respiratory adenomas and carcinomas (NTP, 1986). The USEPA has reviewed the available carcinogenicity data and has categorized methylene chloride as a probable human carcinogen (Group B2) based on inadequate data in humans and sufficient evidence of carcinogenicity in animals (U.S. Environmental Protection Agency, 1991).

V. Mutagenicity

DCM has been shown to be mutagenic in the Ames assay (with and without metabolic activation) (ATSDR, 1987). DCM was positive at inducing chromosomal aberrations in mouse and human lymphocytes (Thilagar and others, 1984). Generally negative results have

been obtained for sister chromatid exchange, point mutation, and DNA damage and repair in cultured mammalian cells (ATSDR, 1987).

VI. Reproductive/Developmental Effects

No studies are available on the reproductive or developmental effects of DCM in exposed humans. Two animal studies were conducted on reproductive effects of DCM; both studies reported negative results (Nitschke and others, 1985; Bornmann and Loesser, 1967). There is limited animal data available which indicates that DCM is a developmental toxin when inhaled at high concentrations (1,250 ppm) (ATSDR, 1987). The significance of these results is not clear, however, since the reported effects were not significantly different from controls, the studies used only one dose, and the exposure concentrations caused maternal toxicity (which in turn may have caused fetal toxicity) (ATSDR, 1987).

VII. Sensitive Populations

Approximately 1 million workers employed in industries such as DCM manufacturing, paint remover formulation, and metal degreasing are at an elevated risk compared to the general population (ATSDR, 1987). Additionally, individuals living near industries producing or utilizing DCM in large quantities are at risk of occasional atmospheric exposure. Individuals with a weakened cardiovascular system may be more sensitive to DCM exposure since low levels of DCM may enhance the severity of existing cardiovascular disease (ATSDR, 1987).

VIII. Chemical Interactions

No information is available on potential interactions with other chemicals.

IX. Dose-Response Parameter Estimates

The dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects:

The Cancer Assessment Group (CAG) of the USEPA has derived an oral cancer potency estimate of 7.5×10^{-3} $(mg/kg/day)^{-1}$ (U.S. Environmental Protection Agency, 1991a). This estimate is based on the arithmetic mean of the potency slope factors determined for hepatocellular adenomas and carcinomas in mice from lifetime inhalation exposure studies conducted by the National Toxicology Program (NTP, 1986) and the National Coffee Association (NCA, 1983), as discussed above. The CAG has also derived an inhalation cancer potency estimate of 1.4×10^{-2} $(mg/kg/day)^{-1}$ based on the combined incidence of adenomas and carcinomas of the liver or lung from the NTP study (NTP, 1986).

Oral Cancer Potency Estimates: 7.5×10^{-3} $(mg/kg/day)^{-1}$ (U.S. Environmental Protection Agency, 1991a).

Inhalation Cancer Potency Estimate: 1.4×10^{-2} $(mg/kg/day)^{-1}$ (U.S. Environmental Protection Agency, 1991a).

Noncarcinogenic Effects:

The USEPA has computed a chronic oral reference dose (RfD) of 6×10^{-2} mg/kg/day for methylene chloride (U.S. Environmental Protection Agency, 1991a) based on a two-year drinking water study with rats, which identified a NOAEL (no-observed-adverse-effect-level) of 6 mg/kg/day (NCA, 1982). Higher doses produced histological alterations of the liver. An uncertainty factor of 100 was incorporated to account for uncertainties in extrapolating animal data to humans (10) and to account for sensitive human subgroups (10) (U.S. Environmental Protection Agency, 1991a). An inhalation RfD of 3 mg/m^3 (U.S. Environmental Protection Agency, 1991b) was derived for methylene chloride by the USEPA.

Oral RfD: 6×10^{-2} mg/kg/day (U.S. Environmental Protection Agency, 1991a).

Inhalation RfD: 3 mg/m^3 (U.S. Environmental Protection Agency, 1991b).

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B.18 NICKEL

I. Occurrence and Use

Nickel (Ni) is a naturally occurring metal in the earth's crust and known to exist in at least 4 valence states. The water soluble nickel salts (+2 state) are the most common form, while other nickel compounds include nickel carbonyl, nickel subsulfide and Nickel dust. Typical levels of nickel in soils range from approximately 6 to 60 ppm (Kabata-Pendias and Pendias, 1986).

Nickel (Ni) is an important element used for electroplating coatings for turbine blades, helicopter rotors, extrusion dyes, coinage, ceramics, storage vessels, batteries, and electronic circuits as well and as in the production of steel and many other alloys.

II. Physical and Chemical Properties of Nickel

Molecular Weight	59.00[1]
Water Solubility, mg/l	0.0E+00[2]
Vapor Pressure, mm Hg	0.0E+00[3]
Bioaccumulation Factor for Fish	1.0E+02[4]
Bioaccumulation Factor for Shellfish	1.0E+02[4]

Source: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Weast, 1979
[3]Callahan et al., 1979
[4]Napier et al., 1980

III. Environmental Fate and Transport

IV. Routes of Exposure, Absorption, Distribution, Transport, and Degradation

The major source of human exposure is in the workplace by inhalation of dust and fumes and skin contact, but it can also affect the general populations by ingestion of contaminated food stuffs and drinking water, usually in the form of nickel salts.

Nickel compounds can be absorbed following inhalation, ingestion. Dermal absorption results in allergic reactions only at the sites of skin contact. The amount absorbed depends on the dose administered and on the chemical and physical form of the particular nickel compound (EPA, 1986). The principal tissues and organs to which nickel and its compounds are

distributed have been reported to include kidneys, pituitary glands, lings, skin, adrenal glands, ovaries, and testes. Major routes of excretion have been studied in rats, which indicate that the principal route of elimination is the urine. Other routes of excretion include the bile, sweat, hair and mother's milk (ATSDR, 1989).

V. Acute Toxicity

Noncarcinogenic effects of nickel exposure include nausea, fever, lung inflammation and respiratory failure following acute incidences, as well as contact dermatitis (skin rashes). Adverse effects associated with acute exposure in animals have included depressed weight gain, altered hematological parameters, and increased iron deposition in the blood, heart, liver and testes (EPA, 1987).

VI. Chronic Toxicity

Chronic ingestion of nickel-containing foods increases the risk of developing skin rashes. Studies performed in animals to estimate the long-term effects of nickel exposure showed a decrease in body and organ weights of rats (may be indicative of disease), as well as a decrease in their appetite.

Chronic or subchronic exposures of experimental animals to nickel salts have been associated with reduced weight gain, degenerative lesions of the male reproductive tract, asthma, nasal septal perforations, rhinitis, sinusitis, hyperglycemia, decreased prolactin levels, decreased iodine uptake, and vasoconstriction of the coronary vessels (Clayton and Clayton, 1981).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity:

Results of mutagenicity assays suggest that nickel is mutagenic. Nickel carbonate caused DNA strand breaks, and reduced the fidelity of DNA replication. In mammalian cells, nickel chloride produced mutations in Chinese hamster V79 and ovary cells, as well as in mouse lymphoma cells. At least ten investigations of nickel causation of chromosomal aberrations have been conducted, with four positive results in cells of mice, hamsters, and humans, and six negative studies in cells of humans, mice and rats (ATSDR, 1989).

Carcinogenicity:

It has been known for over 40 years that inhalation of nickel is associated with the development of lung, nasal and respiratory cancer. However, an evaluation of the carcinogenicity soluble salts of nickel, which are possible contaminants of soil, water, food, has not been performed.

Inhalation exposure of experimental animals to nickel carbonyl or nickel subsulfide induces pulmonary tumors (EPA, 1986). Several nickel salts cause localized tumors when administered by subcutaneous injection or implantation. Epidemiological evidence indicates that inhalation of nickel refinery dust and nickel subsulfide is associated with cancers of the nasal cavity, lung, larynx, kidney and prostate (EPA, 1986).

Teratogenicity:

Nickel can cross the placental barrier, but there is no definitive evidence of teratogenicity. Nickel carbonyl was teratogenic in rats; i.v. doses of $NiCl_2$ (1 to 6.9 mg/kg) on single days 7 through 11 was teratogenic in mice (Carson et al., 1987).

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

Nickel refinery dust and nickel subsulfide (the compound used in this risk assessment) for conservatism by inhalation are both categorized in Group A - Human Carcinogens.

EPA Dose-Response Parameters (IRIS, 1994):

ORAL RFD SUMMARY :
ORAL RFD : 0.02 mg/kg/day
CRITICAL EFFECT : Decreased body and organ weights in rats

ORAL RFD UNCERTAINTY:

UF = 300. An uncertainty factor of 10 is used for interspecies extrapolation and 10 to protect sensitive populations. An additional uncertainty factor of 3 is used to account for inadequacies in the reproductive studies (RTI, 1987; Ambrose et al., 1976; Smith et al., 1990) (see Additional Comments section). During the gestation and postnatal development of F1b litters in the RTI (1987) study, temperatures were about 10 degrees F higher than normal at certain times, which makes evaluation of this part of the reproductive study impossible. In the Ambrose et al. (1976) study, statistical design limitations included small sample size and use of pups rather than litters as the unit for comparison. There were also problems with the statistical analysis of the Smith et al. (1990) study.

ORAL RFD MODIFYING FACTOR:
MF = 1.

ORAL RFD CONFIDENCE:
Study: Low

Data Base: Medium
RfD: Medium

The chronic study (Ambrose et al., 1976) was properly designed and provided adequate toxicological endpoints; however, high mortality occurred in the controls (44/50). Therefore, a low confidence is recommended for the study. The data base provided adequate supporting subchronic studies, one by gavage and the other in drinking water (Po animals of the RTI subchronic study, 1986). A medium confidence level in the data base is recommended since there are inadequacies in the remaining reproduction data.

INHALATION RfD SUMMARY:

No inhalation RfD is available for nickel from IRIS (1994).

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B.19 THALLIUM

I. Occurrence and Use

Thallium is widely distributed in rock formations and soils containing potassium feldspars and micas. Its crustal abundance is about 1 mg/kg (Kazantzis, 1986). Thallium is also found in potash, in association with lead and zinc in fossil fuels. Air levels reported from Nebraska ranged from 0.04 to 0.48 ng/m^3 (Kazantzis, 1986). Concentrations of 0.7 to 88 mg/l have been reported in river water draining a metal mining area in New Brunswick (Kazantzis, 1986). Contamination of drinking water may occur in the vicinity of copper, zinc, and cadmium mining, smelting, and refining operations. Typical dietary intake of thallium in humans has been estimated at about 2 mg/day, based on the sparse data available (Kazantzis 1986).

Thallous sulfate was used on a large scale as a rodenticide, but has been replaced with less toxic substances. Seventy percent of the thallium produced currently is used in the production of photoelectric cells, lamps, semiconductors, and scintillation counters (Kazantzis 1986).

II. Physical and Chemical Properties of Thallium

Molecular Weight	204.38
Solubility mg/l in water	00E+00
Bioaccumulation Factor for Fish	
Bioaccumulation Factor for Shellfish	

Source: TOMES DATABASE

III. Environmental Fate and Transport

Thallium is released into the atmosphere from industrial operations such as coal-fired power plants, smelting operations, and cement factories. Following release, thallium can either be inhaled, or settle from the atmosphere and contaminate surface water or soil. Because plants take up thallium, the primary nonoccupational sources of thallium exposure are through the consumption of fruits and vegetables grown in contaminated soil and through the use of tobacco products (ATSDR 1991). Although thallium is normally detected in the urine of humans (<2.0 µg/l), it is not considered an essential element and no known metabolic functions have been described (Hui 1983; Goyer 1986; Tietz 1986).

IV. Routes of Exposure, Absorption, Distribution, Transport, and Degradation

Soluble thallium is readily absorbed through the gastrointestinal tract. Thallium is somewhat unique among the metals in that it can penetrate the skin and produce serious poisonings (Carson et al. 1986). Following its absorption, thallium uptake into the circulatory system is rapid. Thallium is quickly distributed from the blood to the tissues. The primary routes of thallium excretion for animals and humans are the urine and feces. Thallium is also deposited into hair and nails of both humans and animals, and these are considered important sources of elimination. Other minor routes of thallium elimination include tears, saliva, and milk (Prick et al. 1955; Richelmi et al. 1980). Highest concentrations following exposure are found in the kidney and urine with lesser amounts in the intestines, thyroid, testes, pancreas, skin, bone, and spleen (Hammond and Beliles, 1980).

V. Acute Toxicity

Symptoms associated with acute poisoning in humans include gastrointestinal irritation, liver and kidney damage, pulmonary edema, degeneration of the adrenals, peripheral system, and central nervous system disorders, and severe ocular effects (Carson et al. 1986). In mammals, toxic effects of a high acute dose are delayed for 12 hours to 2 days, while neurological symptoms may not appear for 2 to 5 days (Carson et al. 1986). The estimated lethal dose for humans is 8 to 12 mg/kg (USEPA 1985; Hammond and Beliles, 1980).

VI. Chronic Toxicity

In chronic poisoning, the most striking feature is hair loss (alopecia). Although this does not always occur even at high exposures, it is usually associated with daily intakes of 10 to 20 mg/day (Carson et al. 1986). This characteristic phenomenon of thallium poisoning is probably the result of cessation of cell proliferation in the fair follicles (Kazantzis 1986). Human chronic exposures have been characterized by fatty infiltration and necrosis of the liver, nephritis (kidney inflammation), gastroenteritis, pulmonary edema, degenerative changes in the adrenals, degeneration of peripheral and central nervous system, alopecia, and in severe cases, death (Hammond and Beliles 1980). These cases are usually the result of food contamination or the use of thallium as a depilatory (hair remover).

In experimental studies with rats, chronic thallium poisoning results in hair loss, cataracts, and hind leg paralysis (Hammond and Beliles 1980). Renal (kidney) lesions were observed at necropsy with histologic changes noted in the proximal and distal kidney tubules (Hammond and Beliles 1980). Additionally, degenerative changes were noted in liver and kidney mitochondria.

VII. Mutagenicity, Carcinogenicity, Teratogenicity

Mutagenicity

Very little data is available, but thallium salts have shown a marked antimitotic activity on mammalian, avian and plant cells. Thallium actively induces chromosome breaks in pea plants (Carson et al. 1986).

Carcinogenicity

Thallium was reported to be carcinogenic in an early animal study where chronic oral and cutaneous (skin) doses of thallium salts in mice produced tissue degeneration, papillomas, precancerous lesions, and cancers of the female genital tract (Carson et al. 1986). Experimental details and the incidence of lesions and mortality were not reported. Recent review literature have not identified thallium as carcinogenic. Also, the EPA has not evaluated the carcinogenicity of thallium (USEPA 1990).

Teratogenicity

Carson et al. (1986) indicated that the reproductive effects of thallium have been judged to be no worse than other general cellular toxins. However, thallium does exert an alteration on fetal development and fetal mortality (Carson et al. 1986). Offspring of female rats fed 2.5 mg/kg/day of thallium sulfate showed poor bone development (Carson et al. 1986).

VIII. EPA Carcinogenic Classification and Dose-Response Estimates

Carcinogenic Effects

There is insufficient evidence to classify thallium as a carcinogen. The USEPA has not evaluated the carcinogenicity of thallium; therefore, no dose-response estimates have been computed (USEPA 1990).

Noncarcinogenic Effects

The USEP has derived an oral reference dose (RfD) of 8.0×10^{-5} mg/kg/day based upon a 90-day gavage study involving rats administered thallium sulfate at doses of 0, 0.1, 0.05 and 0.25 mg/kg/day (USEPA 1990). A NOAEL (no-observed-adverse-effect-level) of 0.25 mg/kg/day was identified in this study. Higher doses produced increased SPGOT (blood enzymes indicative of liver damage) and alopecia. An uncertainty factor of 3,000 was incorporated to account for uncertainties in extrapolating animal data to humans (factor of 10), to address sensitive human subgroups (factor of 10), to extrapolate from a subchronic to a chronic exposure (factor of 10), and an additional factor of three was incorporated to compensate for the insufficiency of available reproductive and chronic toxicity data (USEPA 1990).

An inhalation RfD appears to be currently unavailable for thallium; therefore the noncarcinogenic effects of inhaled thallium were not quantitatively evaluated in this assessment.

Oral Reference Dose: 8.0E-05 (mg/kg/day) (USEPA 1992).

Dose-Response Parameter Estimation

The dose-response parameter estimates of carcinogens and noncarcinogens are computed differently by he USEPA; therefore, these estimates are presented separately below.

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B.20 TOLUENE

I. Occurrence and Use

Toluene is a flammable aromatic hydrocarbon commercially produced by the petrochemical industry. It is used in the manufacture of dyes, saccharin, perfumes, caprolactam, pharmaceuticals, detergents and trinitrotoluene (TNT) (Fawell and Hunt, 1988). It is also used as a solvent and is a component in automobile and aviation fuel (Fawell and Hunt, 1988). Atmospheric concentrations of toluene have been linked with automobile exhaust emissions in a number of studies (Fawell and Hunt, 1988). Toluene has been identified in several surveys of raw and treated water in the U.S. at average concentrations of less than 1 mg/l (Fawell and Hunt, 1988). Levels in surface waters were generally less than 1 mg/l, but up to 5 mg/l were found in some groundwaters (Fawell and Hunt, 1988).

II. Physical and Chemical Properties of Toluene

Molecular Weight	92.15[1]
Water Solubility, mg/l	5.4E+02[2]
Vapor Pressure, mm Hg	2.8E+01[2]
Henry's Law Constant, atm-m^3/mole	6.4E-03[3]
Octanol-Water Partition Coefficient	5.4E+02[2]
Organic Carbon Partition Coefficient, L/g	3.0E+02[4]
Bioaccumulation Factor for Fish	7.0E+01[1]
Bioaccumulation Factor for Shellfish	1.2E+01[1]

Sources: [1] HSDB,1989
[2] USEPA, 1984
[3] ICF, 1987
[4] Mabey, et al., 1982

III. Environmental Fate and Transport

Toluene is released into the atmosphere principally from the volatilization of petroleum fuels and toluene-based solvents and thinners and from motor vehicle exhaust. Considerable amounts are discharged into waterways or spilled on land during the storage,transport and disposal of fuels and oils. If toluene is released to soil, it will be lost by evaporation from near-surface soil and by leaching to the groundwater. Biodegradation occurs both in soil and groundwater, but it is apt to be slow especially at high concentrations, which may be toxic to microorganisms. The presence of acclimated microbial populations may allow rapid biodegradation. It will not significantly hydrolyze in soil or water under normal environmental conditions. If toluene is released into water, its concentration will decrease due to evaporation and biodegradation. This removal can be rapid or take several weeks, depending on temperature, mixing conditions, and acclimation of microorganisms. It will not significantly

adsorb to sediment or bioconcentrate in aquatic organisms. If toluene is released to the atmosphere, it will degrade by reaction with photochemically produced hydroxyl radicals (half-life 3 hr to slightly over 1 day) or be washed out in rain. It will not be subject to direct photolysis (HSDB, 1993).

IV. Pharmacokinetics (Routes of Exposure, Distribution, Absorption, Transport, and Degradation)

The primary source of human exposure is from inhalation of contaminated ambient air, especially in traffic or near filling stations, or in occupational atmospheres where toluene-based solvents are used (HSDB, 1993).

The most common route of exposure in humans is by inhalation. In human subjects, pulmonary absorption of toluene was 57 percent after exposure to 430 m/m^3 for one hour and declined to a steady-state level of 37 percent of the dose after 2-4 hours (Nomiyama and Nomiyama, 1974). Several factors are known to affect pulmonary absorption rates such as ventilatory rate, particle size, solubility and for organics, body fat. Animal studies indicate near complete (approximately 100 percent) absorption of toluene from the gastrointestinal tract (USEPA, 1985). Toluene is also absorbed across the skin. Once absorbed, toluene would be expected to distribute to lipid or adipose tissue due to its low solubility and lipophilicity. Studies in rats indicate rapid distribution with tissues reaching maximum levels within hours of dosing. Toluene was distributed in highest concentrations to the liver, brain and blood of mice in several studies (USEPA, 1989). Toluene is rapidly hydroxylated by the mixed function oxidase system to benzyl alcohol. Further metabolism results in the formation of hippuric acid or the glucuronide conjugate of benzoic acid, both of which are eliminated in urine and account for 85-95 percent of an absorbed dose (hippuric acid accounts for 75 percent) (USEPA, 1985). Toluene is also eliminated unchanged in exhaled air. Elimination is rapid with no evidence of bioaccumulation seen in daily samples of occupationally exposed individuals (USEPA, 1985).

V. Acute Toxicity

Acute exposure to toluene results in central nervous system (CNS) depression and membrane irritation, though sudden deaths due to cardiac arrhythmias have been reported in some cases of toluene abuse (toluene and toluene-containing industrial solvents are often abused socially for the intoxication effect) (Fawell and Hunt, 1988). There is little evidence that toluene is acutely toxic to organs other than the CNS, although effects on the lungs, liver, and kidney have been reported following acute inhalation exposures (USEPA, 1989). However, toluene is not considered to be very toxic. The oral LD_{50} in rats is 7.0 g/kg (Fawell and Hunt, 1988).

VI. Chronic Toxicity

Occupational exposures at concentrations of 300 ppm have resulted in CNS abnormalities (Fawell and Hunt, 1988). For example, exposed female shoemakers showed abnormal tendon reflexes, reduced grasping power, and decreased finger agility, while car painters showed impairment of concentration, reduced emotional reactivity and reduced hand agility (Fawell and Hunt, 1988). Studies of habitual "glue sniffers" abusing toluene-containing glues have reported symptoms consistent with those of occupationally exposed subjects and experimental animals. Associated neurological disorders include cerebellar degeneration, encephalopathy (brain degeneration), personality changes, slurred speech, clumsiness, and memory and concentration disturbances (Fawell and Hunt, 1988). Results of animal studies show no organ damage after exposure to very high doses of toluene (Fawell and Hunt, 1988). Rodents exposed to 12,000 ppm of toluene in 3 hour cycles 5 times per week for 8 weeks exhibited inebriation but no lung, liver, or kidney damage (Fawell and Hunt, 1988). In a two-year study of rats exposed to 30, 100, or 300 ppm of toluene, a dose-related reduction in hematocrit values (packed cell volume) was observed in females, but not males (USEPA, 1989a). No other effects were observed.

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity

Chromosomal studies of toluene-exposed workers have produced equivocal results for mutagenicity. Workers exposed to toluene for up to 15 years displayed no significant increase in lymphocyte chromosomal aberrations (Fawell and Hunt, 1988). However, a study of workers in chemical laboratories and a photoprinting factory did indicate a significant increase in the frequency of abnormal lymphocytes and chromosome breaks (Fawell and Hunt, 1988). Toluene has been found to be nonmutagenic in reverse mutation assays with S. typhimurium and other mutagenic assays.

Carcinogenicity

Since human exposure in the workplace occurs often and because toluene is an abused substance, many reports on human exposure exist in the literature. None of these reports, however, associates toluene exposure with an increased rate or incidence of cancer. Likewise, carcinogenicity due to toluene exposure has not been observed in animal studies (USEPA, 1984).

No significant increases in tumor incidence was found in a two-year inhalation study of 120 rats exposed to 0, 30, 100, or 300 ppm of toluene for 6 hours per day, 5 days per week (Fawell and Hunt, 1988). Also, toluene applied to the skin of mice 3 times per week for a lifetime produced no carcinogenic effects (Fawell and Hunt, 1988). Toluene has not been tested for carcinogenicity by the oral route (Fawell and Hunt, 1988). Based on inadequate

evidence in animals and lack of human data, the Carcinogen Assessment Group of the EPA has designated toluene as a Group D carcinogen (not classifiable as to human carcinogenicity) (USEPA, 1993).

Teratogenicity (and other reproductive effects)

Increased embryonic mortality was apparent in mice administered toluene by gavage on days 6-15 of gestation at doses of 0.3, 0.5 or 1.0 ml/kg (Fawell and Hunt, 1988). Also, decreased fetal weight was also noted at 0.5 and 1.0 ml/kg, and a significantly increased incidence of cleft palate was found at the highest dose.

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification

There is insufficient evidence to classify toluene as a carcinogen. It is currently classified by the EPA as a Group D carcinogen (not classified) (USEPA, 1993).

EPA Dose-Response Parameters

INHALATION RfD: 1.1E-1 mg/kg/day
INHALATION RfC: 4E-1 mg/cu.m

CRITICAL EFFECT/TARGET ORGAN : Neurological effects , degeneration of nasal epithelium
INHALATION RfD UNCERTAINTY
UF = 300. An uncertainty factor of 10 is used to account for intraspecies variability and another factor of 10 for the use of a LOAEL. An additional factor of 3 is applied for data base deficiencies, including the lack of data and well-characterized laboratory animal exposures evaluating neurotoxicity and respiratory irritation.

Inhalation RfD Modifying Factor: MF -- None (=1)

INHALATION RfD CONFIDENCE
Study -- Medium
Data Base -- Medium
RfC -- Medium

The study of Foo et al. (1990) indicates adverse neurological effects of toluene in a small worker population. These effects are consistent with more severe CNS effects occurring at abusive concentrations of toluene and could not have been confounded by alcohol as the control and exposed populations did not use alcohol. However, the paucity of exposure information and identification of only a LOAEL is not sufficient to warrant a higher

confidence than medium for this study. Other studies indicate that irritation may occur at around the same concentration, 100 ppm (Baelum et al., 1985; Echeverria et al., 1989). In regard to this effect, the NTP (1989) rat chronic inhalation study was well conducted, established the rat as the most sensitive species, examined an adequate number of animals, and performed histopathology on all major organs, including the brain and the respiratory tract. The sensitive endpoint was the concentration-dependent degeneration of the nasal epithelium characterized by the erosion of the olfactory epithelium and degeneration of the respiratory epithelium in male rats. The NTP study is also given medium confidence, however, as it did not establish a NOAEL. Although this data base has a complement of chronic laboratory animal studies, long-term data in humans are not available for either the neurotoxicity or irritation endpoints. The reproductive/developmental studies in three species were not comprehensive in endpoint evaluation but do identify the rabbit as the most sensitive species. The data base is thus given a medium confidence rating. A medium confidence rating for the RfC follows.

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B.21 TOTAL PETROLEUM HYDROCARBONS

General Background

I. Occurrence and Use

Crude oil of petroleum is comprised of a variety of carbon and hydrogen containing compounds referred to as "petroleum hydrocarbons." Different petroleum products are derived from the crude oil via a series of distillations, which serves to separate the hydrocarbons into groups according to their boiling points. The light distillates have boiling ranges between approximately 50°C and 225°C (CRCS, Inc., 1985), and consist of the lightest hydrocarbon compounds in the size range of C_4 to C_{11}. Gasoline is the most common example of a light petroleum distillate. The middle distillates have a boiling range which is between approximately 190°C and 340°C, and consist generally of longer chain and aromatic compounds and comprise diesel, kerosene, and jet fuels. Heavy distillates such as motor oils and greases (i.e., residual fuels), have a boiling range between 101°C and 588°C, and a size ranging from C_{15} to C_{50}. Distillates of crude oil also may contain small quantities of lead, chromium, cadmium, chlorinated solvents and PCBs.

Types of petroleum products manufactured and/or processed from crude oil at Shaler/JTC include naphtha, white oil (white petroleum or mineral oil), yellow differential lubricant, waxes, and gasoline (i.e., light distillates), as well as middle distillates such as kerosene, motor oils, aviation fuels and oils (fuel oil #6), and sulfur-based high point gear grease. This profile discusses the general toxicologic properties of light, middle and heavy distillates of crude oil, and includes information about specific hydrocarbon mixtures when available.

The main product which was manufactured at the Shaler/JTC site was mineral spirits (petroleum spirits, white spirits of Stoddard solvents), which are derived from the light distillate fractions during the crude oil refining process and are composed of the C_6 - C_{11} compounds, with the majority of the relative mass composed of C_9 - C_{11}.

II. Physical/Chemical Properties

The actual composition for any fuel will vary depending on the source the crude oil, refinery processes used, and product specifications. Light distillates (including gasoline) are comprised of benzene, toluene, and xylene. Physical and chemical properties of these components are listed in individual profiles for each chemical. Middle distillates are comprised of naphthalene benzo(a)anthracene, benzo(a)pyrene, cresols and phenols.

Physical and chemical properties of these components are listed in individual profiles for each chemical.

Mineral spirits are composed of the following general classes of compounds, 50% paraffins (alkanes), 40% cycloparaffins (cyclic alkanes), and 10% aromatics. These percentages may vary for different formulations of mineral spirits (e.g., Stoddard solvents). Values for several of the physical and chemical properties on mineral spirits are listed below (Kostecki and Calabrese, 1993):

Solubility, mg/l 45.88

Vapor Pressure, mm Hg 2.53

Henry's Law Constant, atm-m^3/mol 1.03E-2

Octanol/water partition coefficient 3.24E+3

General properties of the middle and heavy distillates of petroleum hydrocarbons are listed below:

Middle distillates (Norman, 1991)

Solubility, mg/l 10-100

Vapor Pressure, mm Hg 1-10

Vapor Pressure, Kerosene, mm Hg 90

Octanol/water partition coefficient 1.0E+03-3.2E+04

Heavy Distillates (Norman, 1991)

Solubility, mg/l 1-20

Vapor Pressure, mm Hg 1

Octanol/water partition coefficient 1.0E+03-1E+06

III. Environmental Fate and Transport

Chemical, physical, and biological process can influence the chemical fate of a TPH mixture. Chemical processes include dissolution, hydrolysis, photolysis, and oxidation-reduction reactions. Physical processes include advection, volatilization, dispersion, diffusion, and sorption to soil and sediments. Biological processes include biodegradation by naturally occurring microorganisms, and bioconcentration, bioaccumulation, and biomagnification in organisms. These processes are all dependent on site-specific conditions, such as the physical

characteristics of the environmental media, the properties of the chemicals in the mixture, and other factors such a temperature, pH, and humidity (Kostecki and Calabrese, 1993).

Because TPH mixtures are complex mixtures of various organic (and sometimes inorganic) compounds, each individual component of the mixture is important in dictating the fate of the total mixture. In general, light distillates contain constituents with higher water solubility and volatility and lower sorption potential than middle and heavy distillates. Scientific investigations suggest that light distillates such as gasoline migrate readily in soil, potentially threatening or impacting groundwater beneath the source area. Light-fraction hydrocarbons (e.g., BTEX) compounds) can migrate toward the groundwater without leaving a significant amount of residual product in the unsaturated zone. These constituents are generally highly soluble in water, and do not readily sorb to soils; consequently, these compounds are often found in groundwater beneath impacted areas (Kostecki and Calabrese, 1993).

In contrast, TPH mixtures with heavier molecular weight constituents, such as diesel fuel, are generally more persistent in soils, due to their relatively low water solubility, volatility, and high sorption capacity for soil (especially clays and silty soils with a high mineral content). For example, PAHs containing more than 10 carbons which are environmentally important constituents of motor oil, generally tend to range from moderately immobile to immobile in the environment. Because of their low water solubility and high density (i.e., greater than that of water), soil and sediments often serve as sinks of these chemicals in the environment (Kostecki and Calabrese, 1993).

In general, the middle distillates (kerosene, diesel and jet fuels) will probably be distributed among all environmental compartments. Some uncombusted fuel may be emitted to the atmosphere and the resultant disposed hydrocarbons that persist may fall or wash out, possibly bound to particulates. Hydrocarbons bound to soil particulates may enter groundwater or rivers as land runoff (Oviatt et al., 1982 in CRCs, 1985). In the aquatic environment, middle distillates may be photo-oxidized or removed by biodegradation (Gearing and Gearing, 1982 in CRCS, 1985). Approximately 50% of the hydrocarbons will enter the sediment. Biodegradation may also occur in sediment although some hydrocarbons may be persistent in sediments on the order of years. Both light and middle distillates undergo photo-oxidation in the atmosphere, having half-lives on the order of , 1 to 10 days. (Mandry and Kenley, 1979 in CRCS, 1985).

According to Paterson and Mackay (1988), there are two general pathways for the migration of oil and oil constituents following an environmental release. The first involves bulk flow, as a result of infiltration under the influence of gravity and capillary action. Both the light and heavy hydrocarbons will migrate downward in the unsaturated soil due to these forces following a release. After a period of time, the chemicals will reach equilibrium in the soil column, and mobility of the remaining constituents is significantly reduced. Once the volatile fraction of the mixture is released from the soil, and the soluble fraction has leached downward (toward groundwater), the constituents remaining in the soil generally include

relatively heavy hydrocarbons such as PAHs and long-chained normal and branched alkanes. These chemicals are then subject to additional fate processes, governed by the second migration pathway involving diffusion,, dissolution, and abiotic or biotic transformations, which further reduce the concentrations of these chemicals over time (Kostecki and Calabrese, 1993).

Limited information is available in the literature regarding the fate and transport of mineral spirits, but contamination of soil and groundwater in the vicinity of the Shaler/JTC site is a concern. Kostecki and Calabrese (1993) suggest that the fate and transport of this contaminate should be evaluated as a mixture using site-specific information as well as the physical and chemical properties listed in the previous section of this profile. This approach is suggested due to the fact that there is a lack of information on the chemical makeup of the type of mineral spirit which was manufactured, and it is therefore not possible to identify an indicator chemical in the mixture which would serve to estimate the maximum migration of the mineral spirit plume.

IV. Routes of Exposure, Distribution, Absorption, Transport, and Degradation

Inhaiation, oral ingestion, and dermal contact are the most common routes of exposure. Workers are encouraged to wear proper protective clothing to prevent exposure to petroleum hydrocarbons products.

V. Non-Carcinogenic Health Effects

Acute Exposure

Humans: Light distillates of crude oil manufactured at Shaler/JTC (i.e., mineral oil), are not considered to be acutely toxic. Middle and heavy distillates are moderate to severe skin and eye irritants. Acute exposures to gasoline and jet fuels in humans generally produces CNS depression, headache, nausea, drowsiness, dizziness, insomnia, confusion, and tremors.

Laboratory animals: No information is available.

Chronic Exposure

Humans: Little is known about the chronic toxicity of the light distillates manufactured at Shaler/JTC (i.e., mineral oil). Chronic exposure to middle and heavy distillates have been associated with neurological effects, bronchopneumonia, toxicity to the liver, kidneys, hematopoietic system, lymphoid system, and testes.

Laboratory animals: No information is available.

VII. Carcinogenic Health Effects

Acute Exposure

Humans: Middle and heavy distillates are considered to be probable human carcinogens via the dermal route, including kerosene, aviation fuels, diesel fuels and motor oils. In general, the dermal carcinogenic potential of petroleum-derived products is related to the PAH content. Currently, the mechanism of dermal carcinogenesis and its implication in quantitative human health risk assessment are not well understood. Researchers have found that the dermal dose-response curves from different studies of one material have significantly different slopes. Also the reproducibility of dermal carcinogenesis studies is greatly affected by the application frequency, delivery vehicles, and other physical conditions. Epidemiological studies provided no conclusive evidence for carcinogenicity of diesel or jet fuels to humans. Skin painting assays in animals have reported positive results for some fuels, but this response is apparently due to epigenetic processes related to skin irritation, and there, not necessarily relevant to exposure by other routes. Oral or inhalation cancer bioassays were not located.

Laboratory animals: No information is available.

Chronic Exposure

Humans: No information is available.

Laboratory animals: No information is available.

VII. Mutagenicity, Ecotoxicity and Teratogenicity

Mutagenicity

Light distillates of crude are considered to be neither mutagenic, carcinogenic, or teratogenic.

Mutagenicity assays of kerosene and diesel produce negative results. Middle distillate fuels, as a class are weakly to moderately mutagenic

Ecotoxicity

Standardized methods for quantifying ecological hazards associated with exposure to total petroleum hydrocarbon mixtures have not yet been developed. In order to quantify hazards, the toxicity of the mixture must be known. TPH mixtures are highly variable in chemical content, and their content can change over time, complicating efforts to quantify toxicity.

The fate and transport of a mixture are important considerations in evaluation the potential environmental impacts from TPH contamination in the environment. Impacts to biota may

result if exposures to lower concentrations occur over an extended duration. The composition of the mixture, and the chemical and physical characteristics of the major components of the mixture, can by used to predict what environmental media may become impacted over tome. This information can then be used to predict the potential for environmental receptors to become exposed to the mixture at potentially toxic levels (Kostecki and Calabrese, 1993).

Biological fate and transport processes include biodegradation and the uptake of chemicals from biota and the potential transfer in the food chain (i.e., bioconcentration, bioaccumulation, and biomagnification). Certain bacteria present in soil and water are capable of utilizing TPH mixtures as a sole source of carbon for their survival. In fact, studies have shown that elevated concentrations of complex PAH mixtures can lead to localized population increases in microbes. The process of biodegradation can occur in both terrestrial and aquatic environments. Many hydrocarbon compounds have been shown to be ultimately metabolized by both animals and microorganisms to carbon dioxide and water. Lighter hydrocarbons are readily used by microbes, especially alkanes and aromatics. Heavier hydrocarbons (greater that 24 carbons) are much more difficult to degrade, and decay of these compounds occurs much more slowly in the environment. These persistent chemicals can remain in the environment for years, as shown by the half-life data for some common chemical constituents of TPH mixtures. Bioconcentration is defined as the tendency of a chemical to partition into the tissues of plants or animals. A bioconcentration factor (BCF) is often used as a measurement of this tendency, and is described as the ratio of the concentration of a chemical in biota to the concentration of the chemical in water. The BCF can be measured either directly in the laboratory or empirically from the octanol-water partition coefficient (K_{ow}). The BCF and the K_{ow} are often used a measures of the relative potential for a chemical to partition into environmental biota.

Concern over the potential hazards to terrestrial and aquatic wildlife from chemical uptake has increased recently. Currently, only a limited number of studies have provided insight into the complex factors influencing the uptake of chemicals into one species (bioconcentration), the transfer of chemicals to predator species from ingestion of contaminated prey (bioaccumulation), and the magnification of chemical concentrations as chemicals are transferred up the food chain (biomagnification). These factors are important to consider in evaluating potential impacts to environmental receptors from chemical exposures.

Many light hydrocarbons do not readily bioconcentrate in biota; however, as the molecular weight of the chemicals increases, the potential for bioconcentration also tends to increase. Several of the heavier hydrocarbons such as PAHs have been shown to bioconcentrated, bioaccumulate, of biomagnify in terrestrial of aquatic biota. Although a factor to define biomagnification has not been defined, the BCF or K_{ow} is generally used to define bioconcentration of chemicals in the environment.

Teratogenicity and other Reproductive effects

No information is available.

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

The EPA has not classified any of the petroleum hydrocarbon products manufactured at Shaler/JTC with respect to carcinogenicity. The only petroleum products which have currently been evaluated are gasoline, diesel, and jet fuels. Parameter for diesel and jet fuels are discussed below for the purpose of illustration and comparison to concentrations of site petroleum contaminants, but numerical RfDs and slope factors for these contaminants will not be used in the quantitative Shaler/JTC risk assessment. Gasoline parameters are not given in this profile do to the fact that they were derived for automotive gasoline, which was not manufactured at the site.

EPA Carcinogenic Classification:

Based on the available data, diesel fuel and the jet fuels can be assigned to USEPA weight-of-evidence group D; not classifiable as to human carcinogenicity.

Dose-Response Parameters

Interim RfDs have been calculated for diesel, and jet fuels by extrapolation from inhalation data.

For jet fuel #5, pure-bred beagle dogs, Fischer-344 rats, and C57BL mice were exposed continuously to 150 or 750 mg/m^3 of petroleum or shale-derived jet fuel #5 for 90 days (Gaworski et al., 1984; MacEwen and Vernot, 1985). An LOAEL of 150 mg/m^3 was identified based on hepatocellular fatty change and vacuolization in female mice. Although response rates were similar in both low- and high-dose groups, this is a mild, reversible lesion and use of this LOAEL equivalent oral dose (EOD) is affected by assuming equal absorption by the inhalation and oral routes and by using standard reference values (U.S. EPA, 1987b) for female C57BL/6 mouse body weight (0.0246 kg) and inhalation rate (0.040 m^3/day):

$$EOD = 150\ mg/m^3 \times (0.040\ m^3/day) \times (1/0.0246\ kg)$$

Applying the maximum uncertainty factor of 10,000 (reflecting five areas of uncertainty: variation within and between species, use of a LOAEL, extrapolation to chronic duration, and deficiencies in the database) produces a provisional oral RfD of $2x10^{-2}$ mg/kg/day for jet fuel #5.

Studies on jet fuel #4 and marine diesel fuel were conducted in a similar manner to the study on jet fuel #5. Refer to Pham-Mahini et al., 1992 for details.

Confidence in the critical studies (Gaworski et al., 1984; MacEwen and Vernot, 1985) is medium. These studies used adequate numbers of test animals from several species and included examination of a variety of endpoints, including hematology, blood chemistry, and histopathology, but they included only two dose levels, failed to identify NOAEL values, and were not generally well reported. Confidence in the data base is low because it was necessary to use inhalation studies and route-to-route extrapolation to calculate provisional RfDs for oral exposure, the inhalation studies used were of subchronic rather than chronic duration, and no studies of developmental or reproductive toxicity were available. Therefore, overall confidence in these provisional RfDs is low.

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B.22 VANADIUM

I. Occurrence and Use

Vanadium is a naturally occurring element in the earth's crust. Typical levels in pristine soils range from 60 to 110 mg/kg (Kabata-Pendias and Pendias, 1986). Vanadium also occurs naturally in fuel oils and coal. In the environment it is usually combined with other elements such as oxygen, sodium, sulfur, or chloride. The forms of vanadium most likely to be found at hazardous waste sites are not will known. One man-made form, vanadium oxide is most often used by industry, mostly in making steel. Much small amounts are used in making rubber, plastics, ceramics, and certain other chemicals (ATSDR, 1991).

II. Physical and Chemical Properties of Vanadium

Molecular Weight	50.94
Water Solubility, mg/l	0.0E+00
Bioaccumulation Factor for Fish	1.0E+01
Bioaccumulation Factor for Shellfish	3.0E+03

Source: Multi-Media Exposure Assessment Manual, 1989

III. Environmental Fate and Transport

The global biogeochemical cycling of vanadium is characterized by releases to the atmosphere, water, and land by natural and anthropogenic sources, long-range transportation of particles in both air and water, wet and dry deposition, adsorption, and complexing. Vanadium generally enters the atmosphere as an aerosol. Anthropogenic releases of vanadium to the atmosphere are in the form of simple or complex vanadium oxides. Vanadium transported within the atmosphere is eventually transferred to soil and water on the earth's surface by wet and dry deposition and dissolution in sea water. Eventually, in the course of biogeochemical movement between soil and water, these particulates are adsorbed to hydroxides or associated with organic compounds and are deposited on the sea bed. The most likely way for vanadium to get into the environment is when fuel oil is burned. Anthropogenic releases of vanadium to the air account for approximately two-thirds of all vanadium emissions (ATSDR, 1991).

IV. Routes of Exposure, Absorption, Distribution, Transport, and Degradation

The general population is exposed to background levels of vanadium primarily through ingestion of food. Workers in industries processing or using vanadium compounds are

commonly exposed to higher than background levels via the inhalation pathway. Exposure through inhalation is also of importance in urban cities burning large amounts of residual fuel oil. Other populations possibly exposed to higher than background levels include those ingesting foodstuffs contaminated by vanadium-enriched soil, fertilizers, or sludge. Population in the vicinity of vanadium-containing hazardous waste sites may also be exposed to higher than background levels (ATSDR, 1991).

Absorption of vanadium compounds through the lungs is estimated to be about 25 percent for soluble compounds, while ingested vanadium is more poorly absorbed, on the order of 2-3 percent (ICRP, 1960). The largest storage compartment in the body is fat, followed by, and to a lesser extent, bone and teeth (Goyer, 1986). The principal tissues and organs to which vanadium and its compounds are distributed have been reported to include fat, bone, teeth and lungs. Most absorbed vanadium is excreted in the urine within one day following long-term moderate exposure to the dust (ATSDR, 1991).

V. Acute Toxicity

Acute exposure of human volunteers to 0.1 to 1 mg/m3 of vanadium pentoxide stimulates mucous secretions and coughing (Carson et al., 1986). Acute vanadium exposures in animals generally produce effects on the nervous system, hemorrhage, paralysis, and respiratory depression (Goyer, 1986).

VI. Chronic Toxicity

A hypersensitivity reaction has been reported in individuals repeatedly exposed. Occupationally exposed individuals experienced respiratory tract irritation, dermal disorders, sneezing, sore throat, chest pain, and conjunctivitis (eye irritation) (Lagerkvist et al., 1986). Chronic exposure to high concentrations of airborne vanadium is believed to lead to chronic bronchitis, chronic rhinitis (nasal inflammation), and pharyngitis (inflammation of the pharynx) (Lagerkvist et al., 1986). The formation of allergy-like eczematous skin is associated with chronic respiratory exposures in humans and animals (NAS, 1977). Kiviluoto (1980) investigated radiographs and pulmonary function test results of exposed and unexposed workers and found that there was no difference between unexposed workers and those with long-term occupational exposure to vanadium. However, they did note that exposed workers complained more frequently of wheezing. In animals, fatty changes and partial necrosis of the liver was observed following long-term inhalation exposure to vanadium pentoxide, trioxide and chloride (Lagerkvist et al., 1986). There is no evidence of chronic oral toxicity (NAS, 1977).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity and Carcinogenicity:

There is no evidence that vanadium compounds are mutagenic, nor are they considered to be carcinogenic (Lagerkvist et al., 1986)

Teratogenicity:

Very little data is available on the reproductive and developmental effects of vanadium compounds. However, two reports have reported skeletal abnormalities in offspring of hamsters and mice injected with vanadate during mid-gestation (Carlton et al., 1982, Wide, 1984).

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

The EPA has not classified Vanadium in terms of carcinogenicity.

EPA Dose-Response Parameters:

There are no dose-response parameters for Vanadium reported in IRIS (1994). HEAST 1993 reports oral RfDs of 7.0E-03 mg/kg/day for vanadium, 9.0E-03 mg/kg/day for vanadium pentoxide, and 2.0E-02 mg/kg/day for vanadium sulfate.

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B.23 XYLENES

I. Occurrence and Use

Total xylenes, composed of ortho-, meta- and para-isomers, is an organic solvent used in paint thinners, and lacquers, as paint removers, in the paper coating industry, as solvents and emulsifiers for agricultural products, as fuel components, and commonly in the chemical industry as intermediates. Their ubiquitous occurrence in air and water is also a result of their presence in coal tars and tobacco smoke (HSBD, 1993).

II. Physical and Chemical Properties of Total Xylenes

Molecular Weight	106.17[1]
Water Solubility, mg/l	2.0E+02[2]
Vapor Pressure, mm Hg	1.0E+01[2]
Henry's Law Constant, atm-m^3/mole	7.0E-03[3]
Octanol-Water Partition Coefficient	1.8E+03[2]
Organic Carbon Partition Coefficient (L/g)	2.4E+02[1]
Bioaccumulation Factor for Fish	1.8E+02[1]
Bioaccumulation Factor for Shellfish	3.3E+01[1]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Dawson and others, 1980
[3]ICF-Clement Associates, 1987

III. Environmental Fate and Transport

Xylenes will enter the atmosphere primarily from fugitive emissions and exhaust connected with their use in gasoline. Industrial sources include emissions from petroleum refining, and their use as solvents and chemical intermediates. Discharges and spills on land and waterways result from their use in diesel fuel and gasoline; and storage and transport of petroleum products. Most of the xylenes are released into the atmosphere where they may photochemically degrade by reaction with hydroxyl radicals (half-life 1-18 hrs). The dominant removal process in water is volatilization. Xylenes are moderately mobile in soil and may leach into groundwater where they are known to persist for several years, despite some evidence that they biodegrade in both soil and groundwater. Xylenes have been shown to persist for up to 6 months in soil. The combined water solubility and organic partitioning indicate that xylene and its isomers will exhibit some degree of environmental mobility. Biodegradation is also an important fate process in both soils and the aquatic environment. Bioconcentration is not expected to be significant (Hazardous Substance Database, 1993).

IV. Routes of Exposure, Distribution, Absorption, Transport, and Degradation

The primary source of exposure is from air, especially at occupational sites where xylenes are used, and in areas with high traffic (Hazardous Substance Database, 1993).

Toxic effects of xylenes exposure ensue by ingestion, inhalation and skin contact. Data from animals and humans suggest that approximately 60 percent of an inhaled dose is absorbed (U.S. Environmental Protection Agency, 1985). In one human study, six men exposed to an industrial xylene mixture at concentrations of either 100 ppm and 200 ppm absorbed 60 percent of the amount of xylenes supplies to the lungs (Astrand and others, 1978). The concentration in alveolar air was relatively low throughout the entire exposure. In another study of humans voluntarily exposed to a commercial xylene mixture at levels of either 46 ppm or 92 ppm for eight hours, 64 percent of xylene isomers was absorbed (Toftgard and Gustafsson, 1980).

Inference from metabolism and excretion studies suggests that absorption of orally administered xylenes is nearly complete. Dermal absorption is reported to be minor following exposure to xylenes vapor but may be significant following contact with the liquid (U.S. Environmental Protection Agency, 1985).

V. Acute Toxicity

Inhalation causes depression of the central nervous system, upper respiratory irritation, and liver and kidney impairment. In humans, acute inhalation exposures to relatively high concentrations of xylenes adversely affects the central nervous system and lungs and can irritate mucous membranes (U.S. Environmental Protection Agency, 1987). Savolainen and others (Savolainen and others, 1980) observed that body balance and manual coordination were impaired in eight human male subjects following inhalation exposure to m-xylene. However, tolerance against the observed effects developed during one work week.

VI. Chronic Toxicity

Chronic effects of human xylene exposure include dryness of the eyes, nose and throat, dermatitis (skin disorders), central nervous system impairment, tremors, headaches, nausea, anemia and other blood disorders. A human inhalation study by Hake and others (Hake and others, 1981) demonstrated CNS effects and nose and throat irritation following inhalation exposures to 20 ppm, 7.5 hours/day for 5 days.

In experimental rats, long-term inhalation exposure to o-xylene resulted in hepatomegaly (Tatrai and others, 1981). Oral exposure to 200 mg/kg xylene in the diet for up to 6 months was also associated with liver toxicity, specifically development of intracellular vesicles (Bowers and others, 1982).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

Mutagenicity

Xylenes have been shown to be nonmutagenic in nearly all test systems. Xylenes have yielded negative results in the Ames assay using various strains of the bacterium Salmonella typhimurium, both with and without metabolic activation; and in bacterial DNA repair test systems. Xylenes have apparently not been associated with chromosomal aberration or sister chromatid exchange in mammalian cell either in vitro or in vivo (Agency for Toxic Substances and Disease Registry, 1989).

Carcinogenicity

Inadequate evidence exists regarding the carcinogenicity of xylenes. Epidemiological investigations of xylene carcinogenicity in humans was absent from the available literature. Evidence derived from bioassays involving laboratory animals has likewise revealed inadequate evidence (Agency for Toxic Substances and Disease Registry, 1989). In a National Toxicology Program study, male and female rats and mice were orally administered xylene for two years (National Toxicology Program, 1986). Histological data revealed an absence of significant changes in the incidence of treatment-related neoplastic and nonneoplastic lesions. Negative results have also been obtained in bioassays involving inhalation and dermal exposures.

Teratogenicity (and other reproductive effects)

No studies were located regarding reproductive effects in humans following inhalation exposure to mixed xylene or to xylene isomers (Agency for Toxic Substances and Disease Registry, 1989).

Total xylenes appear to be fetotoxic and may increase the incidence of visceral and skeletal malformations in offspring of exposed experimental animals (Mirkova and others, 1983).

VIII. USEPA Carcinogenic Classification and USEPA Dose-Response Parameters

USEPA Carcinogenic Classification

The USEPA (U.S. Environmental Protection Agency, 1993) classifies xylenes in Group D - Not Classifiable as to human carcinogenic potential due to inadequate evidence of carcinogenicity in animals

USEPA Dose-Response Parameters (U.S. Environmental Protection Agency, 1993)

ORAL RfD: 2E+0 mg/k$_{\ell}$ ..y

CRITICAL EFFECT/TARGET ORGAN: Hyperactivity, decreased body weight and increased mortality
ORAL RfD UNCERTAINTY : UF = 100.
An uncertainty factor of 100 was chosen: 10 for species-to-species extrapolation and 10 to protect sensitive individuals.

ORAL RfD MODIFYING FACTOR : MF = 1.

Oral RfD Confidence
Study: Medium
Data Base: Medium
RfD: Medium

The NTP (National Toxicology Program, 1986) study was given a medium confidence level because it was a well-designed study in which adequately sized groups of two species were tested over a substantial portion of their lifespan, comprehensive histology was performed, and a NOAEL was defined; but clinical chemistries, blood enzymes, and urinalysis were not performed. The data base was given a medium confidence level because, although supporting data exist for mice and teratogenicity and fetotoxicity data are available with positive results at high oral doses, a LOAEL for chronic oral exposure has not been defined. Medium confidence in the RfD follows.

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B.24 ZINC

I. Occurrence and Use

Zinc is an essential element (i.e., necessary for normal functioning of the human body) which is ubiquitous in the environment and is present in most food stuffs, water and air. Natural levels of zinc in soils range from 20 to 110 mg/kg (Kabata-Pendias and Pendias, 1986).

Zinc is used industrially in the manufacture of automotive components, builder's hardware, domestic appliances, industrial agricultural and commercial machinery, electric components, sporting goods and toys, scientific equipment and audio and television equipment (Clayton and Clayton, 1981).

II. Physical and Chemical Properties of Zinc

Molecular Weight	65.38[1]
Water Solubility, mg/l	NA
Bioaccumulation Factor for Fish	2.0E+03[2]
Bioaccumulation Factor for Shellfish	1.0E+04[2]

Sources: [1]Multi-Media Exposure Assessment Manual, 1989
[2]Napier et al., 1980

NA = Not available

III. Environmental Fate and Transport

The primary sources of zinc in the environment (air, water, and soil) are related to metallurgic wastes from smelter and refining operations. Releases to surface and groundwater are probably the greatest source of zinc in the ambient environment. Zinc is not volatilized to any significant extent, but is primarily deposited on sediments as a result of discharge from industrial operations and weathering processes. Zinc has a tendency to settle out of the water column and adsorb to sedimentary material. Severe zinc contamination thus tends to be confined to sources of emission. Small amounts of the metal (relative to water emissions are released to the atmosphere, primarily as a result of the handling of dry raw and concentrated ore at zinc production facilities and lead smelting factories. Zinc released to the atmosphere is often converted to a stable species such as zinc oxide. Most of the zinc released to the environment partitions to water, soil, and sediments (ATSDR, 1989).

The principle route of human exposure is by inhalation of zinc fumes in the workplace, and by ingestion of the metal in food and water by the general population. Zinc is efficiently absorbed in humans following oral exposure; however, insufficient data are available to evaluate absorption following inhalation exposure (ATSDR, 1989). Zinc can also enter the body via inhalation of zinc dust or fumes from zinc-smelting or -welding operations. The amount of zinc that passes directly through the skin is relatively small. The most important route of exposure near hazardous waste sites is likely to be through the drinking of zinc contaminated water. Normally, zinc is excreted in the urine and feces.

V. Acute Toxicity

Ionic zinc is poorly absorbed, but acidic salts containing zinc are corrosive to the skin and gastrointestinal tract. Ingestion of 2 grams or more of zinc produces toxic symptoms in humans. Zinc sulfate in these amounts irritates the gastrointestinal tract and causes vomiting. Acidic beverages made in galvanized containers have produced mass poisonings. Fever, nausea, vomiting, stomach cramps, and diarrhea develop 3 to 12 hours after ingestion (Carson et al., 1987).

VI. Chronic Toxicity

The long-term health effects of oral exposure to zinc have been studied in rats. A 2-year dietary study using zinc cyanide demonstrated a decrease in weight gains, thyroid malfunction, and nerve degeneration. Oral administration of zinc phosphide in a 13-week study resulted in loss of appetite, loss of weight, hair loss, and degeneration of liver, kidney and brain.

Chronic and subchronic inhalation exposures of humans to zinc have been associated with gastrointestinal disturbances, dermatitis and metal fume fever, a condition characterized by fever, chills, coughing, dyspnea and muscle pain (ATSDR, 1989). Chronic oral exposure of humans to zinc may cause anemia and altered hematological parameters. Reduced body weights have been observed in studies in which rats were administered high levels of zinc in the diet (ATSDR, 1989).

VII. Mutagenicity, Carcinogenicity, and Teratogenicity

There is no evidence that zinc is mutagenic, carcinogenic or teratogenic (ATSDR, 1989).

VIII. EPA Carcinogenic Classification and EPA Dose-Response Parameters

EPA Carcinogenic Classification:

Zinc is not classifiable as to human carcinogenicity (Group D), based on inadequate evidence in humans and animals.

EPA Dose-Response Parameters (IRIS, 1994):

Carcinogenic Effects:

The EPA has not promulgated any carcinogenic dose-response parameters for zinc.

Noncarcinogenic Effects:

ORAL RfD SUMMARY:
RfD : 0.3 mg/kg/day
CRITICAL EFFECT/TARGET ORGAN : 47% decrease in erythrocyte superoxide dismutase concentration in human female adults after 10 weeks of zinc exposure.

ORAL RFD UNCERTAINTY :
UF -- An uncertainty factor of 3 was used, based on a minimal LOAEL from a moderate-duration study of the most sensitive humans and consideration of a substance that is an essential dietary nutrient.

ORAL RFD MODIFYING FACTOR :
MF -- 1.

ORAL RFD CONFIDENCE
Study -- Medium
Data Base -- Medium
RfD -- Medium

The level of confidence in the studies is medium since they are well-conducted clinical studies with many biochemical parameters investigated but only few numbers of humans were tested. The confidence in the overall database is medium since these studies are all of short duration. Medium confidence in the RfD follows.

Via the dermal route, patients died after antiscabies ointment containing chromium (VI) was applied to the skin (Brieger 1920). Symptoms included necrosis at the application site, nausea, vomiting, shock and coma. Autopsies revealed tubular necrosis and hyperemia of the kidney. Other reviews of death after dermal exposure to chromium compounds include Major (1922) and Fritz, et al., (1959). It is important to note that these cases involved damaged rather than intact skin.

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B.25 1,1,2,2-TETRACHLOROETHANE

Introduction

1,1,2,2-Tetrachloroethane is a synthetic, colorless, dense liquid that does not burn easily and is volatile. It has a penetrating sweet, odor similar to chloroform. In the past, it was used in large amounts to produce other chemicals. It was also used as an industrial solvent, a chemical separator, a cleaner and degreaser for metals, and in paints and pesticides. Since less toxic chemicals are now available to replace 1,1,2,2-tetrachloroethane, large scale production of the chemical has stopped and its use is now very limited in the United States. Most 1,1,2,2-tetrachloroethane released to the environment makes its way into the air or groundwater where it has a half-life of approximately 1 to 3 months. Its half-life in air is approximately 2. As it degrades by losing chlorine atoms, it produces breakdown products which may be more toxic than the original chemical. Due to its volatility, 1,1,2,2-tetrochloroethane is not generally found in surface soils, nor has it been reported in food. It does not have a tendency to bioaccummulate (ATSDR 1995; USEPA 1996a).

Toxicokinetics

The most probable routes of exposure are inhalation and dermal contact, although ingestion can also occur. Many of the metabolic byproducts of 1,1,2,2-tetrachloroethane are more toxic than the original chemical. Most of the chemical leaves the body within a few days (ATSDR 1995; Andrews and Snyder 1991).

Toxicity

Noncarcinogenic Effects

Exposure to small amounts of 1,1,2,2-tetrachloroethane are not life-threatening; however, chronic ingestion or inhalation of small amounts can cause liver damage, stomach aches, or dizziness. In addition, inhalation of concentrations high enough to detect an odor can cause fatigue, vomiting, dizziness, and possibly unconsciousness. Recovery from these effects is generally rapid following exposure to fresh air (ATSDR 1995). The USEPA has not published oral or inhalation reference doses for 1,1,2,2-tetrachloroethane; however, an oral reference dose is currently under review (USEPA 1996b).

Carcinogenicity

1,1,2,2-tetrachloroethane has been classified by the USEPA as a Group C possible human carcinogen, based on an increased incidence of hepatocellular carcinomas in mice exposed to the chemical and a lack of human carcinogenicity data. Army workers exposed to 1,1,2,2-tetrachloroethane vapors in a clothing processing plant showed a very slight increase in the incidence of death due to genital cancers or to leukemia and other lymphomas when compared to similar workers outside the plant. However, limitations to this study (e.g., low concentrations and lack of control over the subjects) result in the data being inconclusive as to the carcinogenicity of 1,1,2,2-tetrachloroethane. No other studies were located regarding the carcinogenic effects in animals following inhalation exposures. Likewise, no studies were located regarding carcinogenic effects in humans following oral exposures. Oral exposure studies in mice have shown an increase in the incidence of carcinomas in the liver. The

USEPA has calculated an oral slope factor of 0.2 per milligrams per kilogram body weight per day $(mg/kg\text{-}day)^{-1}$ for 1,1,2,2-tetrachloroethane, based on this study. An inhalation unit risk of 5.8×10^{-5} per micrograms per cubic meter $(\mu g/m^3)^{-1}$ has also been established (USEPA 1996b). No studies were located regarding carcinogenic effects of dermal exposures in humans or animals (ATSDR 1995).

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Dugway Proving Ground Closure Plans
Database Field Definitions
File: DPGSWMU2.dbf

Field Name	IRDMIS Standard	Definition
MEDIA_TYPE	✓	Defines soil (CSO), surface water (CSW), or groundwater (CGW)
SITE_TYPE	✓	Defines site location type
SITE_ID	✓	Location
TESTNAME	✓	Analyte code name
LAB	✓	Laboratory
SAMPDATE	✓	Sample date
LOT	✓	Lot
LOTNO	✓	Lot number
METHOD	✓	Method (EPA SW-846 number or AEC method for agent breakdown products)
METH_MATRX	✓	Sample matrix
FC	✓	Flag code
DATA_QUALS	✓	AEC data qualifiers
EPAQUAL		EPA validation code (Added by Ebasco/FW)
DEPTH	✓	Depth
BOOLEAN	✓	Boolean (LT and ND = nondetect; blank or GT = detect)
VALUE	✓	Concentration value
UNITS	✓	Concentration unit
FLDSAMPNO	✓	Field sample number (tag number)
LAB_ANLY_N	✓	Lab analysis number
DIL_FAC	✓	Dilution factor
ANLY_TYPE	✓	Analysis type (target vs. nontarget)
SWMU		SWMU number
MDL		Method detection limit
SOILLAYER		Designates surface vs. subsurface soil samples
BKGDVAL_S		Soil background value
GT_BKGD_S		Detected concentration greater than background value?
PRIM_CONT	✓	Prime contractor
MOB		Ebasco/FW mobilization (1, 2, or 3)
B_S		Indicates background or SWMU sample

✓ - For further clarification of this field refer to the IRDMIS Users' Manual, Volume II, Data Dictionary

EPAQUAL

Note: All rejected data have been removed from this database.

Dugway Proving Ground Closure Plans
Database Field Definitions
File: EBESBKGD.dbf

Field Name	IRDMIS Standard	Definition
MEDIA_TYPE	✓	Defines soil (CSO), surface water (CSW), or groundwater (CGW)
SITE_TYPE	✓	Defines site location type
SITE_ID	✓	Location
TESTNAME	✓	Analyte code name
LAB	✓	Laboratory
SAMPDATE	✓	Sample date
LOT	✓	Lot
LOTNO	✓	Lot number
METHOD	✓	Method (EPA SW-846 number or AEC method for agent breakdown products)
METH_MATRX	✓	Sample matrix
FC	✓	Flag code
DATA_QUALS	✓	AEC data qualifiers
DEPTH	✓	Depth
BOOLEAN	✓	Boolean (LT and ND = nondetect; blank or GT = detect)
VALUE	✓	Concentration value
UNITS	✓	Concentration unit
FLDSAMPNO	✓	Field sample number (tag number)
LAB_ANLY_N	✓	Lab analysis number
DIL_FAC	✓	Dilution factor
ANLY_TYPE	✓	Analysis type (target vs. nontarget)
EPAQUAL		EPA validation code (Added by Ebasco/FW)
Fields 22, 24-31		*Fields used to assist data validation effort*
DEPTH_VAL	✓	DEPTH field presented as a numeric field
SWMU		SWMU number
MDL		Method detection limit
CONCSTAT		Concentration used in statistics (detections=VALUE, nondetections=0.5*MDL)
SOILLAYER		Designates surface vs. subsurface soil samples
BKGDVAL_S		Soil background value
RBSL		Risk-based screening level
MOB		Ebasco/FW mobilization (1, 2, or 3)
CLASS		Indicates metal or organic constituent
B_S		Indicates background or SWMU sample
PRIM_CONT	✓	Prime contractor

✓ - For further clarification of this field refer to the IRDMIS Users' Manual, Volume II, Data Dictionary

EPAQUAL

Note: All rejected data have been removed from this database.