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**BASELINE RISK ASSESSMENT
FOR HADNOT POINT INDUSTRIAL
AREA OPERABLE UNIT
SHALLOW SOILS AND
CASTLE HAYNE AQUIFER**

DRAFT FINAL

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LIST OF ACRONYMS AND ABBREVIATIONS

µg/L	micrograms per liter
µg/dL	micrograms per deciliter
AOC	Area of Concern
ARAR	Applicable or Relevant and Appropriate Requirements
AWQC	Ambient Water Quality Criteria
BaP	Benzo(a)pyrene
Bis2HEP	bis-(2-ethylhexyl)-phthalate
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
cfs	cubic feet per second
COC	Chemical of Concern
CSF	cancer slope factor
CTS	chemical toxicity score
EPA	Environmental Protection Agency
ESE	Environmental Science & Engineering, Inc.
ft	feet
ft-bls	feet below the land surface
FTU	Formazin Turbidity Unit
HOLF	Helicopter Outlying Landing Field
HPIA	Hadnot Point Industrial Area
K _{oc}	soil sorption coefficient
LANTDIV	Naval Facilities Engineering Command - Atlantic Division
MCAS	Marine Corps Air Station
MCB	Marine Corps Base
MCL	Maximum Contaminant Level
mg/L	milligrams per liter
mg/kg/day	milligrams per kilogram per day
mi ²	square miles
MSL	mean sea level
NTU	Nephelometric Turbidity Unit
OLF	Outlying Landing Field
PAHs	Polynuclear Aromatic Hydrocarbons
PCBs	Polychlorinated Biphenyls
ppm	parts per million
RA	Risk Assessment

RAGS	Risk Assessment Guidance for Superfund
RfD	Reference Dose
RI/FS	Remedial Investigation/Feasibility Study
RME	Reasonable Maximum Exposure
SEAM	Superfund Exposure Assessment Manual
SPHEM	Superfund Public health Evaluation Manual
SR	State Road
SVOC	Semivolatile Organic Chemical
TCL	Target Compound List
TCLP	Toxicity Characteristic Leaching Procedure
TCLVOA	Target Compound List Volatile Organic Analytes
TOSCA	Toxic Substances Control Act
USGS	United States Geological Survey
VOC	Volatile Organic Chemical
WoE	Weight of Evidence

EXECUTIVE SUMMARY

MCB Camp Lejeune is a training base for the Marine Corps, located in Onslow County, North Carolina. It covers approximately 170 square miles, and is bounded to the southeast by the Atlantic Ocean, to the west by U.S. Highway 17, and to the northeast by State Road 24. The base is bisected by the New River estuary, which occupies approximately 30 square miles of the total area of the facility.

The Hadnot Point Industrial Area (HPIA) of MCB Camp Lejeune is located on the east side of the New river estuary. The HPIA is comprised of approximately 75 buildings and facilities. These include maintenance shops, has stations, administrative offices, commissaries, snack bars, warehouses, storage yards, and a dry cleaning facility. A steam plant and training facility occupy the southwest portion of HPIA. In addition, underground storage tanks, stormwater drains, and oil/water separators are present. As a result of marine operations and activities, wastes that contain hazardous and toxic organic compounds are generated at the base. This has resulted in the storage, disposal, and/or spillage of these wastes. Several of the base's water supply wells at HPIA have been shut down as a result of the presence of organic compounds, thus suggesting that some of the wastes may have entered the groundwater.

Due to the potential of spillage of wastes in the HPIA, several investigations have been conducted to date on the Hadnot Point Operable Unit, which is defined as that area bounded by Holcomb Boulevard to the west, Sneads Ferry Road to the north, Louis Street to the east, and the Main Service Road to the south. The Hadnot Point Operable Unit also includes the two primary hydrologic units; an unconfined surficial aquifer and a semi-confined potable aquifer (Castle Hayne Aquifer).

A transformer storage yard (Site 21) and a fuel tank farm (Site 22) are located within the northern portion of HPIA. Two other study areas, the industrial area fly ash dump (Study Area 24) and the Hadnot Point burn dump (Study Area 28) lie to the south and southwest of the site. These areas of concern are not included in the operable unit, and will be considered in separate studies at a later date.

The investigation of the HPIA has been completed as a phased approach, with the results of one investigation being the basis for the next phase. Three major investigations or Studies have been completed at the installation prior to the completion of this report. These investigations are described below.

An Initial Assessment Study (IAS) was conducted under the NACIP program at MCB Camp Lejeune in 1983. The IAS report (Water and Air Research, 1983), which was a record search of the installation, identified a number of areas within MCB Camp Lejeune, including the HPIA, as potential sources of contamination. As

a result of this study, Environmental Science and Engineering, Inc. (ESE) was contracted by the Navy to investigate the HPIA, as well as other potential source areas.

The initial ESE investigation, referred to as the Confirmation Study, is divided into two investigation steps: the Verification Step and the Characterization Step. The Verification Step at HPIA was conducted to determine if areas of suspected contamination, as documented in the IAS, were indeed contaminated. This investigation was conducted from April 1984 through January 1985, and involved the installation of three shallow groundwater monitoring wells and the sampling of the potable water supply wells in the HPIA, as well as the investigation of other sites within Camp Lejeune. This step identified the presence of volatile organic compounds (VOCs) in the shallow aquifer in the vicinity of the Hadnot Point Industrial Area Tank Farm (Site 22) and in a single potable supply well (602).

Based on the results of the Verification Step, the Characterization Step was performed at HPIA during the period of 1986 through 1988. This phase was designed to evaluate the extent of the VOC contamination identified in the Verification Step within the HPIA. The Characterization Step consisted initially of a records search of available base records, a physical inspection of each building within HPIA, and a soil gas survey targeted to those areas identified by the records search as being potential contamination sources.

Each of the areas identified by the records search as potential sources of VOCs was investigated with the use of the soil gas technique that focused on TCE as the contaminant of concern. Areas that exhibited TCE or other VOC contamination in the soil included the area around Buildings 901, 902, and 903, Building 1202, and Buildings 1502, 1601, and 1602.

Following analysis of the record search and soil gas data, locations were chosen for the installation of 27 shallow (25-foot), 3 intermediate (75-foot), and 3 deep (150-foot) monitoring wells to determine if contamination identified during the soil gas investigation had migrated to the shallow and deeper groundwater. All new and existing HPIA monitoring wells and nearby water supply wells were then sampled.

Aquifer testing of one deep potable supply well was conducted to evaluate the hydraulic parameters of the Castle Hayne Aquifer and to determine the transport mechanisms between the shallow and Castle Hayne aquifers.

The Confirmation Study served to narrow the list of source areas to three primary areas, being the areas surrounding Buildings 902, 1202, and 1601.

The Supplemental Characterization Step performed at PHIA in 1990-1991, was designed to further evaluate the extent of contamination in the Castle Hayne Aquifer and to characterize the contamination within the shallow soils at suspected source locations. The supplemental Characterization Step consisted of 30 soil borings at the

3 suspected source locations (Buildings 902, 1201, and 1601) to characterize shallow soil contamination, installation of additional intermediate and deep monitoring wells into the Castle Hayne aquifer, and sampling of all new and existing HPIA monitoring wells and nearby water supply wells.

Based on these investigations, the shallow soils at the areas investigated do not appear to be significantly contaminated. Volatile compounds detected in the soil gas remain in the vapor phase and have not adhered to the soils. Some semi-volatile compounds were detected in low concentrations in the soil.

The groundwater sampling and analysis program continues to reflect two nodes of VOC and/or petroleum hydrocarbon contamination within the shallow aquifer. The northern node consists of two separate sources of contamination--one centered near the maintenance facility associated with Building 901, and another centered at the Hadnot Point Fuel Tank Farm (Site 22). Contaminant isopleth modeling suggests that these two source areas may have effectively coalesced into one larger node of contamination. The southern node is centered near the maintenance facility associated with Building 1601. The surficial aquifer will initially be remediated under an Interim Remedial Action, which is the subject of reports prepared under separate cover.

A risk assessment has been completed for the shallow soils at the three remaining areas of concern. This assessment has shown that the low levels of contamination detected within the soils do not pose a human or ecological threat. This RA also addressed the groundwater within the Castle Hayne Aquifer. While contaminants have been detected in one monitor well and in several potable wells, no current risk was identified. Additional studies addressing the extent of contamination within the Castle Hayne Aquifer are being undertaken under separate cover.

1.0 INTRODUCTION

1.0 INTRODUCTION

A baseline human health and ecological risk assessment (RA) was conducted for the Hadnot Point Industrial Area (HPIA) within the Marine Corps Base (MCB) Camp Lejeune, Jacksonville, North Carolina. The RA includes identification of chemicals of concern, exposure assessment, toxicity assessment, risk characterization, and a component of the remedial investigation/feasibility study (RI/FS) for the MCB Camp Lejeune. The RI/FS and the RA are being completed by Environmental Science & Engineering, Inc. (ESE) for the Naval Facilities Engineering Command-Atlantic Division (LANTDIV), as authorized under the A&E Contract Number N62470-83-C-6106.

This RA summarizes and interprets surface soil (0 to 2 feet deep) and groundwater (intermediate and deep) data from the RI (ESE, 1991) to determine potential human health and environmental risks associated with these media at four areas of concern (AOCs) within HPIA: buildings 902, 1202, 1602, and Hadnot Point fuel tank farm. The RA presents the health risks associated with these four areas under baseline conditions, in the absence of any remedial action (the no-action alternative). Soil samples (deeper than 50 feet) were not evaluated in the risk assessment because the type of worker activity (i.e., vehicle maintenance) does not involve soil excavation, therefore, these workers are not exposed to deeper soils.

Offsite (outside the HPIA) risks associated with contamination migrating from the four AOCs were not addressed because offsite contaminant migration was not within ESE's scope of work. Offsite wells were not installed for this investigation; all soil samples were collected from within the HPIA.

Based on results of the risk characterization, which identifies the degree of human and environmental health impacts posed by each AOC, the RA will identify whether these areas require remediation. If the results of the baseline RA indicate that particular areas and contaminants require remediation, then the risk results will be used to prioritize remedial activities and to develop health-based cleanup goals as potential remedial action objectives. The results of the baseline RA will be obtained by:

- Evaluating the analytical data obtained during remedial investigations;
- Identifying the site-related contaminants of most significant health concerns;
- Identifying potential exposure pathways of human and nonhuman populations for Chemical of Concern (COCs); and
- Evaluating the actual or potential health impacts associated with the exposure of these populations to the reasonable maximum concentration of site-related contaminants.

Guidance available in the following documents provided the methods to determine the reasonable maximum baseline conditions of a site: Risk Assessment Guidance for Superfund (RAGS) Volumes I and II, Human

Health Evaluation Manual and Environmental Evaluation Manual (U.S. Environmental Protection Agency [EPA], 1989); Guidance for Conducting Remedial Investigations (EPA, 1988); Superfund Exposure Assessment Manual (EPA, 1988); and the RAGS Supplemental Guidance: Standard Default Exposure Factors (EPA, 1991).

Results of the data evaluation indicate that the data does not represent the extent of contamination for several compounds due to the limited number of samples collected and low frequency of detection. Thus, the approach taken for this baseline RA was to evaluate the reasonable maximum exposure (RME) based on the maximum concentration observed in the available data for each AOC. Although this assessment may present an overly conservative view of the nature and extent of contamination, this approach is consistent with RAGS (EPA, 1989), which states that maximum detected concentrations may be used as a screening approach to place an upperbound limit on exposure. These conclusions are also consistent with onsite investigations in that each investigation and sampling round attempted to identify the most likely sources of contamination. For those AOCs where the risk results indicate remediation is required, confirmatory sampling is recommended as part of the FS to ascertain the true extent of contamination.

The following sections describe the history of the AOCs, summarize the significance of findings during previous studies, and present the scope and organization of the RA.

1.1 SITE BACKGROUND

1.1.1 SITE LOCATION AND HISTORY

MCB Camp Lejeune is a training base for the Marine Corps, located in Onslow County, North Carolina. The facility covers approximately 170 square miles (mi²) and is bounded to the southeast by the Atlantic Ocean, to the West by U.S. Highway 17, and to the northeast by State Road (SR) 24. The base is bisected by the New River estuary, which occupies approximately 30 mi² of the facility's total area (Figure 1-1).

Construction of Camp Lejeune began in the late 1930s at Hadnot Point, where functions were centered. During World War II, the Vietnam War, and the Korean conflicts, Camp Lejeune was used as a training area to prepare Marines for combat. There are five major areas of development within the Camp Lejeune facility, including: Camp Geiger, Montford Point, Mainside, Courthouse Bay, and the Rifle Range area. Marine Corps Air Station (MCAS) New River, a helicopter base, is a separate command on the west side of the New River. Helicopter Outlying Landing Field (HOLF) Oak Grove and Outlying Landing Field (OLF) Camp Davis are also under the command of MCAS New River. The HOLF Oak Grove is no longer active, however, the property has some camping facilities and is occasionally used for recreation by scout troops.

The HPIA of MCB Camp Lejeune is located to the east of the New River and is defined as the area bounded by Holcomb Boulevard to the west, Sneads Ferry Road to the north, Louis Street to the east, and the Main Service Road to the south (Figure 1-2). The area is comprised of 75 buildings and facilities, including:

maintenance shops, gas stations, administrative offices, commissaries, snack bars, warehouses, storage yards, and a dry cleaning facility. A steam plant and training facility occupy the southwest portion of HPIA. In addition, numerous underground storage tanks, stormwater drains, and oil/water separators are present.

A transformer storage yard (Site 21) and a fuel tank farm (Site 22) are located on the north side of HPIA. Both of these are potential AOCs. However, only Site 22 was included in the 1991 RA scope of work.

The aquatic ecosystems within MCB Camp Lejeune consist of small lakes, the New River estuary, numerous tributary creeks, and part of the intracoastal waterway. The terrestrial ecosystems include four habitat types: long leaf pine, loblolly pine, loblolly pine/hardwood, and oak/hickory. Camp Lejeune is predominantly wooded with large amounts of softwood and substantial stands of hardwood species. More than 60,000 of the 112,000 acres within the base are under forestry management, with loblolly pine as the main timber source of the area.

Prior to 1941, the water supply for the base was furnished by wells that tapped a potable aquifer 50 to 300 feet below the base. In 1941, a water treatment system, which included 21 water supply wells, was placed on-line at HPIA. This system was used by most of the base until the 1950's. At that time, additional wells and treatment facilities were installed. In 1991, eight water treatment facilities and over 160 water supply wells serve the Camp Lejeune installation. There are wells within the Hadnot Point Area (not confined to the industrial area) that are drawing water from the deep aquifer. All water from these wells is processed by a treatment facility prior to distribution for potable use.

1.2 SUMMARY OF PREVIOUS SITE STUDIES

A series of studies and investigations have been performed at MCB Camp Lejeune to evaluate the extent of contamination from disposal activities at the facility. Based on the results of these investigations, four areas within the HPIA were identified as potential AOCs to be further evaluated in the remedial investigation:

- Buildings 901, 902;
- Buildings 1200, 1202;
- Buildings 1600, 1601, 1602; and
- Site 22, Hadnot Point fuel tank farm.

Table 1-1 lists the studies and investigations conducted at HPIA by ESE, along with a brief summary of the significant findings. For a detailed discussion of all previous studies and investigations and information obtained from additional site characterization efforts, refer to the Comprehensive RI report (ESE, 1991).

In 1990, an Initial Assessment Study was conducted at MCB Camp Lejeune as part of the Department of Defense's Installation Restoration Program, during which a number of areas within MCB Camp Lejeune were identified as potential sources of contamination. ESE was contracted by LANTDIV to conduct a Confirmation

Table 1-1. Summary of Previous Investigations Conducted at HPIA.

Title	Contractor	Study Description
Confirmation Study	ESE	The Confirmation Study is analogous to an RI/FS performed for Superfund sites. This study focused on the areas of concern identified in the Initial Assessment Study. The Confirmation Study is divided into two investigation steps: the Verification Step and the Characterization Step.
Verification Step	ESE	The Verification Step at HPIA was conducted in 1985 and identified the presence of volatile organic compounds within the shallow aquifer in the vicinity of HPIA fuel tank farm. Maximum contaminant concentration detected include 17,000 µg/L of benzene and 27,000 µg/L of toluene. From the results of this study, five water supply wells were found contaminated with VOCs and were subsequently shutdown.
Characterization Step	ESE	The Characterization Step was designed to evaluate the extent of VOC contamination identified in the Verification Step. This study involved five tasks which included a records search and building inspection, soil gas survey at potential contamination sources, installation of 27 shallow, 3 intermediate and 3 deep monitoring wells, sampling of all existing HPIA wells, and an aquifer test to evaluate the hydraulic parameters of the deep aquifer.
Shallow Groundwater Feasibility Study	ESE	In 1988, ESE conducted a focused Feasibility Study for remediating shallow groundwater at HPIA. A pump and treat alternative was determined to be the most feasible remedial alternative (ESE, 1985).
RI/FS for HPIA and Limited Scope Investigations at Sites 6, 48, and 69	ESE	This effort was a continuation of the Confirmation Step performed by ESE in 1984-1988. This study characterized shallow and deep groundwater contamination and shallow soils contamination at HPIA, and groundwater, surface water, sediment, and shellfish tissue contamination at Sites 6, 48, and 69.

Source: ESE, 1990.

1-6

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Study, which is analogous to an RI/FS performed for EPA on federal Superfund sites. The confirmation study was divided into two investigative steps: the verification step and the characterization step.

The Verification Step took place from April 1984 through January 1985. Results of this investigation indicate the presence of volatile organic compounds (VOCs) within the shallow aquifer in the vicinity of HPIA fuel tank farm and in water supply well 602. The maximum contaminant concentrations observed in groundwater include 17,000 $\mu\text{g/L}$ of benzene and 27,000 $\mu\text{g/L}$ toluene collected from the tank farm area. Benzene was also detected in supply well 602 at concentrations of 38 $\mu\text{g/L}$, which exceeds the federal maximum contaminant level (MCL) of 5 $\mu\text{g/L}$. Analytical data for xylene and ethylbenzene were not provided. Although not included in this analysis, that data will be included at a later date.

Due to the results of the verification step, supply well 602 was closed and other wells in the area were sampled. Four additional supply wells (601, 608, 634, and 637) were found to have elevated levels of VOCs, including trichloroethylene in wells 601 and 608 and methylene chloride in well 634. Figure 2-5 shows the location of the monitor wells.

In 1986, the characterization step was conducted for HPIA to determine the extent of the VOC contamination identified. During the characterization step, multiple tasks were completed, including: a soil gas survey to target areas identified as being potentially contaminated, installation of 27 shallow (25 foot), 3 intermediate (75 foot), and 3 deep (150 foot) monitoring wells, sampling of all HPIA monitoring wells and nearby water supply wells, and aquifer testing to evaluate the hydraulic parameters of the deep aquifer.

Results of the characterization study revealed that five of the areas within HPIA showed elevated levels of VOCs in soil gas: 1) Buildings 901, 902 and 903; 2) Building 1100; 3) Buildings 1101, 1102, 1202, 1301, and 1302; 4) Buildings 1502, 1601; and 5) Buildings 1709 and 1710. Results of the shallow monitoring well analyses revealed the presence of elevated levels of a number of petroleum related compounds, including: benzene, xylene, ethylbenzene, trans-1,2-dichloroethene, trichloroethene, oil and grease, and lead. Groundwater analyses from the Confirmation Study investigations are summarized and presented in Tables 1-2 and 1-3. Inorganics, including mercury, were detected in several of the deep aquifer wells, but detected levels were within EPA MCLs or assistant water quality criteria guidelines (AWQCs).

Site 22 (the Hadnot Point fuel tank farm) is located within the area of HPIA and was included as part of the scope for this risk assessment. Site 21, the transformer storage yard, will be further addressed in a separate assessment to be conducted at a later date.

Two shallow groundwater monitoring wells and water supply well 602 were sampled during the 1984 investigation. Samples collected were analyzed for lead, VOCs, and oil and gas. The concentration of benzene (17000 $\mu\text{g/L}$)

Table 1-2. Summary of Target Analytes Detected in Groundwater From the 1988 Confirmation Investigation for HPIA.

Chemical	Frequency of Detection Shallow Aquifer	Maximum ($\mu\text{g/L}$)	Frequency of Detection Deep Aquifer	Maximum ($\mu\text{g/L}$)
Bis2HEP	N/A	---	1/6	13
Benzene	13/81	13000	7/6	720
Chloroform	3/81	3.2	N/A	---
Chloromethane	3/81	7.2	N/A	---
1,1-Dichloroethane	1/81	12	N/A	---
1,2-Dichloroethane	0/81	---	2/6	46
trans-1,2-Dichloroethene	15/81	6400	14/6	700
Ethylbenzene	5/81	1800	1/6	8
Oil and Grease	42/81	32000	N/A	---
Lead	16/81	130	---	---
Methylene Chloride	7/81	300	3/6	130
Tetrachloroethane	1/81	3.6	4/6	24
Toluene	9/81	24000	3/6	12
1,1,1-Trichloroethane	1/81	13	N/A	---
Trichloroethene	14/81	13000	11/6	1600
Trichlorofluoromethane	2/81	96	1/6	3
Vinyl Chloride	2/81	250	1/6	18
Xylene	6/81	9000	N/A	---
Methyl Ethyl Ketone	0/81	---	2/6	290

Bis2HEP = bis-(2-ethylhexyl)-phthalate

Source: ESE, 1990, 1988

Table 1-3. Inorganics Analyzed in ESE 1986 Sampling for HPIA.

Chemical	Frequency of Detection (Shallow Aquifer)	Maximum (µg/L)
Barium (total)	4/4	43.4
Nitrogen (total)	1/4	42
Nitrogen (NO ₂)	1/4	42
Total Iron	4/4	15200
Chloride	4/4	68300
Manganese (total)	4/4	134
Sodium (total)	4/4	12300
Sulfate	3/4	5,170,000
Turbidity (FTU/NTU)	4/4	18.0
Chromium	4/4	574
Copper	4/4	14.1
Mercury	4/4	0.7
Zinc	4/4	3200

FTU = Formazin Turbidity Unit
NTU = Nephelometric Turbidity Unit

Source: ESE, 1990.

was substantially greater than the North Carolina groundwater standard of 0.70 $\mu\text{g}/\text{L}$ (ESE, 1990). Concentrations of chloroform, ethylbenzene, and toluene also exceeded groundwater standards. The sample from supply well 602 contained six VOCs and lead. Benzene was detected at a concentration of 380 $\mu\text{g}/\text{l}$.

Based on the results of these studies, four areas around Buildings 902, 1202, 1602, and the Hadnot Point fuel tank farm were evaluated in the risk assessment to determine if the surface soils and deep groundwater pose unacceptable health risks based on the exposure assumptions evaluated.

1.3 SCOPE AND ORGANIZATION OF THE RISK ASSESSMENT

The RA process, as it applies to MCB Camp Lejeune, is a systematic approach to characterizing the probability of adverse human health effects and ecological impacts resulting from exposure to the chemicals identified in the environmental media at the four study areas. The RA consists of the following four sequential steps (Figure 1-3):

- Identification of (COCs),
- Exposure assessment,
- Toxicity assessment, and
- Risk characterization.

The RA was performed for the four study areas of concern based on the available analytical data presented in the Comprehensive RI (ESE, 1991) and was conducted in accordance with the guidelines and methods presented in the Risk Assessment Guidance for Superfund (RAGS), Volumes I and II; the Superfund Public Health Evaluation Manual (SPHEM), the Superfund Exposure Assessment Manual (SEAM), the Guidance for Conducting Remedial Investigations and Feasibility Studies under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), and additional EPA guidance and directives applicable to each component of the RA process. A detailed discussion of each component of the RA for MCB Camp Lejeune is presented in the following sections.

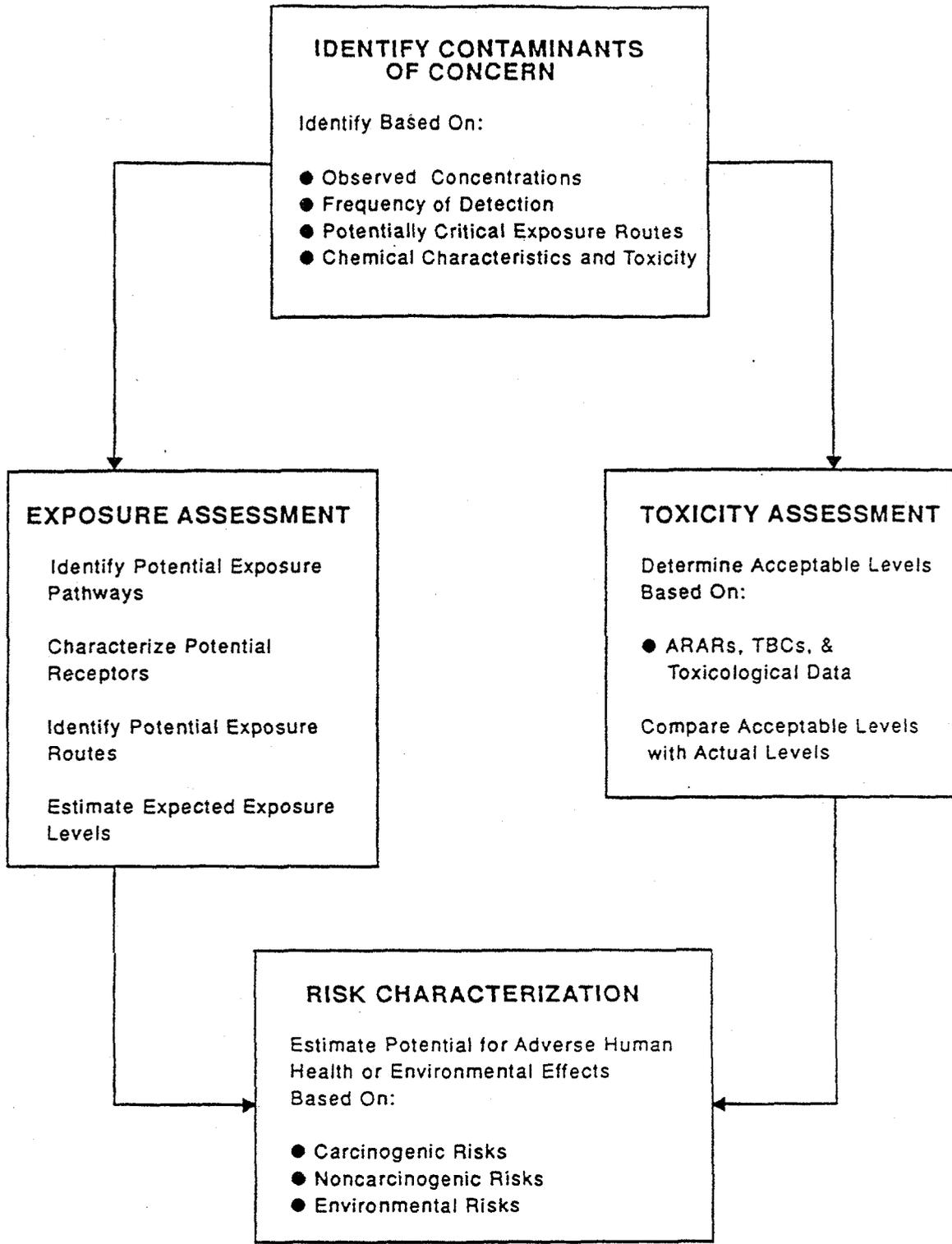


Figure 1-3
RISK ASSESSMENT PROCESS



MARINE CORPS BASE
CAMP LEJEUNE

SOURCE: ESE.

**2.0 IDENTIFICATION OF POTENTIAL
CHEMICALS OF CONCERN**

2.0 IDENTIFICATION OF POTENTIAL CHEMICALS OF CONCERN

To make the most effective use of the available information, a subset of chemicals, collectively termed the chemicals of concern (COCs), are evaluated in the baseline risk assessment. COCs are those site-related constituents that pose the most critical health concerns to human and environmental receptors. To select COCs, data are evaluated based on two sets of criteria. The second set include This focused approach of identifying a subset of chemicals for risk analysis is based on the premise that site remedial actions to reduce the concentrations of COCs to acceptable levels will also result in acceptable levels of other similar, but less hazardous, chemicals at the site. To ensure that the most significant COCs are selected, analytical data should be considered that will identify any trends in the chemical concentrations (i.e., concentrations increasing or decreasing over time), as well as all possible exposure pathways to site-related contaminants. This results in the selection of COCs based on data obtained during previous investigations and any information collected during additional site characterization efforts, such as the Comprehensive RI (ESE, 1991).

2.1 PROCESS OF SELECTING CHEMICALS OF CONCERN

The object of the chemical selection process is to limit the chemicals to be addressed in the risk assessment to those likely to contribute to the majority of the total risk (e.g., those most frequently detected) as a result of potential exposure to contaminated media, including groundwater and surface soil. In this manner, the final baseline risk assessment focuses on the most significant COCs, those which are site-related contaminants posing the majority of the total health and environmental risk. This approach allows the baseline risk assessment to focus on those chemicals and areas of most significant concern, while making the most effective use of a large chemical database (EPA, 1989).

The chemical selection process begins by establishing a set of analytical data to be used in the risk assessment. Once the appropriate analytical data are identified and summarized, the two sets of evaluation criteria are identified in order to determine the COCs to be addressed in the risk assessment. The first set are chemical/site specific criteria and include mobility, persistence, and frequency and location of detections (EPA, 1989).noncarcinogenic and carcinogenic toxicity ranking values that are based on the concentration and toxicity of the potential COC. Once the two sets of evaluation criteria are identified, they are evaluated to reduce the number of COCs.

2.1.1 DEVELOPING A SET OF CHEMICAL DATA

The first step in selecting COCs is to develop a set of chemical data and associated information to be used in the RA as described in the Guidance for Data Useability in Risk Assessment (EPA, 1990). This requires gathering all analytical data generated during the site investigation and sorting the data by medium; evaluating analytical methods; evaluating the quality of data with respect to sample quantitation limits, qualifiers, codes, and

blanks; evaluating tentatively identified compounds; comparing potential site-related contamination with background concentrations; and producing a set of data that qualifies for use in the RA. For the purposes of this RA, all data points above instrument detection limits (U qualified data) were considered quantifiable values.

Within HPIA, four AOCs were sampled for intermediate and deep groundwater, and for surface soils. The location of the AOCs are shown in Figure 2-1 and the specific locations of soil sample collection within each AOC are illustrated in Figures 2-2 through 2-4. ESE's scope of work did not allow soil samples to be collected from Site 22, therefore, only the contaminants associated with groundwater were addressed in this RA. A more detailed description of actual samples collected and analyses conducted for each matrix and AOC are summarized in the following sections.

2.1.1.1 Soil

Shallow soil borings (0 to 2 feet deep) were collected at three of the AOCs using carbon steel split spoons. The objective of the soil sampling was to evaluate the chemical and physical nature of shallow (above the water table) soil contamination in the vicinity of Buildings 902, 1202 and 1601-1602. Samples were collected from each boring for chemical analysis, with ten percent of the samples analyzed for full Target Compound List (TCL) parameters. The remaining 90 percent were analyzed for volatile organic compounds (VOAs), pesticides and polychlorinated biphenyls (PCBs), and Toxicity Characteristic Leaching Procedure metals (TCLP metals).

Soil boring samples numbered 1 through 10 were collected within the immediate vicinity of Building 902, while samples 11 through 20, and 21 through 30 were collected from areas around Buildings 1202 and 1602, respectively. Only the data derived from samples collected from the ground surface to a depth of 2 feet were used for the quantification of risk associated to soil exposure. Surface soil samples were not collected for soil borings 14, 18, and 27. A total of 27 surface soil samples were analyzed. The deeper soil samples (2 to 10 feet deep) were addressed qualitatively for comparative purposes only, and not to determine the extent of contaminant leaching. Results of the surface soil sample analyses for all four AOCs are summarized in Table 2-1.

2.1.1.2 Intermediate and Deep Groundwater

In December 1990, four groundwater monitoring well clusters (Figure 2-5) were installed downgradient of the four AOCs. Both intermediate and deep wells were installed at each location to evaluate the vertical distribution of contamination in the groundwater downgradient of the AOCs. The intermediate and deep wells tap the same aquifer, therefore, data from both types of wells were combined for the determination of exposure concentrations. The maximum detected concentrations were used for the exposure concentration. All groundwater samples were analyzed for full TCL parameters and in-field measurements of pH, specific conductance, and temperature. Locations of these wells are shown in Figure 2-5. Results of the chemical analyses for intermediate and deep monitoring well samples are presented in Tables 2-2 and 2-3, respectively.

HADNOT PT RA 791 LB

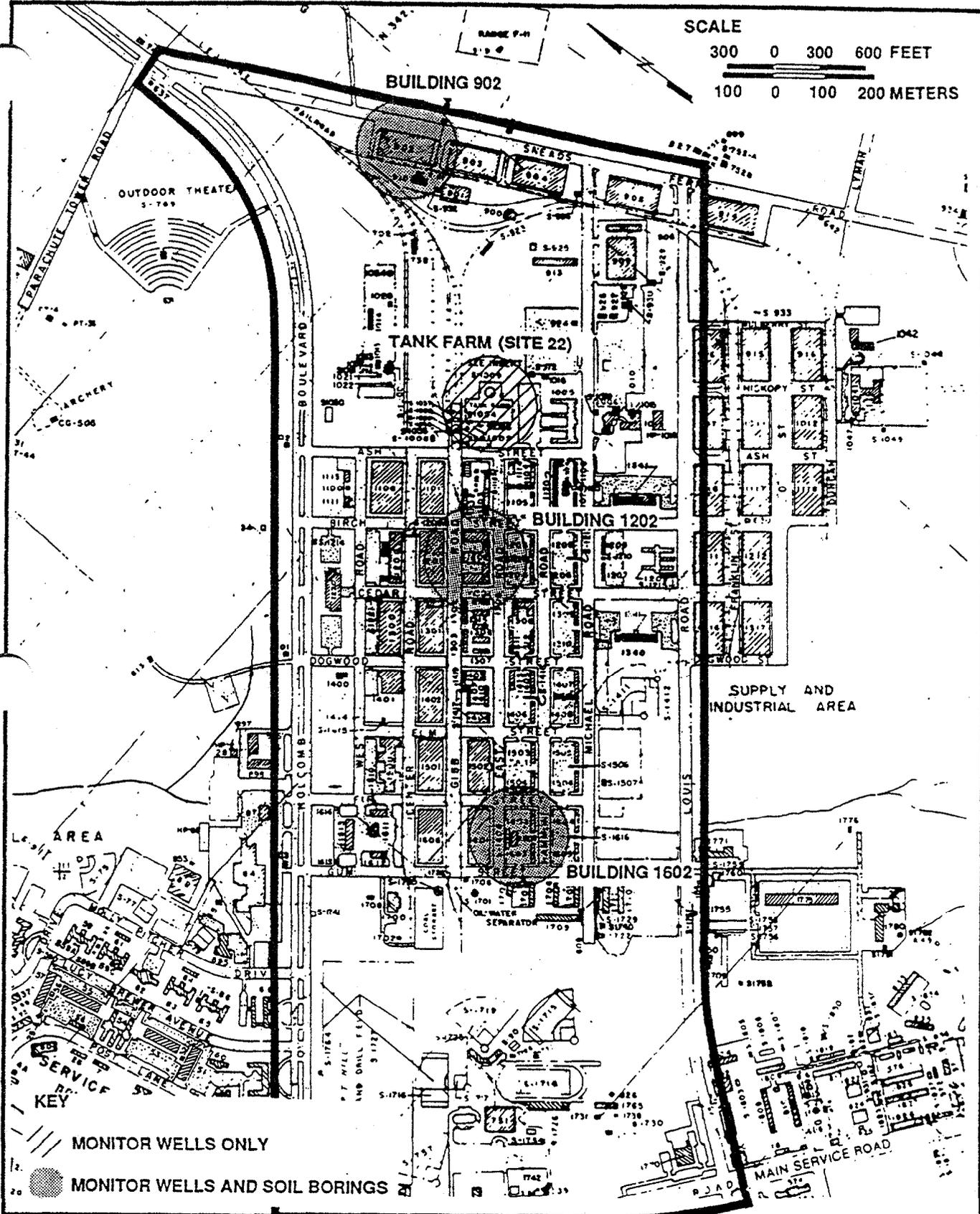


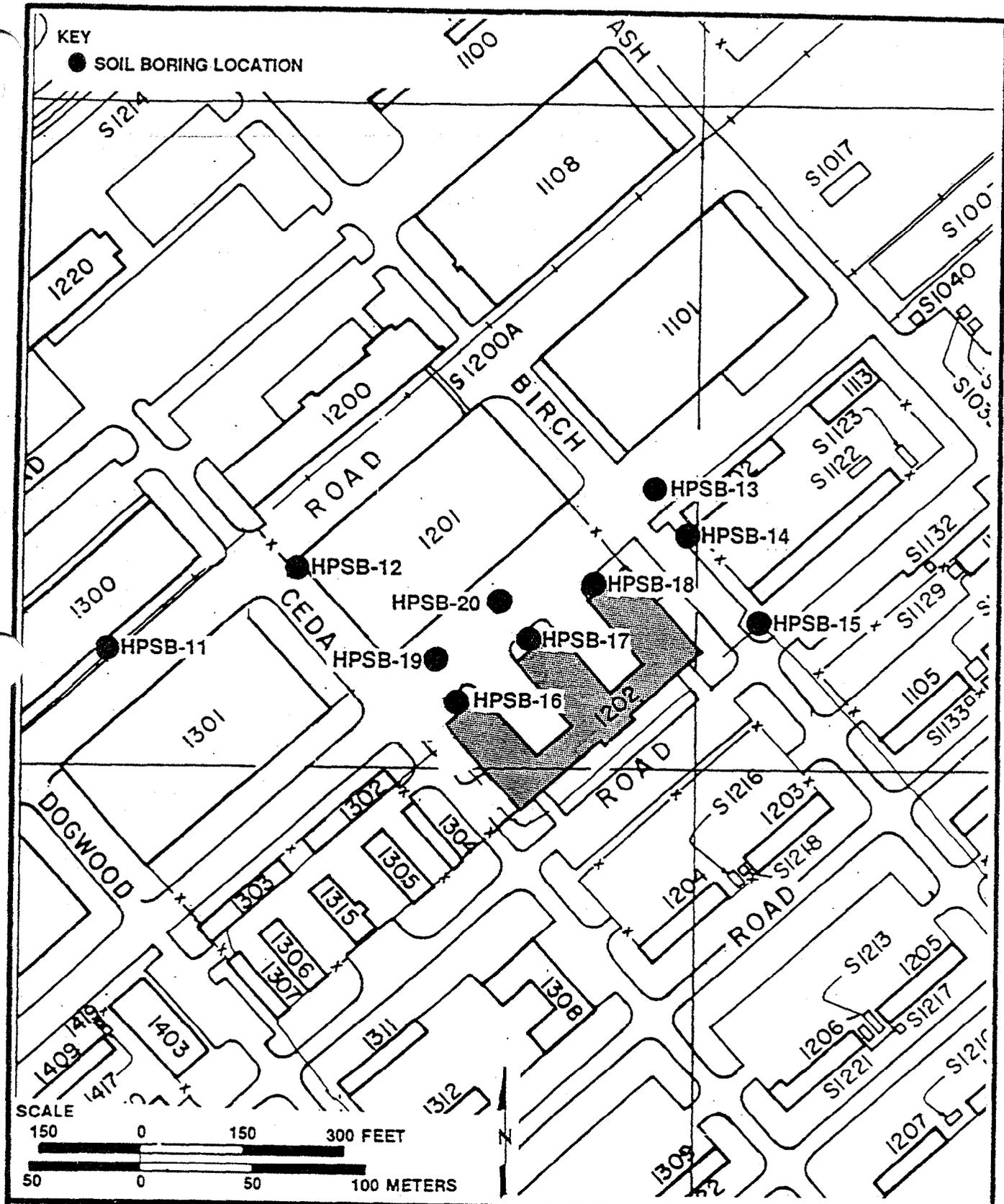
Figure 2-1
LOCATION OF AREAS OF CONCERN WITHIN HPIA



MARINE CORPS BASE
CAMP LEJEUNE

SOURCE: CAMP LEJEUNE, 1987.

HADNOT PT RA 791 LB



MARINE CORPS BASE
CAMP LEJEUNE

Table 2-1. Inorganic, Pesticide, Volatile, and Semivolatile Chemicals Identified in Shallow Soils Collected from HPIA (Surface to 2 feet deep) (Page 1 of 2).

Chemical/Units	Range	Mean	Frequency of Detection ^a
<u>Inorganics (mg/kg)</u>			
Aluminum	1740 - 5620	3451	7/8
Antimony	5.40 - 9.60	6.91	7/8
Arsenic	0.50 - 1.40	0.74	5/8
Barium	6.0 - 19.6	10.20	7/8
Cadmium	0.80 - 3.00	1.30	7/8
Calcium	1450 - 62700	14418	7/8
Chromium	0.59 - 11.80	6.91	8/8
Cobalt	0.93 - 1.70	1.57	7/8
Copper	0.39 - 11.80	4.21	8/8
Iron	5.40 - 5090	1826	7/8
Lead	2.30 - 84.80	24.40	8/8
Magnesium	116 - 1210	450	7/8
Manganese	2.50 - 155	32.70	7/8
Nickel	1.70 - 5.80	2.86	8/8
Potassium	113 - 1190	269	8/8
Selenium	0.21 - 0.45	0.32	2/8
Silver	0.98 - 1.10	1.04	2/8
Sodium	68 - 297	134	8/8
Vanadium	2.60 - 7.40	5.11	7/8
Zinc	0.80 - 61.20	16.80	8/8
<u>Pesticides (µg/kg)</u>			
Aroclor 1254	780	780	1/27
Aroclor 1260	290	290	1/27
Dieldrin	38 - 92	65	2/27
4,4-DDE	78 - 97	87.50	2/27
4,4-DDT	40	40	1/27
<u>Volatile Organic Chemicals (µg/kg)</u>			
Methylene Chloride	1 - 14	2.89	19/27
Acetone	5 - 360	38.37	19/27
<u>Semivolatile Organic Chemicals (µg/kg)</u>			
Acenaphthene	42 - 72	56	2/8
Anthracene	67 - 180	123.50	3/8
Benzo(a)anthracene	41 - 280	132.75	4/8
Benzo(a)pyrene	64 - 240	152	3/8
Benzo(b)fluoranthene	39 - 250	137.25	4/8
Benzo(k)fluoranthene	48 - 210	156	4/8
Benzo(g,h,i)perylene	72 - 110	91	2/8
Bis(2-ethylhexyl)phthalate	16 - 54	35	2/8

Table 2-1. Inorganic, Pesticide, Volatile, and Semivolatile Chemicals Identified in Shallow Soils Collected from HPIA (Surface to 2 feet deep) (Page 2 of 2).

Chemical/Units	Range	Mean	Frequency of Detection ^a
Chrysene	44 - 260	142.25	4/8
Dibenzofuran	51 - 72	61.50	2/8
Di-n-butylphthalate	72	72	1/8
1,4-Dichlorobenzene	47 - 48	47.50	2/8
Fluoranthene	100 - 690	340	4/8
Fluorene	48 - 63	55.50	2/8
Indeno(1,2,3-cd)pyrene	37 - 130	83	3/8
Naphthalene	220	220	1/8
2-Methylnaphthalene	300	300	1/8
Phenanthrene	94 - 500	224	5/8
Pyrene	94 - 530	258	4/8

^a = Number of Samples in which the chemical was positively detected over the number of samples collected.

Source: ESE, 1991.

Table 2-2. Inorganic, Pesticide, Volatile and Semivolatile Chemicals Identified in Intermediate Wells from HPIA.

Chemical/Units	Range	Mean	Frequency of Detection ^a
<u>Inorganics (µg/L)</u>			
Aluminum	170 - 2760	1253	7/7
Barium	17.80 - 82.10	39.51	7/7
Beryllium	0.61 - 2.10	1.60	3/7
Calcium	20100 - 190000	91971	7/7
Chromium	2.40 - 14.60	8.58	6/7
Copper	7.30 - 12.70	9.24	7/7
Iron	354 - 4950	1985	7/7
Lead	2.70 - 27.10	9.90	7/7
Magnesium	727 - 3290	1895	7/7
Manganese	6.60 - 51.10	24.50	7/7
Nickel	6.90	6.90	1/7
Potassium	1040 - 106000	27525	7/7
Silver	1.80 - 2.20	2	2/7
Sodium	7710 - 32900	11638	7/7
Vanadium	4 - 11.20	7.82	4/7
Zinc	44.50 - 106	75.47	7/7
<u>Volatile Organic Chemicals (µg/L)</u>			
Acetone	6 - 19	12.75	4/7
Benzene	2 - 27	10.66	3/7
Carbon Disulfide	9 - 22	13.75	4/7
1,2-Dichloroethylene (Total)	11 - 12	11.50	2/7
Ethyl Benzene	0.70 - 2	1.35	2/7
Toluene	1 - 31	11	3/7
Vinyl Chloride	12	12	1/7
Xylene	1 - 8	3.66	3/7
<u>Semivolatile Organic Chemicals (µg/L)</u>			
Acenaphthene	1 - 5	3	2/7
Bis(2-ethylhexyl)phthalate	1 - 2	1.66	3/7
2-Methylnaphthalene	2 - 9	5.50	2/7
Naphthalene	56 - 270	163	2/7

^a = Number of samples in which the chemical was positively detected over the number of samples collected.

Source: ESE, 1991

Table 2-3. Inorganic, Pesticide, Volatile and Semivolatile Chemicals Identified in Deep Wells from HPIA.

Chemical/Units	Range	Mean	Frequency of Detection ^a
<u>Inorganics (µg/L)</u>			
Aluminum	105 - 2200	669.83	6/6
Antimony	13.50	13.50	1/6
Arsenic	1.60 - 5.60	3.60	2/6
Barium	7.60 - 235	57.75	6/6
Beryllium	0.89	0.89	1/6
Calcium	36100 - 120000	66216	6/6
Chromium	2.50 - 10.30	5.96	5/6
Copper	4.60 - 12.60	8.50	6/6
Iron	149 - 23700	4746	6/6
Lead	1.20 - 3.90	2.20	4/6
Magnesium	131 - 2150	1221	6/6
Manganese	3.80 - 65.40	30.12	5/6
Nickel	6 - 9.60	7.80	2/6
Potassium	1160 - 63400	19525	6/6
Silver	2.50	2.50	1/6
Sodium	6440 - 39100	17238	6/6
Vanadium	6.20 - 7.30	6.75	2/6
Zinc	34.30 - 87.40	51.60	6/6
<u>Volatile Organic Chemicals (µg/L)</u>			
Acetone	4 - 27	14.60	3/6
2-Butanone	5	5	1/6
Carbon Disulfide	4 - 6	4	2/6
Ethyl Benzene	12	12	1/6
Methylene Chloride	0.80 - 2	3.66	3/6
Toluene	34	34	1/6
Xylene	51	51	1/6
<u>Semivolatile Organic Chemicals (µg/L)</u>			
Bis(2-ethylhexyl)phthalate	2 - 3	2.50	2/6

^a = Number of samples in which the chemical was positively detected over the number of samples collected.

Source: ESE, 1991

2.1.1.3 Water Supply Wells

Water supply wells were sampled during the 1991 field investigation (Figure 2-5). The wells include 601 (replaced and renumbered as 660), 602, 603, 608, 630, 634, 637, 642, and 652. Water supply well 642 was considered to be representative of background concentrations because it was the closest active well to HPIA (ESE, 1988; 1991).

Water supply well samples were analyzed for full TCL parameters and in-field measurements of pH, specific conductance, and temperature. Locations of the water supply wells and monitoring wells are shown in Figure 2-5. Results of the chemical analyses are shown in Table 2-4 and the results of the in-field water quality measurements are presented in the RI document.

2.1.2 ESTABLISH A SET OF CHEMICAL/SITE-SPECIFIC EVALUATION CRITERIA

Establishing chemical/site-specific evaluation criteria is part of the second step in selecting COCs and involves:

- 1) examining historical information to identify the types of chemicals reliably associated with site activities;
- 2) identifying chemicals that are potentially carcinogenic (i.e., benzene) as indicated by their weight-of-evidence (WoE) classification (Tables 2-5 and 2-6);
- 3) evaluating chemicals for their mobility, persistence, frequency of detection (Tables 2-1 to 2-4), and their bioaccumulation potential in the environment;
- 4) considering exposure to chemicals through special routes (i.e., some chemicals are highly volatile and may pose significant inhalation risk due to the home use of contaminated water, particularly for showering [EPA, 1989a]);
- 5) evaluating the treatability of chemicals since some chemicals are more difficult to treat than others during remediation; and
- 6) identifying chemicals that exceed Applicable or Relevant and Appropriate Requirements (ARARS) (i.e., drinking water standards) (Table 2-7), site-specific, or literature derived background values (Table 2-8).

A list of the inorganic and organic chemicals detected in HPIA intermediate and deep groundwater and surface soil samples is presented in Tables 2-1 through 2-4, as are the minimum and maximum concentrations, and the frequency of detection in each media sampled. The maximum concentration is the highest quantified concentration within each medium. The frequency of detection presents the number of positive values versus the total number of samples for each chemical in each medium.

2.1.3 DETERMINATION OF CONCENTRATION-TOXICITY EVALUATION CRITERIA

As part of the second step in selecting COCs, a concentration-toxicity screen was performed on the analytical database to provide toxicity ranking values for each chemical detected at the site. This screening process consists of three steps: 1) calculating individual scores for each chemical in the medium of concern (surface soil and groundwater); 2) calculating total chemical scores for each medium; and 3) eliminating chemicals from the final list of COCs based on an evaluation of chemical scores and chemical/site-specific selection criteria (EPA, 1989).

HADNOT PT RA 8/91 LB

SCALE

300 0 300 600 FEET

100 0 100 200 METERS

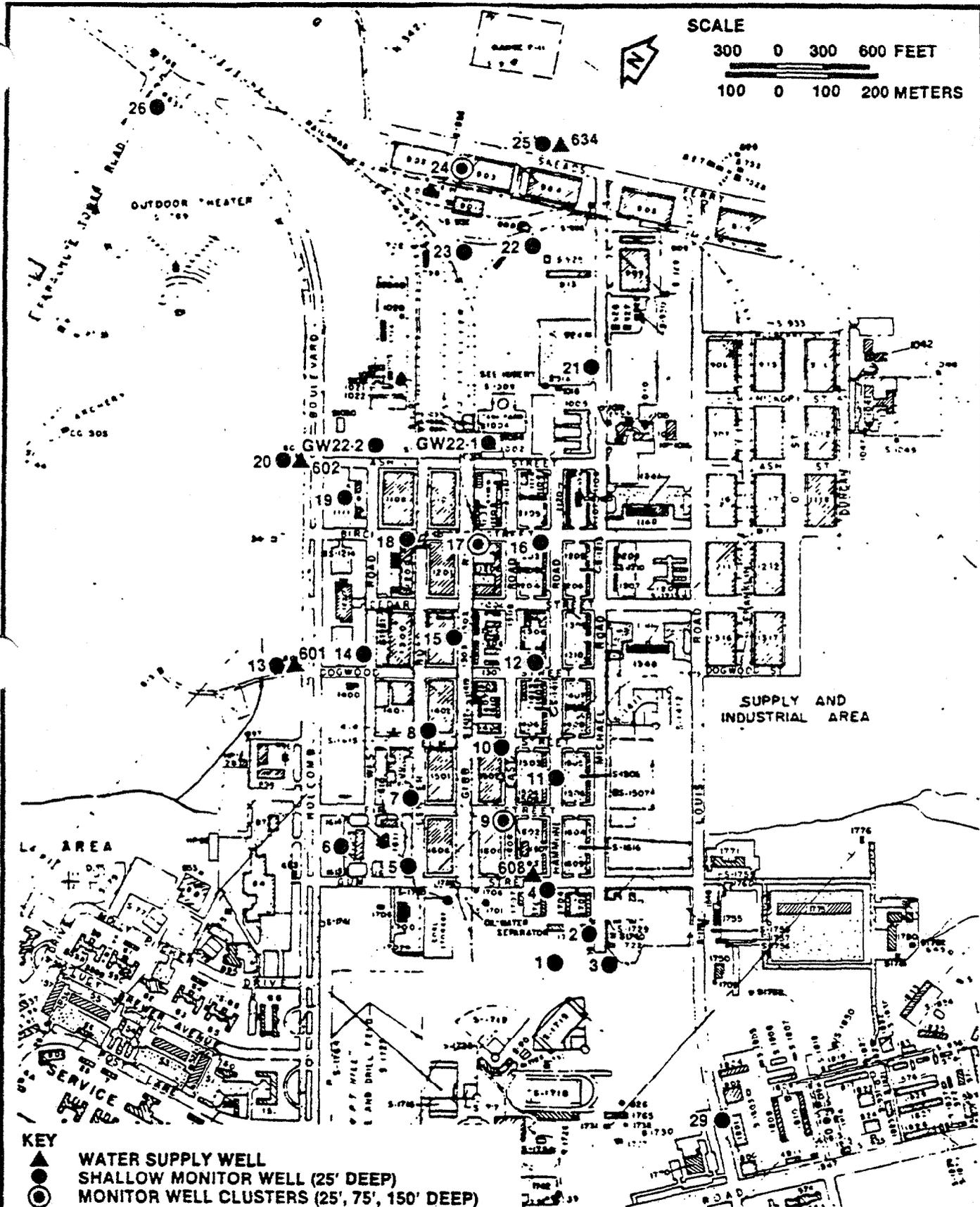


Figure 2-5
MONITORING WELLS AND WATER
SUPPLY WELLS AT HADNOT POINT
INDUSTRIAL AREA

SOURCE: ESE, 1987.



MARINE CORPS BASE
CAMP LEJEUNE

Table 2-4. Inorganic, Pesticide, Volatile and Semivolatile Chemicals Identified in Water Supply Wells from the Hadnot Point Area.

Chemical/Units	Range	Mean	Frequency of Detection ^a	Background Levels (642) ^b
<u>Inorganics ($\mu\text{g/L}$)</u>				
Aluminum	95.20	95.20	1/6	BDL
Barium	4.80 - 376	69.92	6/6	7.60
Calcium	58900 - 128000	83650	6/6	74100
Chromium	1.70	1.70	1/6	BDL
Copper	4.90 - 97.10	29.48	5/6	8.50
Iron	1030 - 65000	16062	6/6	1150
Lead	3.30 - 32.80	16.67	4/6	BDL
Magnesium	1190 - 5440	2705	6/6	1690
Manganese	12.50 - 151	68.26	6/6	24.60
Potassium	890 - 2620	1703	6/6	1390
Silver	2.20	2.20	1/6	BDL
Sodium	5410 - 12500	9036	6/6	7730
Vanadium	2.40 - 2.70	2.55	2/6	BDL
Zinc	23.40 - 18100	3825	6/6	38.60
<u>Volatile Organic Chemicals ($\mu\text{g/L}$)</u>				
Benzene	17	17	1/6	BDL
1,2-Dichloroethane	8	8	1/6	BDL
1,2-Dichloroethylene (Total)	1 - 12	5	3/6	BDL
Methylene Chloride	20 - 21	20.50	2/6	BDL
Trichloroethene	0.70 - 1	0.90	4/6	BDL
<u>Semivolatile Organic Chemicals ($\mu\text{g/L}$)</u>				
Bis(2-ethylhexyl)phthalate	3	3	1/6	BDL

^a = Number of samples in which the chemical was positively detected over the number of samples collected.

^b = Background sample collected from water supply well 642.

Source: ESE, 1991

Table 2-5. Health Effects Assessment of Potential Chemicals of Concern for Hadnot Point Industrial Area (Carcinogenicity: Subchronic and Chronic Toxicity) (Page 1 of 5).

Chemical	Carcinogenicity Classification		Slope Factor (ug/L) ⁻¹ or [mg/kg/day ⁻¹]		Inhalation RfC mg/m ³ (mg/kg/day ⁻¹)		Oral RfD ^a (mg/kg/day ⁻¹)		
	Inhalation	Oral	Inhalation	Oral	Subchronic ^c	Chronic ^c	Subchronic ^c	Chronic ^c	
<u>Inorganic</u>									
Aluminum	NA	NA	NA	NA	NA	NA	NA	NA	NA
Antimony					ND	ND	4E - 4	4E - 4	
Arsenic	A	A	4.3E - 3 [50]	1.75 ^d	ND	ND	1E - 3	1E - 3	
Barium					5E - 3	5E - 4	5E - 2	5E - 2	
Beryllium	B2	B2	2.4E - 3	1.2E - 4					
Cadmium	B1	ND	1.8E - 3 [6.1]	ND	ND	ND	ND	5E - 4	
Calcium									
Chromium									
Cobalt									
Copper					ND	ND	1.3 mg/l	1.3 mg/l	
Iron	NA	NA	NA	NA	NA	NA	NA	NA	NA
Lead	B2	B2	ND	ND	ND	ND	5E - 4 ^b	ND	
Magnesium									
Manganese					4E - 4	4E - 4	1E - 1	1E - 1	

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Table 2-5. Health Effects Assessment of Potential Chemicals of Concern for Hadnot Point Industrial Area (Carcinogenicity: Subchronic and Chronic Toxicity) (Page 2 of 5).

Chemical	Carcinogenicity Classification		Slope Factor (ug/L) ⁻¹ or [mg/kg/day ⁻¹]		Inhalation RfC mg/m ³ (mg/kg/day ⁻¹)		Oral RfD (mg/kg/day ⁻¹)		
	Inhalation	Oral	Inhalation	Oral	Subchronic ^c	Chronic ^c	Subchronic ^c	Chronic ^c	
Nickel	A	ND	2.4E - 4	ND	ND	ND	2E - 2	2E - 2	
Potassium									
Selenium									
Silver					ND	ND	3E - 3	3E - 3	
Sodium									
Vanadium					ND	ND	7E - 3	7E - 3	
Zinc					ND	ND	2E - 1	2E - 1	
<u>Pesticides</u>									
Aroclor 1254	B2	B2							
Aroclor 1260	B2	B2	ND	2.2E - 4 [7.7]					
Dieldrin	B2	B2	4.6E - 3 [16]	4.6E - 4 [16]	ND	ND	5E - 5	5E - 5	
4,4-DDE	B2	B2	ND	9.7E - 6 [0.34]					
4,4-DDT	B2	B2	9.7E-5 [0.34]	9.7E-6 [0.34]	ND	ND	5E - 4	5E - 4	

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Table 2-5. Health Effects Assessment of Potential Chemicals of Concern for Hadnot Point Industrial Area (Carcinogenicity: Subchronic and Chronic Toxicity) (Page 3 of 5).

Chemical	Carcinogenicity Classification		Slope Factor (ug/L) ⁻¹ or [mg/kg/day ⁻¹]		Inhalation RfC mg/m ³ (mg/kg/day ⁻¹)		Oral RfD (mg/kg/day ⁻¹)	
	Inhalation	Oral	Inhalation	Oral	Subchronic ^c	Chronic ^c	Subchronic ^c	Chronic ^c
<u>Volatile Organic Chemicals</u>								
Acetone					ND	ND	6E - 1	6E - 2
Benzene	A	A	8.3E-6 [0.03]	8.3E-7 [0.03]				1E + 2 ^a
2-Butanone								
Carbon Disulfide					1E - 2	1E - 2	1E - 1	1E - 1
1,2-Dichloroethane	B2	B2	2.6E-5 [0.09]	[0.091]			1E + 0	1E - 1
1,2-Dichloroethene (tot)					ND	ND	1E - 1	1E - 2
Ethyl Benzene					1E + 0	1E + 0	1E + 0	1E - 1
Methylene Chloride	B2	B2	4.7E - 7	2.1E - 7 [0.0075]	3E + 0	3E + 0	6E - 2	6E - 2
Toluene					2E + 0	2E + 0	2E - 0	2E - 1
Trichloroethene	B2	B2		5.1E - 2				
Vinyl Chloride	A	A	8.4E - 5	5.4E - 5 [1.9]				
Xylene (total)					3E - 1	3E - 1	4 E + 0	2E + 0

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Table 2-5. Health Effects Assessment of Potential Chemicals of Concern for Hadnot Point Industrial Area (Carcinogenicity: Subchronic and Chronic Toxicity) (Page 4 of 5).

Chemical	Carcinogenicity Classification		Slope Factor (ug/L) ⁻¹ or [mg/kg/day ⁻¹]		Inhalation RfC mg/m ³ (mg/kg/day ⁻¹)		Oral RfD (mg/kg/day ⁻¹)	
	Inhalation	Oral	Inhalation	Oral	Subchronic ^c	Chronic ^c	Subchronic ^c	Chronic ^c
<u>Semi-Volatile Organic Chemicals</u>								
Accenaphthene					ND	ND	6E - 1	6E - 2
Anthracene					ND	ND	3E + 0	3E - 1
Benzo(a)-anthracene	B2	B2	NA	ND				
Benzo(a)pyrene	B2	B2	1.7E-3 [6.1]	3.3E-4 [11.5]				
Benzo(b)-flouranthene	B2	B2	ND	ND				
Benzo(k)-flouranthene	B2	B2	ND	ND				
Benzo(g,h,i)perylene								
Bis(2 ethyl-hexyl)phthalate	B2	B2	ND	4E - 7 [0.014]	ND	ND	2E - 2	2E - 2
Chrysene	B2	B2	ND	ND				
Dibenzofuran	NA	NA	NA	NA	NA	NA	NA	NA
Di-n-butyl phthalate					ND	ND	1E + 0	1E - 1
1,4-Dichlorobenzene					ND	ND	4E - 1	4E - 2

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Table 2-5. Health Effects Assessment of Potential Chemicals of Concern for Hadnot Point Industrial Area (Carcinogenicity: Subchronic and Chronic Toxicity) (Page 5 of 5).

Chemical	Carcinogenicity Classification		Slope Factor ($\mu\text{g/L}$) ⁻¹ or [mg/kg/day] ⁻¹		Inhalation RfC mg/m^3 (mg/kg/day) ⁻¹		Oral RfD (mg/kg/day) ⁻¹	
	Inhalation	Oral	Inhalation	Oral	Subchronic ^c	Chronic ^c	Subchronic ^c	Chronic ^c
Fluoranthene					ND	ND	4E - 1	4E - 2
Fluorene					ND	ND	4E - 1	4E - 2
Indeno(1,2,3-cd) pyrene	B2	B2	ND	ND				
Naphthalene					ND	ND	4E - 2	4E - 3
2-Methylnaphthalene								
Phenanthrene								
Pyrene					ND	ND	3E - 1	3E - 2

Group A = Human Carcinogen.

Group B = Probably Human Carcinogen; B1 = limited evidence of carcinogenicity, B2 = sufficient evidence of carcinogenicity in animals with lack of evidence in humans.

Group C = Possible Human Carcinogen.

Note: ND = Not detected.

Source: EPA, 1991.

* calculated using a unit risk of $5\text{E}-5(\mu\text{g/L})^{-1}$ (EPA, 1991).

a RfD is reported based upon human oral TD_{10} (Layton et al. 1987).

b Provisional RfD based on proposed MCL of 0.005 mg/L and assumes that a healthy 10kg child consumes 1L/day water (Layton et al. 1987).

c Data extracted from EPA IRIS program or Health Effects Assessment Summary Tables

d The Carcinogenic Slope Factor (CSF) for benzo(a)pyrene (BaP) is used for all carcinogenic PAHs (polynuclear aromatic hydrocarbons). Also, the reference dose (RfD) for pyrene is used for all non-carcinogenic PAHs without a RfD.

e While sub-chronic RfDs are listed, only chronic RfDs are used in the

Table 2-6. Weight-of-Evidence Categories for Potential Carcinogens.

EPA Category	Description of Group	Description of Evidence
Group A	Human carcinogen	Sufficient evidence from epidemiologic studies to support a causal association between exposure and cancer.
Group B1	Probable human carcinogen	Limited evidence of carcinogenicity in humans from epidemiologic studies.
Group B2	Probable human carcinogen	Sufficient evidence of carcinogenicity in animals but inadequate data in humans.
Group C	Possible human carcinogen	Limited evidence of carcinogenicity in animals.
Group D	Not classified	Inadequate evidence of carcinogenicity in animals.
Group E	No evidence of carcinogenicity in humans	No evidence of carcinogenicity in at least two adequate animal tests or in both epidemiologic and animal studies.

Source: EPA, 1991.

Table 2-7. Comparison of Promulgated Standards in Various Media (µg/L) (Page 1 of 2).

Chemical	Federal Fresh Water Quality Standards		North Carolina Water Quality Standards		Federal Marine Water Quality Standards		Federal Water Quality Standards	Safe Water Drinking Water Act MCL
	Acute	Chronic	Fresh Water	Marine	Acute	Chronic	Water & Fish	
<u>Inorganics</u>								
Aluminum	NS	NS	NS	NS	NS	NS	NS	MCLs/MCLGs are found in Appendix D
Antimony	9000	1600	NS	NS	NS	NS	146	
Arsenic (Total)	NS	NS	50	50	2319	13	0.0022	
Barium	NS	NS	NS	NS	NS	NS	1000	
Beryllium	130	5.3	6.5	NS	NS	NS	0.0068	
Cadmium	HD	HD	2.0	5.0	43	9.3	10	
Calcium	NS	NS	NS	NS	NS	NS	NS	
Chromium (Total)	HD	HD	50	20	1100	50	50	
Cobalt	NS	NS	NS	NS	NS	NS	NS	
Copper	HD	7.0 ¹	NS	2.9	2.9	NS	1300	
Cyanide	22	5.2	5.0	1.0	1.0	1.0	200	
Iron	NS	NS	1000 ¹	NS	NS	NS	300	
Lead	HD	HD	25	25	140	5.6	50	
Magnesium	NS	NS	NS	NS	NS	NS	NS	
Manganese	NS	NS	NS	NS	NS	5	50	
Mercury	2.4	0.012	0.012	0.025	2.1	0.025	0.000144	
Nickel	HD	HD	NS	NS	75	8.3	13.4	
Potassium	NS	NS	NS	NS	NS	NS	NS	
Selenium	260	35	NS	NS	410	54	10	
Silver	HD	HD	0.06 ¹	NS	2.3	NS	50	
Sodium	NS	NS	NS	NS	NS	NS	NS	
Thallium	1400	40	NS	NS	2130	NS	13	
Vanadium	NS	NS	NS	NS	NS	NS	NS	
Zinc	HD	HD	50 ¹	NS	95	86	NS	

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Table 2-7. Comparison of Promulgated Standards in Various Media ($\mu\text{g/L}$) (Page 2 of 2).

Chemical	Federal Fresh Water Quality Standards		North Carolina Water Quality Standards		Federal Marine Water Quality Standards		Federal Water Quality Standards	Safe Water Drinking Water Act MCL
	Acute	Chronic	Fresh Water	Marine	Acute	Chronic	Water & Fish	
Organics								
1 2-DCA	118000	20000	NS	NS	113000		0.94	
1 2-DCE	11600	NS	NS	NS	224000	NS	NS	
Acenaphthene	1700	520	NS	NS	970	710	NS	
Benzene	5300	NS	NS	NS	5100	700	0.66	
Di-n-butylphthalate	NS	NS	NS	NS	NS	NS	35 mg	
Dichlorobenzene	1120	763	NS	NS	1970	NS	400	
Ethyl Benzene	32000	NS	NS	NS	430	NS	1.4 mg	
Fluoranthene	3980	NS	NS	NS	40	16	42	
Methylene Chloride	NS	NS	NS	NS	300	NS	2.8 mg	
Naphtalene	2300	620	NS	NS	2350	NS	NS	
Tetrachloroethene	5280	840	NS	NS	2130	NS	13	
Toluene	17500	NS	NS	NS	6300	5000	14.30	
Trichloroethene	45000	21900	NS	NS	2000	NS	2.70	
Vinyl Chloride	NS	NS	NS	NS	NS	NS	2.00	
Xylene	NS	NS	NS	NS	NS	NS	1.4 mg	
Pesticides								
DDE	1050	NS	NS	NS	14		NS	
DDT	1.10	0.001	NS	NS	0.13	0.001	0.024	
Dieldrin	2.5	0.0019	NS	NS	0.71	0.0019	0.071	
PCB's	2.0	0.014	NS	NS	10	0.03	0.079	

NS = No Standard

HD = Hardness Dependent

¹ = Action Level, Not a Standard

* = Promulgated Criterion (enforceable criterion) under the National Primary Drinking Water Regulations

Source: SDWA, 1989 and NCWQS, 1990.

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Table 2-8. Literature Derived Values of Background Concentrations of Inorganic Chemicals in Soil for the United States (expressed in $\mu\text{g/g}$ or percent^a).

Analyte	<u>Eastern United States (east of 96th meridian)^a</u>		<u>Conterminous United States^a</u>	
	Average	Range	Average	Range
Aluminum	3.30*	0.70 - >10*	72,000	700 - <10,000
Arsenic	4.80	<0.10 - 73	7.20	<0.10 - 97
Barium	290	10 - 1500	580	10 - 5000
Beryllium	0.55	<1 - 7	0.92	<1 - 15
Cadmium	NA	NA	NA	NA
Calcium	0.34*	0.01 - 28*	24000	100 - 320000
Chromium	33	1 - 1000	54	1 - 2000
Cobalt	5.90	<0.30 - 70	9.10	<3 - 70
Copper	13	<1 - 700	25	<1 - 700
Iron	1.40*	0.01 - >10*	26000	100 - >100000
Lead	14	<10 - 300	19	<10 - 700
Magnesium	0.21*	0.005 - 5*	9000	50 - >100000
Manganese	260	<2 - 7000	550	<2 - 7000
Mercury	0.081	0.01 - 3.40	0.09	<0.01 - 4.60
Nickel	11	<5 - 700	19	<5 - 700
Potassium	1.20*	0.005 - 3.70*	15000	50 - 63000
Selenium	0.30	<0.10 - 3.90	0.39	<0.10 - 4.30
Silver	NA	NA	NA	NA
Sodium	0.25*	<0.05 - 5*	12000	<500 - 100000
Thallium	7.70	2.20 - 23	9.40	2.20 - 31
Vanadium	43	<7 - 300	80	<7 - 500
Zinc	40	<5 - 2900	60	<5 - 2900
Cyanide	NA	NA	NA	NA

a = Values were derived from (Boerngen, J.G. and H.T. Shacklett, 1984).

* = Values expressed as percent.

Source: Boerngen, J.G. and H.T. Shacklett, 1984.

The individual score of a chemical is based on its concentration and toxicity. Risk factors are calculated separately for noncarcinogenic and carcinogenic compounds by multiplying the maximum detected concentration of the chemical in a medium by its corresponding toxicity value, which is the reciprocal of the Reference Dose (1/RfD) for noncarcinogens or by the cancer slope factor (CSF) for the carcinogens. The RfDs and CSFs of potential COCs are presented Table 2-5. Chemical-specific scores are summed for each medium to obtain the total risk factor for all potential COCs in a medium. Separate sums are obtained for carcinogenic and noncarcinogenic effects for each medium and are summarized in Tables 2-9 and 2-10, respectively. The ratio of the chemical score for each chemical to the total chemical score approximates the relative risk for each chemical in a medium (EPA, 1989).

Once the chemical scores are determined for each potential COC, the chemicals that contribute less than 1 percent of the overall total score (a lower fraction would be required if the site risks are high) may be eliminated from consideration for further analysis in the RA. Chemicals without toxicity values, such as aluminum, 2-butanone, and benzo(a)anthracene (Table 2-2), cannot be screened using this procedure and are evaluated separately in the RA as potential COCs by considering site-specific criteria such as drinking water criteria, frequency of detection, and toxicity (Section 2.1.2). In general, a majority of the chemicals for which no RfDs or CSFs have been determined, are represented by one of the COCs that are from the same class (inorganic or organic).

2.2 FINAL LIST OF CHEMICALS OF CONCERN

The final list of COCs for the HPIA RA was determined by evaluating the two sets of evaluation criteria the chemical/site-specific evaluation criteria and the concentration-toxicity ranking values. The primary criteria for selecting COCs were toxicity and frequency of detection in surface soil, intermediate and deep groundwater at the site, (as has already been presented in Tables 2-1, 2-2, and 2-3). Several chemicals were also selected based on chemical/site-specific criteria, such as carcinogenicity, mobility, persistence, bioaccumulation potential, or exceedance of an ARAR.

Based on the evaluation of the two sets of selection criteria (chemical/site-specific criteria and concentration-toxicity scores) the final list of COCs at the HPIA include Polynuclear Aromatic Hydrocarbons (PAHs) (both noncarcinogenic and carcinogenic), benzene, 1,2-dichloroethene (total), and lead. These COCs were selected to represent the volatile and inorganic contaminants in the RA for the HPIA (Table 2-11) and represent the most toxic, persistent, mobile, and prevalent contaminants at the four areas of concern.

The following sections present summaries of the rationale for the selection or exclusion of site related contaminants as COCs.

Table 2-9. Chemical Toxicity Scores Derived for Carcinogenic Potential Chemicals of Concern Identified in Surface Soil, Intermediate, and Deep Groundwater for HPIA.

Chemical	Slope Factor (mg/kg/day) ⁻¹ or µg/m ³ *	Maximum Concentration			Chemical Score		
		Inter. GW	Deep GW	Soil	Inter. GW	Deep GW	Soil
Arsenic	1.75			1.40			
*Beryllium	0.00012	2.10	0.89		0.0002	0.0001	
Aroclor (1254 & 1260)	7.70			780			6006
DDT and DDE	0.34			97			33
Dieldrin	16			92			147
Benzene	0.03	27			0.78		
Bis(2-ethylhexyl) phthalate	0.014	2	3	54	0.028	0.042	0.76
Methylene Chloride	0.0075		2	14		0.015	0.11
Vinyl Chloride	1.90	12			22.80		
Benzo(a)pyrene	11.5			240			2760
TOTAL SCORE					23.60	0.057	8747

GW = Groundwater

Source: EPA, 1991; ESE, 1991.

Table 2-10. Chemical Toxicity Scores Derived for Noncarcinogenic Potential Chemicals of Concern Identified in Surface Soil, Intermediate and Deep Groundwater for HPIA.

Chemical	RfD (mg/kg/day) ⁻¹	Maximum Concentration			Chemical Score		
		Inter. GW	Deep GW	Soil	Inter. GW	Deep GW	Soil
Antimony	0.0004		13.50	9.60		33750	24000
Arsenic	0.001		5.60	1.40		5600	1400
Barium	0.05	82.10	235	19.60	1642	4700	392
Cadmium	0.0005			3			6000
Copper	1.3	12.70	12.60	11.80	9.77	9.69	9
Manganese	0.1	51.10	65.40	155	511	654	1550
Nickel	0.02	6.90	9.60	5.80	345	480	290
Silver	0.003	2.20	2.50	1.10	733	833	367
Vanadium	0.007	11.20	7.30	7.40	1600	1043	1057
Zinc	0.2	106	87.40	61.20	530	437	306
Acetone	0.06	19	27	360	316	450	6000
1,2-Dichloroethene	0.01	12			1200		
Carbon Disulfide	0.1	22		6	220		60
Ethylbenzene	0.1	2		12	20		120
Toluene	0.2	31		34	155		170
Xylene	2.0	8		51	4		25
Acenaphthene	0.06	5		42	83		700
Anthracene	0.3			180			600
Di-n-butylphthalate	0.1			72			720
Fluoranthene	0.04			690			17250
Fluorene	0.04			63			1575
Naphthalene	0.004	270		220	67500		55000
Pyrene	0.03			580			19333
TOTAL SCORE					74870	47957	136925

GW = Groundwater

Source: EPA, 1991; ESE, 1991.

Table 2-11. Chemicals of Concern by Area of Concern and Media (Page 1 of 2).

Area of Concern	Chemical of Concern*	Media in which Chemical was Detected		
		Surface Soil	Groundwater ^a	
902	Lead	X	X	
	Benzene	ND	X	
	1,2-DCE	ND	X	
	<u>PAHs</u>			
	Acenaphthene	X	X	
	Anthracene	X	ND	
	Benzo(a)anthracene	X	ND	
	Benzo(b)fluoranthene	X	ND	
	Benzo(k)fluoranthene	X	ND	
	Benzo(g,h,i)perylene	X	ND	
	Benzo(a)pyrene	X	ND	
	Chrysene	X	ND	
	Fluoranthene	X	ND	
	Flourene	X	ND	
	Indeno(1,2,3cd)pyrene	X	ND	
	2-Methylnaphthalene	ND	X	
	Naphthalene	ND	X	
	Phenanthrene	X	ND	
	Pyrene	X	ND	
	1202	Lead	X	X
Benzene		ND	ND	
1,2-DCE		ND	X	
<u>PAHs</u>				
Acenaphthene		X	X	
Anthracene		X	ND	
Benzo(b)fluoranthene		X	ND	
Benzo(k)fluoranthene		X	ND	
Benzo(g,h,i)perylene		X	ND	
Benzo(a)pyrene		X	ND	
Chrysene		X	ND	
Flouranthene		X	ND	
Flourene		X	ND	
Indeno(1,2,3cd)pyrene		X	ND	
2-Methylnaphthalene		ND	X	
Naphthalene		ND	X	
Phenanthrene		X	ND	
Pyrene		X	ND	
1602		Lead	X	X
		Benzene	ND	ND
	1,2-DCE	ND	X	

Table 2-11. Chemicals of Concern by Area of Concern and Media (Page 2 of 2).

Area of Concern	Chemical of Concern*	Media in which Chemical was Detected	
		Surface Soil	Groundwater ^a
	<u>PAHs</u>		
	2-Methylnaphthalene	X	ND
	Naphthalene	X	ND
Site 22 ^b	Lead	NA	X
	Benzene	NA	X

NA = Not Available, see footnote b.

ND = Not Detected.

X = Chemical was identified as a chemical of concern.

* = Based on all selection criteria and concentration-toxicity screen.

a = Intermediate and Deep groundwater data were combined.

b = Soil Samples were not collected for Site 22.

Source : ESE, 1991

2.2.1 INORGANIC CHEMICALS

The inorganic compounds most frequently detected at the site were:

Aluminum	Iron	Sodium
Copper	Potassium	Calcium
Magnesium	Barium	Manganese
Antimony	Lead	Zinc

Lead was chosen as the COC to represent the heavy metal compounds due to its potential toxicity and frequency of detection in both soil and groundwater. All other inorganics were excluded because of low frequency, low toxicity, or concentrations below water quality criteria and MCLs. Most metals had elevated concentrations as compared to national averages (Table 2-8), some due to naturally geologic conditions. Concentrations of calcium, potassium, sodium, and magnesium in soils and groundwater were not regarded as COCs since they are common elements in the area.

2.2.2 PESTICIDES

Although pesticides were detected in several soil samples, they were disregarded as COCs for a variety of reasons. The levels observed for the PCBs fell below the 1.0 parts per million (ppm) concentrations regarded as hazardous. According to the Toxic Substances Control Act (TSCA) guidance for the cleanup of PCB levels in soil, concentrations of 10 to 25 ppm are considered acceptable for industrial and 1 ppm for residential exposures (Federal Register, 40 CFR Ch. 1 7-1-87 edition). Guidance notes that the industrial remediation goals, 10 to 25 ppm, are protective of human health "even assuming exposure equivalent to that in residential areas".

Although the chemical toxicity scores (CTS) for pesticides were high, the frequency of detection was low. Historically, these chemicals were not disposed on HPIA, therefore, detection of these chemicals could be due to applications for weed control or aerial contamination from Site 21, a storage yard for transformers. Pesticides in the HPIA were not considered a threat to human health or environmental receptors.

2.2.3 SEMIVOLATILE ORGANIC CHEMICALS

Of the semivolatile organic chemicals detected at the site, the PAHs were chosen as COCs based on the CTS ranking (Tables 2-9 and 2-10) associated with noncarcinogenic and carcinogenic effects. PAHs were chosen based on toxicity (several PAHs may be potent carcinogens), the chemical and physical properties (many PAHs are persistent), and history of use and disposal at the site (vehicle maintenance).

Because the number of compounds in this class and the lack of toxicological information on specific compounds, the PAHs were discussed as two groups: noncarcinogenic and potentially carcinogenic. The potentially carcinogenic PAHs detected at the site include:

Benzo(a)anthracene	Benzo(a)pyrene
Benzo(b)fluoranthene	Chrysene
Benzo(k)fluoranthene	Indeno(1,2,3-cd)pyrene

Since available toxicological data are inadequate to completely characterize each of the compounds in the potentially carcinogenic group of PAHs, the approach used by EPA for developing Ambient Water Quality Criteria (AWQC) for PAHs (EPA, 1980d) was applied in the RA. The EPA approach developed criteria for an individual carcinogenic PAH, specifically BaP, that would also lead to effective control of the other chemicals in this group. Data indicate that BaP is one of the most potentially carcinogenic PAHs. The EPA AWQC for PAHs is based on the assumption that each compound is as potent a carcinogen as BaP and that the carcinogenic effect of the compounds proportional to the sum of their concentrations. BaP is the only potentially carcinogenic PAH for which adequate dose-response data are available for the oral exposure route. EPA (1980) concludes, therefore, that cumulative exposures to mixtures containing PAHs should result in a risk less than that predicted for BaP alone. Until the cancer potencies of individual PAHs have been determined and finalized by EPA, BaP was selected to represent the potentially carcinogenic PAHs (as a class) for risk calculation purposes.

The noncarcinogenic PAHs detected at Hadnot Point include:

Acenaphthene	Fluoranthene	2-Methylnaphthalene
Anthracene	Fluorene	Phenanthrene
Benzo(g,h,i)perylene	Naphthalene	Pyrene

Although naphthalene and 2-methylnaphthalene belong to the noncarcinogenic class of PAHs, these chemicals were evaluated separately due to their different physicochemical properties. The physicochemical properties of naphthalene include high water solubility and low Koc (soil sorption constant) (EPA, 1989)

2.2.4 VOLATILE ORGANIC CHEMICALS

Based on the analytical results of the 1991 field investigations performed by ESE, VOCs identified in groundwater and surface soils include:

Acetone	1,2-Dichloroethane	Toluene
Benzene	1,2-Dichloroethylene	Trichloroethene
2-Butanone	Ethyl Benzene	Vinyl Chloride
Carbon Disulfide	Methylene Chloride	Xylene

The results indicate that most of these chemicals did not occur in soil (except acetone and methylene chloride). Several groundwater samples contained detectable levels of benzene, ethyl benzene, and toluene. The presence of parking areas, the abundance of roads, and the historical use of vehicles can contribute to the presence of these three compounds as a result of urban runoff during storm events.

Because benzene was identified in groundwater within the AOCs at concentrations exceeding water quality criteria and is considered a potential human carcinogen, it was included as a COC for further analysis. The inclusion of benzene in the RA is expected to result in risk estimates that are also protective of the less toxic

ethyl benzene, toluene, and xylene. 1,2-DCE was also included as a COC due to its toxicity. Acetone and methylene chloride were not considered COCs because these chemicals frequently occur as laboratory contaminants and were not historically disposed in the AOCs. The remaining compounds were excluded from COC selection due to low frequency and low toxicity.

2.2.5 SUMMARY OF COCS FOR EACH AREA OF CONCERN AT HPIA

The final list of chemicals of concern and the rationale behind their selection is presented in Table 2-12. These chemicals serve to represent the more hazardous COCs of interest for HPIA. In the event that the subsequent toxicity assessment and risk characterization demonstrate that remediation of the source areas is necessary for reducing the levels of identified COCs to acceptable concentrations, the remediation is also expected to result in acceptable concentrations of other less toxic, less mobile, and less prevalent constituents.

2.2.5.1 Area 902

COCs identified in groundwater from area 902 include: lead, benzene, 1,2-dichloroethene, acenaphthene, 2-methylnaphthalene, and naphthalene. Chemicals identified in soils as potential COCs include lead, acenaphthene, anthracene, benzo(a)anthracene, benzo(b,k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, chrysene, fluoranthene, fluorene, indeno(1,2,3 cd)pyrene, phenanthene, and pyrene.

2.2.5.2 Area 1202

Chemicals identified for potential concern in area 1202 groundwater include lead: 1,2-dichloroethene acenaphthene, 2-methylnaphthalene, and naphthalene. COCs identified in soils include lead, acenaphthene, anthracene, benzo(b,k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, phenanthene.

2.2.5.3 Area 1602

COCs identified in area 1602 groundwater include lead and 1,2-dichloroethene. Lead, 2-methylnaphthalene and naphthalene were identified COCs in area 1602 surface soils.

2.2.5.4 Site 22

COCs identified in Site 22 groundwater include lead and benzene. Surface soils were not sampled from Site 22.

3.0 EXPOSURE ASSESSMENT

3.0 EXPOSURE ASSESSMENT

The exposure assessment utilizes information obtained from the characterization of the exposure setting to identify completed exposure pathways and to estimate actual or potential concentrations of the COCs. Behavioral or physiological factors influencing exposure frequency and exposure levels are then presented in a series of exposure scenarios in order to quantify chemical intake levels by receptor populations for each significant completed exposure pathway. The results of the exposure assessment are used in conjunction with the information summarized in the toxicity assessment (Section 4.0 and Appendix A) to determine the potential human health and environmental risks associated with each area of concern at HPIA.

3.1 CHARACTERIZATION OF EXPOSURE SETTING

3.1.1 PHYSICAL SETTING

The MCB Camp Lejeune complex covers an area of approximately 171 square miles and includes five major activity areas: Marine Corps Base; Marine Corps Air Stations; New River; Naval Hospital; and Naval Dental Clinic. The major commands that occupy the MCB include: Marine Corps Base host; the 2nd Marine Division; 2nd Marine Amphibious Force; and the 2nd Force Service Support Group. The Navy Medical and Dental Commands are separate units that occupy the Complex.

The military complex is located in Onslow County in southeastern North Carolina, approximately 45 miles south of the city of New Bern and 47 miles north of Wilmington. The county seat, as well as the primary commercial center, is the City of Jacksonville, the largest developed area in the county. Jacksonville's southern boundary is adjacent to the northern boundary of the MCB Camp Lejeune. The second largest developed area in Onslow County is West Onslow Beach. There are two smaller county communities, Verona and Sneads Ferry, that are older residential communities, typified by single family mobile home residential growth adjacent to the southern boundary of the MCB Camp Lejeune. The two forest preserves existing in Onslow County, Great Sandy Run Forest and Hofman Forest, represent two large areas of undeveloped land in close proximity to the MCB Camp Lejeune complex.

Of the developed areas in MCB Camp Lejeune, Hadnot Point comprises the most concentrated zone of development. This area includes the organizational offices for the Host Activity and for the Headquarters of the 26 Marine Amphibious Unit, as well as the Headquarters and regimental areas for the 2nd Division of the Marine Corps, 2nd Marine Amphibious Force, 6th Marine Amphibious Brigade, 22nd Marine Amphibious Unit, 24th Marine Amphibious Unit, the Central Exchange and Commissary, and the Naval Dental Clinic Headquarters. Directly north of Hadnot Point are the family housing areas, which are concentrated throughout the wooded areas of the central Complex and along the shores of the New River. Also located in this north central area are major personnel support uses, including the newly-constructed Naval Hospital, school sites,

recreational areas, and additional family housing areas (quarters developments, Midway Park, and Tarawa Terrace I and II).

3.1.1.1 Topography

MCB Camp Lejeune is situated on relatively flat terrain that includes swamps, estuaries, savannas, and forest lands. Land surface elevations range from mean sea level (msl) to 72 feet above msl. Average elevations are between 20 and 40 feet above msl. The surface water drainage at MCB Camp Lejeune is predominantly toward the New River and its tributaries, although coastal areas drain directly to the Atlantic Ocean via the Intercoastal Waterway. Natural drainage has been changed in developed areas with the installation of drainage ditches, storm sewers, and extensive paving. Relatively few areas of steep slopes, defined as those exceeding a ten percent grade, exist at MCB Camp Lejeune.

3.1.1.2 Soils/Surface Hydrology

Soils are generally poorly to very poorly drained. Thirty-one soil types exist throughout MCB Camp Lejeune, ranging from sandy loam to fine sand and mud. The soil type can be classified generally as sandy loam although soil conditions are quite heterogenous. The majority of the soils are well suited to produce abundant crops of timber and forage for wildlife, with only a small proportion of the soils being low in organic matter and fertility.

The principle watershed drainage areas are the New River, Northeast Creek, Southwest Creek, Wallace Creek, French Creek, Rear Creek, Freeman Creek and Duck Creek. Because of the shallow slope and relatively few streams, drainage is the most critical factor determining the suitability of soil for development. The MCB is encompassed by vast areas of old growth timber and swampland that evolved due to these topographic features.

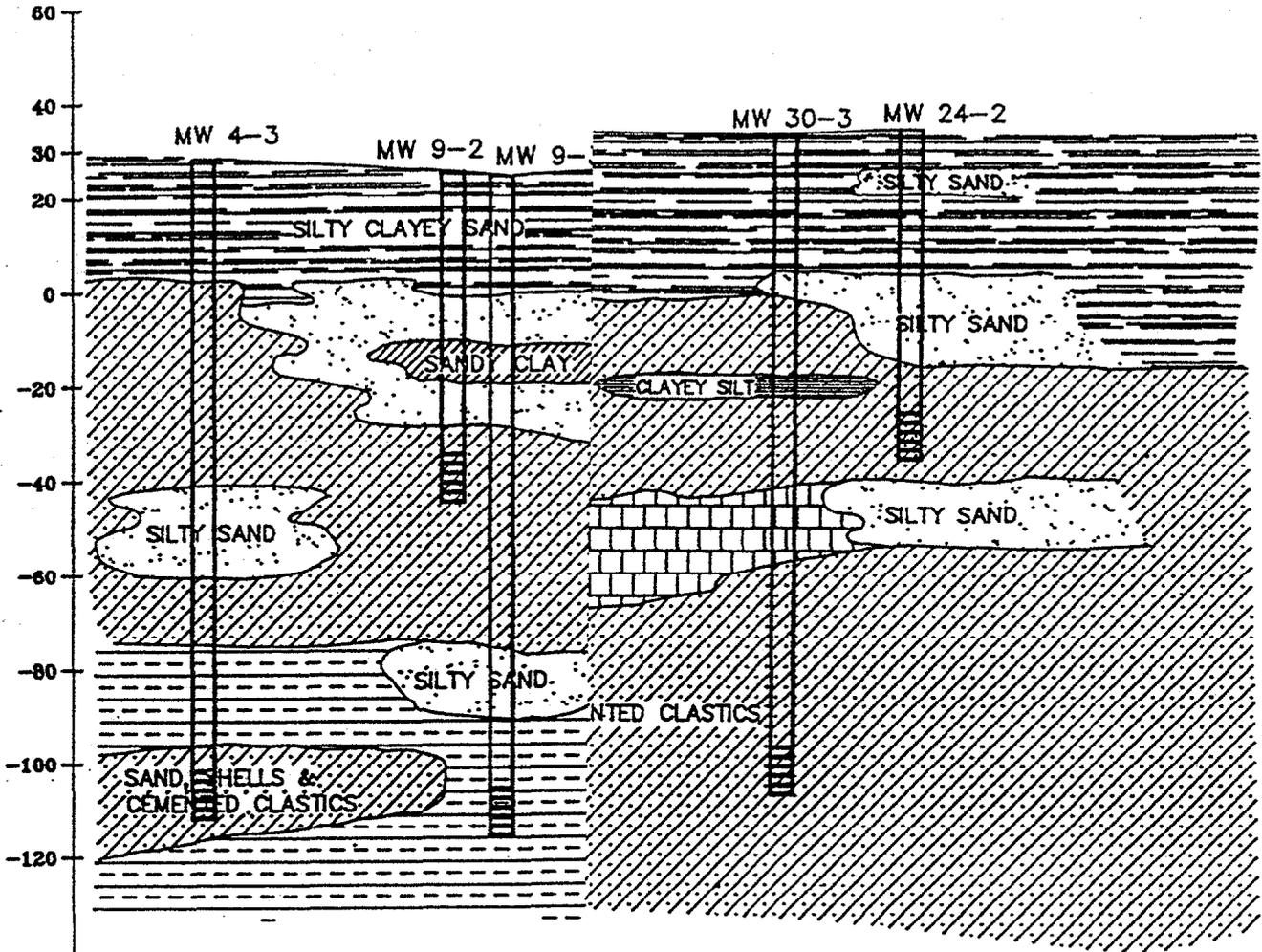
3.1.1.3 Geology

Three geologic formations occur in the MCB Camp Lejeune area. The oldest is the Trent formation, which dates from the late Oligocene epoch and is overlain by the Yorktown formation from the Miocene age. The youngest, upper layer consists of Pleistocene and Holocene sediments.

Within the Hadnot Point area, the site is underlain primarily by silty sand and extensive, but discontinuous, layers of silty clay and silty-sandy-clay that dip toward the south-southwest (Figure 3-1). The southwestern side of HPIA is covered by a shallow layer of peat that reflects the lesser developed state of this area. Other peat-covered areas, common in coastal marshland environments, may have been present in the past, but have been removed during development. Additionally, a deeper layer of sand-peat was identified in the northernmost, section of HPIA at a depth of approximately 18 ft below the surface. Marl, a combination of calcium carbonate, mud, and clay, was identified in the southeast corner and central portion of HPIA. A more detailed description of HPIA geology is found in the ESE 1988 and 1991 reports.

SW

NE



NOTE: All contacts inferred unless penetrated by boring.

HORIZONTAL SCALE : 1"=400'

VERTICAL SCALE : 1"=40'

V.E.=10

Figure 3-1
GENERALIZED CROSS SECTION OF HAD

SOURCE: ESE.



MARINE CORPS BASE
CAMP LEJEUNE

3.1.1.4 Geohydrology

A shallow aquifer (See Figure 3-1A) is encountered at a depth of less than ten feet below the land surface in most areas and in many areas it is at or just below the surface (Figure 3-2). In general, shallow groundwater flows toward the New River. The direction of flow actually ranges from south-southwest in the northern corner of HPIA to west-southwest in the southwest. Groundwater mounding appears to occur in the west-central and southeastern areas. This may be due to increased surface infiltration and a drainage ditch in the west-central and southern sections, respectively (ESE, 1988). The horizontal flow gradient over most of the area is proximately 0.003 ft/ft, but does increase to 0.02 ft/ft in the southwest corner of the site.

The deep aquifer, which is the producing zone for all of the water supply wells at HPIA and throughout MCB Camp Lejeune, is encountered at a depth of approximately 100 feet. This deep zone can be 100 feet or more in thickness. Between the deep and shallow aquifers is an alternating sequence of sands, silts, and clays (ESE, 1988). Water levels measured in deep and intermediate wells are similar to those observed in nearby shallow wells. However, it is expected that deep groundwater flows to the east southeast, towards the Atlantic Ocean (ESE, May 1988). Small-scale regional changes in groundwater flow may occur in the deep aquifer due to local pumping of water supply wells. The United States Geological Survey (USGS) (Harned *et al.*, 1989) notes that flow gradients may range from 15 feet/mile (0.0028 ft/ft) in areas unaffected by pumping to 150-200 feet/mile (0.0284-0.0378 ft/ft) in areas near active water supply wells.

3.1.1.5 Climate

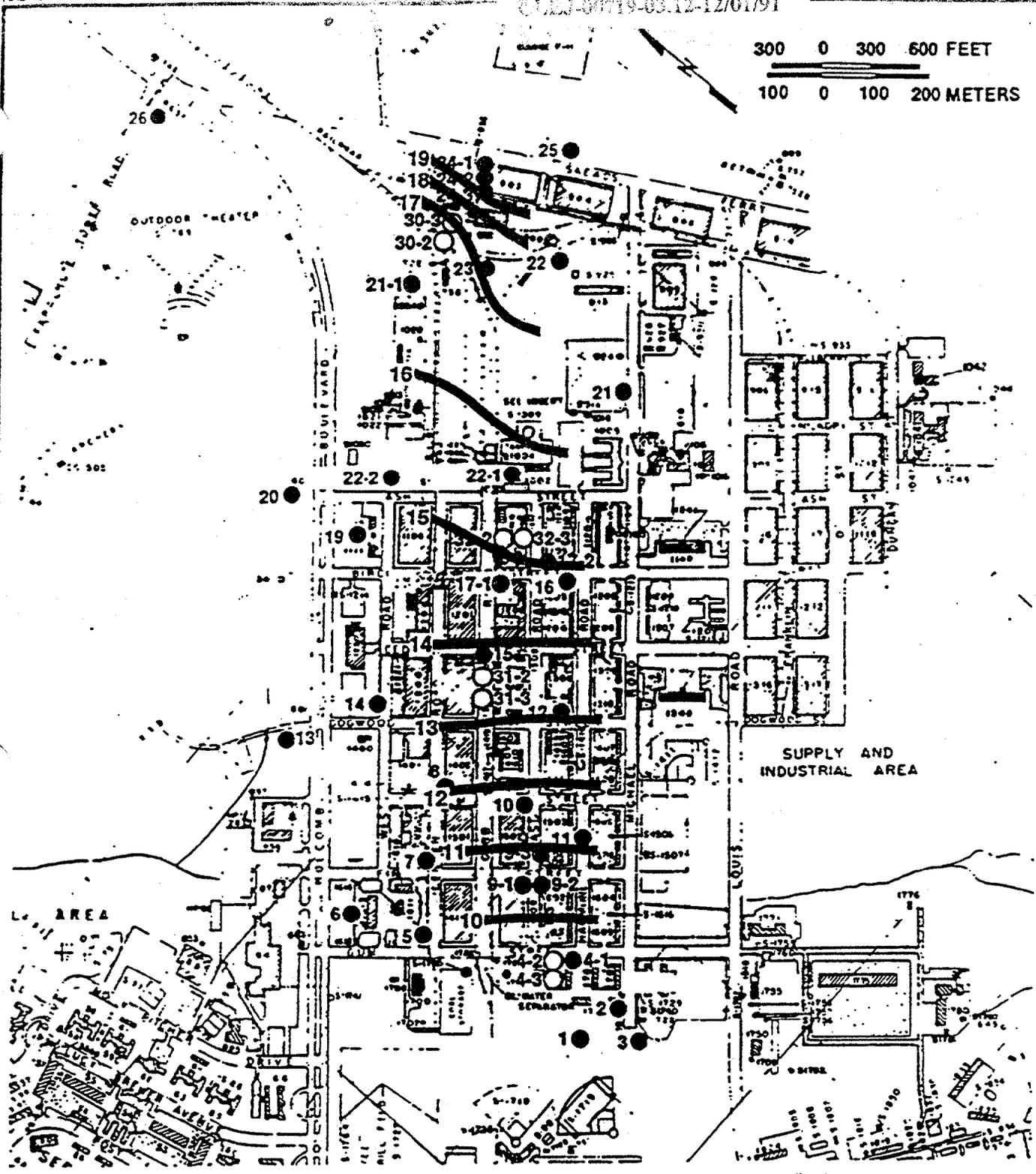
MCB Camp Lejeune has a mild climate, being generally hot and humid in the summer and cool in the winter. Rainfall averages four to five inches per month, with the higher amounts occurring the summer months, and the annual average precipitation is 55.96 inches. The mean temperature is approximately 60.9 °F. Hurricanes move through the area every few years.

Snow occasionally occurs, but persistence is rare. The prevailing wind direction is from the southwest; however, sea breezes are a regular occurrence along the coastline. The mild climate provides a long growing season, typically in excess of 230 days (Camp Lejeune, 1987). Table 3-1 summarizes important climatological data for MCB Camp Lejeune. Predominant wind patterns are illustrated in Figure 3-3.

3.1.1.6 Demographics

Results of a June 1990, census conducted for MCB Camp Lejeune indicated a total of 42,953 active duty individuals working within the MCB Camp Lejeune area. Approximately 42,448 dependents reside off-base, in the surrounding cities of Jacksonville and New Bern (USMC, 1990b). Approximately 12,266 dependents reside on-base within MCB. A total population of 54, 714 dependents exist both on and off the MCB facilities.

300 0 300 600 FEET
100 0 100 200 METERS



KEY

- EXISTING MONITOR WELL
- NEW MONITOR WELL

Figure 3-1A
POTENTIOMETRIC SURFACE MAP
DEEP AQUIFER

SOURCE: ESE (2-20-91)



MARINE CORPS BASE
CAMP LEJEUNE

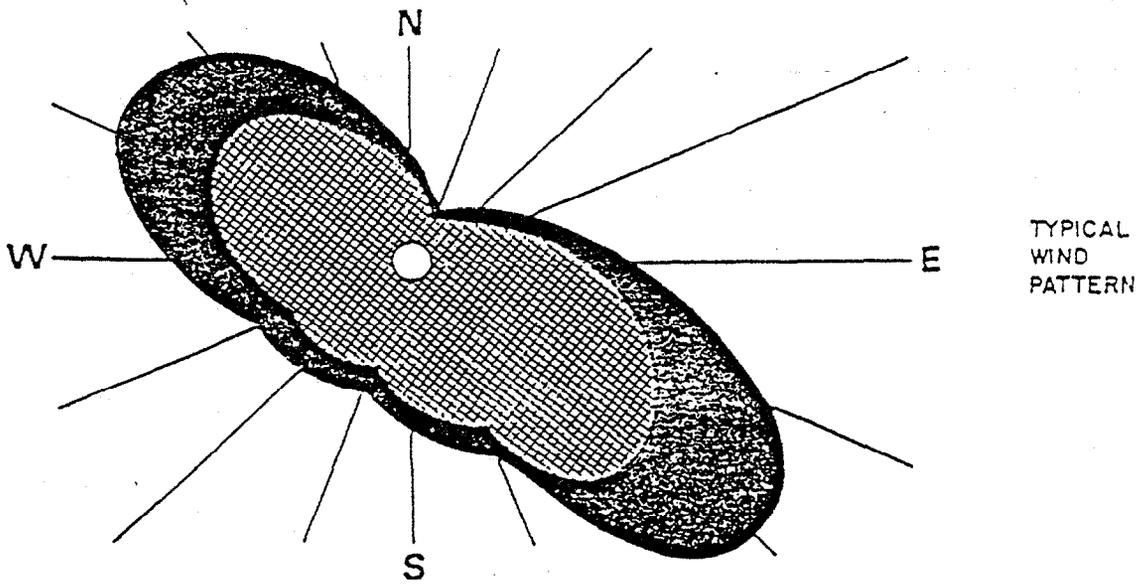
Table 3-1. Climatological Data for MCB Camp Lejeune Throughout the Year of 1990.

	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
<u>Temperature (°F)</u>												
Mean Monthly	44	47	54	62	70	77	80	80	75	65	56	48
Mean Daily Maximum	54	57	64	73	80	85	88	88	83	75	67	59
Mean Daily Minimum	34	36	43	51	60	67	72	71	66	54	45	37
<u>Humidity (Percent)</u>												
Mean Relative Humidity at 0400	78	77	79	81	87	88	89	90	89	85	82	80
Mean Relative Humidity at 1300	58	54	51	47	54	59	62	63	61	57	54	57
<u>Precipitation (Inches)</u>												
Mean Monthly	3.90	4.10	3.70	2.80	4.00	5.30	7.90	6.10	4.80	3.00	3.10	3.90
<u>Wind (Kts)</u>												
Most Frequent Direction	N	N	W	S	S	S	S	S	N	N	N	N
Mean Speed	7	7	8	8	7	6	6	5	6	7	6	7

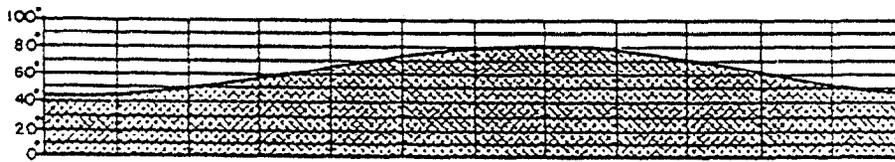
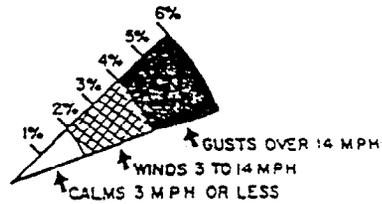
Kts = Knots

Source: USMC, 1990a.

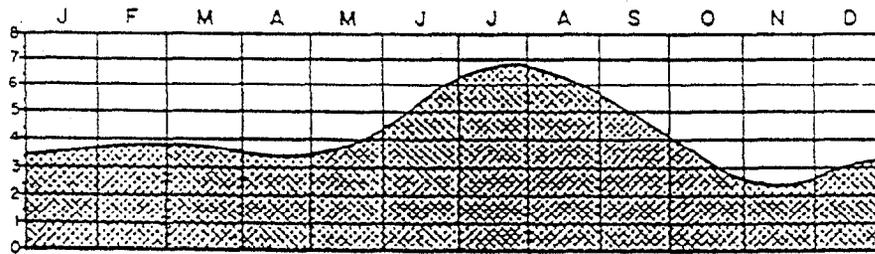
HADNOT PT RA 7/91 LB



% OF WIND COMING FROM INDICATED DIRECTION



AVERAGE MONTHLY TEMPERATURE



AVERAGE MONTHLY RAINFALL

Figure 3-3
 REGIONAL CLIMATIC CONDITIONS
 AT CAMP LEJEUNE
 SOURCE: CNAVACENGCOM, 1975



MARINE CORPS BASE
 CAMP LEJEUNE

3.1.1.7 Water Supply Source

The water supply for MCB Camp Lejeune comes from water wells 50 to 300 feet deep water wells located within the boundaries of the installation. Before distribution, water is piped from the 160 supply wells to the eight treatment plants located within the MCB Camp Lejeune complex. Each treatment plant has a water well system, ground and elevated water storage tanks, and distribution systems. Two plants, Hadnot Point-French Creek and Holcom Boulevard, use rapid sand filtration and lime softening. Rapid Sand Filters are mainly used in water treatment facilities to improve the quality of water by removing suspended solids. The sand filters are generally gravity type and are housed in open concrete basins. Smaller units may be housed in tanks and be subjected to pressure and gravity induced flows. The basic operation for sand filters is to introduce water from the top of the basin through a porous medium such as sand, or crushed anthracite coal. As the water moves downward through the pore-spaces, some of the fine suspended floc collides with sand surfaces and adheres to the sand particles. As the water passes through pore constrictions, some of the fine floc is brought together, flocculation occurs, and the enlarged floc settles on the top of the sand particles immediately below the constrictions. Also, the buildup of the floc that has been removed in the filter creates a straining action and some of the incoming floc is removed by straining. The Hadnot Point-French Creek distribution system serves the Hadnot Point Industrial Area, Division Billeting area, old Naval Hospital area, and French Creek (USMC, no date).

Results of the chemical verification efforts at HPIA identified the presence of VOCs in eight water supply wells. Five of these wells (601, 602, 608, 634, 637) were shut down and removed from the system by MCB Camp Lejeune utilities personnel. The five wells were located within close proximity to HPIA, while the three remaining wells are located in areas that may not be affected by similar VOC contamination (ESE, 1988).

3.1.2 POTENTIALLY EXPOSED HUMAN POPULATIONS

The military population of MCB Camp Lejeune is approximately comprised of 42,953 active duty personnel. The military dependent community is in excess of 42,448. Approximately 12,266 of these personnel and dependents reside in Base housing units. An additional 4,412 civilian employees perform facilities management and support functions (USMC, no date). However, due to the Gulf Crisis, the number of military personnel onsite has varied over the last year.

3.1.2.1 Proximity of Receptors to Sites

The two potential receptor populations associated with exposure to contaminants at Hadnot Point include onsite military personnel and offsite military dependants in the surrounding areas. The four areas of concern at Hadnot Point are located in areas that are actively used. The exact number of personnel in and around the buildings is unknown.

3.1.2.2 Current and Future Land Use

Based on the nature of work performed at the installation, the current major land use at HPIA is industrial. The industrial work activity is primarily conducted indoors with current work activity occurring in the areas of concern. The type of current land use of the areas surrounding HPIA are primarily industrial, residential, and some commercial.

Troop housing is generally located next to personnel support facilities, such as the Exchange or recreational areas. Community uses include all types of non-commercial personnel support facilities, such as dining facilities, libraries, child care facilities, and schools. Recreational facilities include playing fields, tennis and basketball courts.

Maintenance uses include vehicle and equipment servicing and repair and are generally situated adjacent to supply and storage areas. The existing land use patterns within and around the HPIA are illustrated in Figure 3-4.

Future land use plans include modifying building uses (commercial, residential, or industrial), resolving incompatibilities, and promoting the overall attractiveness of Hadnot Point. Currently, two troop housing facilities within HPIA are considered incompatible due to their proximity to supply/maintenance work areas, and therefore, the extension of these facilities in the future is unlikely (USMC, no date).

Within 15 miles of MCB Camp Lejeune are three large, publicly owned tracts of land: the Croatan National Forest, The Hofman Forest, and Camp Davis Forest. Because of the low elevations in the Coastal Plain the majority of the area is composed of wetlands that have been exploited to some extent by agricultural and silvacultural activities. The remaining land use surrounding MCB Camp Lejeune is agricultural, with typical crops of soybean, small grains, and tobacco. Productive estuaries along the coast support commercial finfish and shellfish industries. Tourism and residential resort areas are also located within the area.

Some areas of the New River at MCB Camp Lejeune are classified under Title 15 of the North Carolina Administrative Code as Class SC, while others are classified as Class SA. Class SC waters are useable for fishing and secondary recreation, but not for primary recreation or shellfish marketing. Class SA waters are the highest estuaring classification, useable for shellfish marketing (Figure 3-5).

3.1.2.3 Subpopulations of Potential Concern

Data concerning the number of persons less than 5 years of age and greater than 62 years of age residing in the area of HPIA is unavailable. These age groups represent subpopulations generally considered more sensitive to disease and illness than the general population.

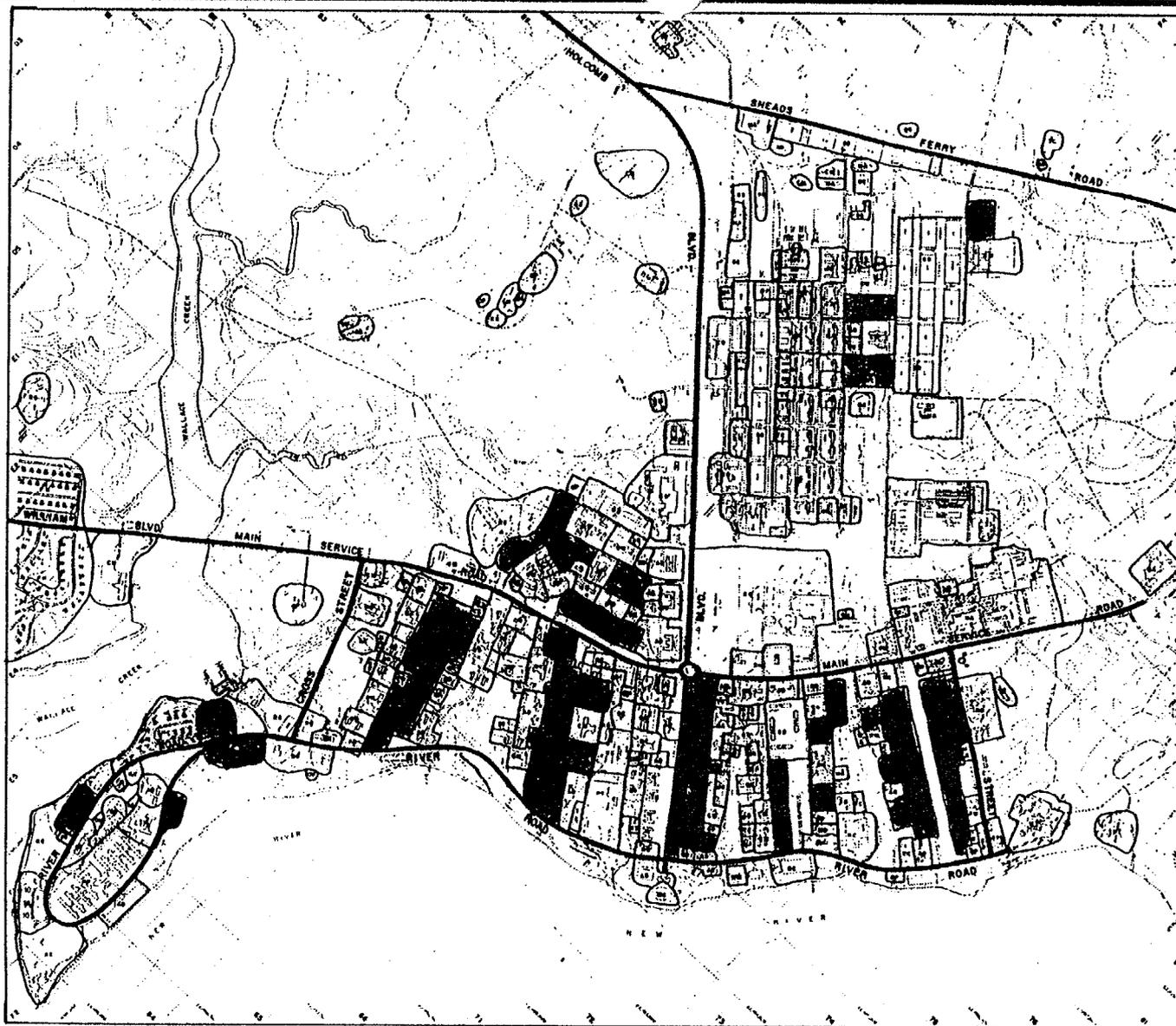
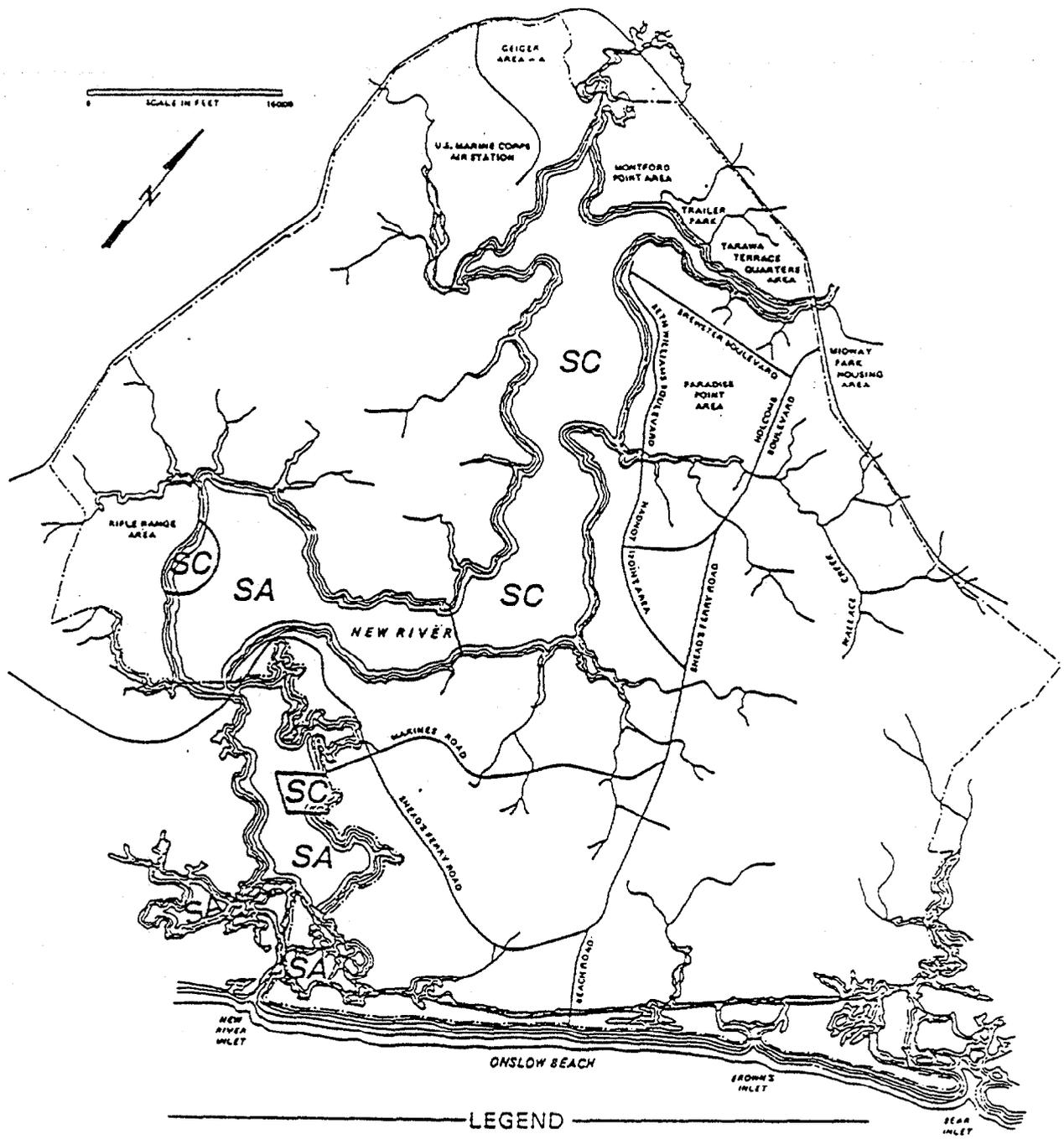


Figure 3-4
 EXISTING LAND USE PATTERNS AT HADNOT POINT
 CAMP LEJEUNE, NORTH CAROLINA
 SOURCE: U.S. Naval Engineering Command; ESE, 1991



**MARINE CORPS BASE
 CAMP LEJEUNE**

HADNOT PT RA 7/91 LB



LEGEND

SC ESTUARINE WATERS NOT SUITED FOR BODY CONTACT SPORTS OR COMMERCIAL SHELLFISHING

SA ESTUARINE WATERS SUITED FOR COMMERCIAL SHELLFISHING

Figure 3-5
 WATER QUALITY CLASSIFICATIONS
 FOR NEW RIVER, CAMP LEJEUNE
 SOURCE: North Carolina Department of Natural Resources, 1977



**MARINE CORPS BASE
 CAMP LEJEUNE**

HPIA residential areas are comprised of two troop housing facilities for bachelors. Occupation is limited to single persons for short durations (less than 5 years). Several family housing and troop housing facilities are located within the immediate areas adjacent to HPIA (see Figure 3-4).

3.1.3 POTENTIALLY EXPOSED WILDLIFE AND AQUATIC POPULATIONS

A large percentage of MCB Camp Lejeune is comprised of forested area that provides adequate habitat for a diversity of wildlife species. Several surface water drainages are located within MCB Camp Lejeune that provide habitat for aquatic species. In addition, MCB Camp Lejeune is bordered by the New River Estuary, which provides finfish and shellfish fisheries.

Vegetation is abundant within the MCB Camp Lejeune complex. Extensive tracts of both pure pine and pine-hardwood mixtures dominate the landscape. Pines include loblolly and longleaf, while hardwoods are represented by southern red oak, white oak, turkey oak, willow oak, and hickory. Areas on the periphery of the forests contain several species of shrubs, vines, and herbs. Acidic soil areas contain species of carnivorous plants, including the venus flytrap, sundew, and pitcher plants. The upland swamps are commonly referred to as pocosins and are overgrown with fetterbush, cyrilla, pond pine, greenbrier, and harvested species of pine.

Within the HPIA there is minimal habitat available for wildlife or aquatic life. Observations made during the 1991 field activities revealed a single surface water drainage located due east of the Hadnot Point Tank Farm (Site 22). The water sources appear to be contributed by runoff and a small pipe (source unknown). The drainage had an average width and depth of 2 feet and 0.5 feet respectively, with a flow of approximately 0.5 to 1 cubic feet per second (cfs). At the time of observation, no fish or aquatic invertebrate activity was noted.

Due to the extent of industrialization at Hadnot Point, it was determined that a minimal amount of onsite exposure to nonhuman organisms would occur. A minimal amount of riparian area exists to the west of the fuel tank farm and no activity or sign of small mammals was observed within the area during the field investigation activities.

3.1.3.1 Threatened/Endangered Species and State Special Animals

The United States Fish and Wildlife Service and the North Carolina Department of Natural Resources were contacted to obtain a list of threatened and endangered species. Table 3-2 identifies endangered and threatened species observed at MCB Camp Lejeune and their preferred habitat areas. The species that have an impact on carrying out the mission of the Military Complex are the Red-Cockaded Woodpecker, Atlantic Loggerhead Sea Turtle, Green Sea Turtle, Eastern Brown Pelican, and the American Alligator. Protection of habitat and foraging areas is essential to the survival of these species.

Table 3-2. Endangered and Threatened Species that Occur in the Camp Lejeune Complex.

Species	Common Name	Preferred Habitat	Status
<u>MAMMALS</u>			
Balaenoptera physalus	Finback Whale		Endangered
Magaptera novaeangliae	Humpback Whale		Endangered
Felis concolor cougar	Eastern Cougar		Endangered
<u>BIRDS</u>			
Picoides borealis	Red-Cockaded Woodpecker	Primary in longleaf timber types	Endangered
Pelecanus occidentalis	Brown Pelican	Coastal fringe along beach and inlets	Endangered
<u>REPTILES</u>			
Caretta caretta	Atlantic Loggerhead Sea Turtle	Warm ocean water. Frequent nesting along Onslow Beach.	Threatened
Chelonia mydas	Atlantic Green Sea Turtle	Schoal waters with submarine vegetation.	Threatened
Lepidochelys kempfi	Atlantic Ridley Turtle	Shallow coastal waters, observed in Intercoastal Waterway.	Endangered
Dermochelys coriacea	Atlantic Leatherback	Open sea waters along the coast.	Endangered
Eretomochelys imbricata	Atlantic Hawksbill Turtle	Reefs and shallow coastal waters.	Endangered
Alligator mississippiensis	American Alligator	Salt marshes, tidal streams and estuaries.	Endangered
<u>PLANTS</u>			
Dionaea muscipula	Venus' Fly Trap	Wet margins of open savannahs	Threatened
Sarracenia flava	Yellow Pitcher Plant	Wet bogs, ditches and savannahs	Threatened
Sarracenia rubra	Sweet Pitcher Plant	Shrub bogs and savannahs	Threatened
Sarracenia minor	Hooded Pitcher Plant	Wet bogs, ditches, and savannahs	Threatened
Sarracenia purpurea	Pitcher Plant; Flytrap	Wet bogs and savannahs	Threatened

Source: USMC, no date.

It was determined from the 1991 ESE investigations and consultation with the U.S. Fish and Wildlife Service and the North Carolina Department of Fish and Game that HPIA does not provide adequate habitat to support any of the species listed on Table 3-2 and, therefore, these species were not considered potential receptors of concern within HPIA.

3.2 ENVIRONMENTAL FATE AND TRANSPORT OF CHEMICALS OF CONCERN

3.2.1 SOURCES AND RECEIVING MEDIA

The following sections give a brief description of the waste disposal practices that have occurred at the areas of concern within the HPIA. A number of potential source areas within HPIA were identified and for the most part were associated with vehicle maintenance facilities. Four specific areas exhibited a higher probability of actually being the source of the observed contamination: 1) Buildings, 901,902, and 903; 2) Building 1202; 3) Buildings 1502 and 1601; and 4) Site 22 - Hadnot Point fuel tank farm.

Results of the 1988 characterization study (ESE, 1988) indicated that three primary zones of contamination were present at HPIA, centered respectively in the vicinities of Building 902, Building 1602, and Site 22. Intermediate and deep monitoring well data revealed VOC contamination occurring in wells adjacent to Buildings 1202 and 1601. For the purposes of this RA, the areas that immediately encompass Buildings 902, 1202 and 1602 were addressed as the areas of concern within HPIA. Site 22 was only partially characterized during the 1991 field activities because groundwater was the only media sampled.

3.2.1.1 Buildings 900-902

During the records search, Buildings 900-902 were found to have an underground tank used for storage of trichloroethene adjacent to Building 902. The contents of that tank have been drained and sent to the Defense Reutilization and Marketing Office (DRMO). No information regarding spills, leaks, or discharges from the tank was available (ESE, 1988). The area around Building 902 was identified as a long-term general vehicle maintenance area. The results of the soil gas investigation identified the presence of trichloroethene (TCE) vapors in the soil column in the vicinity of Building 902 (ESE, 1988).

3.2.1.2 Buildings 1200-1202

Building 1202, the Base Maintenance Shop, was identified as a potential source of contamination due to documented VOC storage and usage. Inspection of Building 1202 during the confirmation study identified several potential sources of VOC contamination. The most significant areas are the locations of former underground storage tanks and storage areas for drums and other containers of waste thinners, paints, and solvents.

3.2.1.3 Buildings 1600-1602

Building 1602, a heavy maintenance facility, had a long record of VOC storage and use (since 1942-1943). Earlier studies identified the presence of a 440-gallon underground storage tank of TCE. The current status is unknown. The confirmation study records search documented heavy solvent and petroleum, oil, and lubricant usage and storage in the building. Results of soil gas investigations corroborate the records search data. The soil vapors in the area between Buildings 1601 and 1502 had highly detectable levels of TCE, with concentrations as high as 703,000 parts per billion (ppb).

3.2.1.4 Hadnot Point Industrial Area Fuel Tank Farm

The HPIA fuel tank farm, constructed in the 1940s, is located east of the intersection of Gibb Road and Ash Streets and covers an area of approximately 4 acres, encompassing 14 underground storage tanks and one above ground tank. Several fuel leaks have occurred throughout the years, the latest being a 100-gallon leak of diesel fuel in 1981. In 1979, a fuel leak of an estimated 20,000 to 30,000 gallons of diesel and unleaded fuel occurred in an underground line near the tank truck loading facility.

3.2.2 CHEMICAL/PHYSICAL PROPERTIES

The chemical and physical properties of the COCs, including water solubility, $\log K_{ow}$, K_{oc} , vapor pressure, and Henry's Law constant, are given in Table 3-3. These values provide a perspective on the potential fate and transport of the COCs (i.e., high K_{oc} indicates chemicals most likely to adhere to soil particles). The following sections provide brief summaries of the chemical and physical properties of the COCs. A more in-depth discussion of these properties are presented in the COC toxicity profiles in Appendix A.

3.2.2.1 1,2-dichloroethene

Most of the 1,2-dichloroethene (1,2-DCE) released to the environment partitions to air or water. 1,2-DCE is very volatile and rapidly transforms in the troposphere by oxidation with hydroxyl radicals and photolysis. Biotransformation is also an important degradation process in surface soils.

3.2.2.2 Lead

In general, lead compounds produced industrially are soluble. However the actual lead compounds found in the environment are usually not mobile in groundwater and surface water because the lead leached from ores either becomes absorbed by oxides or combines with carbonate or sulfate ions to form insoluble compounds.

3.2.2.3 Benzene

Benzene has a vapor pressure of 95.2 millin(mm) Hg at 25°C and readily volatilizes from water and air. The half-life of benzene for air and water is approximately 6 and 1 to 6 days respectively. Evaluation of the soil-water partition coefficient and water solubility of Benzene indicates that this chemical will exhibit environmental mobility.

Table 3-3. Chemical and Physical Properties of the Chemicals of Concern.

COC	Molecular Weight	Water Solubility (mg/L)	Koc (mL/g)	log Kow	Vapor Pressure (mm Hg)	Henrys Law Constant (atm x m ³ /mol)	Fish BCF
1,2-DCE	96.94	8.5E3	1.4E1		324	4.5 x 10 ⁻³	
Lead	207	NA	--	--	1.0 (980°C)	--	60
Benzene	78.12	820	0.3 - 100	1.56 - 2.15	95.18	5.5 x 10 ⁻³	53 to 8450
<u>PAHs</u>							
Anthracene	178	0.07		4.45	1.0 (145°C)		485 (Fathead)
Benzo(a)anthracene	228	0.014	2.0 x 10 ⁵	5.61	2.2 x 10 ⁻⁸	1 x 10 ⁻⁶	
Benzo(a)pyrene	252	0.0038	5.5 x 10 ⁶	6.04	5.6 x 10 ⁻⁹	4.9 x 10 ⁻⁷	930 (Gambesia)
Benzo(g,h,i)perylene	276	0.00026		7.23			
Naphthalene	128	30		3.37	1.0 (52.6°C)		310 (Bluegill)

BCF = Bioaccumulation factor.

Koc = Organic partition coefficient.

NA = Data not available.

log Kow = Log octanol water partition coefficient.

Sources: Eisler, 1987; EPA, 1980; Sax, 1984.

3.2.2.4 Polynuclear Aromatic Hydrocarbons (PAHs)

PAHs vary in molecular weight from 128.16 (naphthalene) to 300.36 (coronene). PAHs with higher molecular weights are fairly immobile and have low volatility and solubility. Physical and chemical properties vary in relation to molecular weight. With increasing molecular weight, aqueous solubility decreases and octanol water coefficient increases.

3.2.3 POTENTIAL MIGRATION PATHWAYS

The potential for COCs to migrate from the source to other media (i.e., soil to groundwater) was identified for the accurate completion of this risk assessment. The following sections describe possible routes of COC migration at the four areas of concern within HPIA. In each case, the media initially contaminated (i.e., soil) was considered the starting point for contaminant migration.

3.2.3.1 Soil-to-Groundwater

During precipitation events, water may percolate through the contaminated material to provide a vehicle for dissolved chemicals to reach the shallow aquifer. As the leachate travels through the subsurface environment, varying fractions of the chemicals may be adsorbed onto organic matter or clays in the soils, thus reducing their capacity for migration (EPA, 1979b). Site soils are comprised predominantly of sandy loam and sandy clay and have moderately low to low permeabilities. The chemical fractions that do not readily adsorb and/or have relatively high water solubilities may infiltrate the shallow aquifer, located within 25 feet below land surface (bls) over the site (ESE, 1990).

3.2.3.2 Soil-to-Air

Chemicals in site soils may enter the atmosphere in two ways, by volatilizing from the soil or via suspended particulates (i.e., fugitive dusts). Soil particulates may enter the atmosphere via natural forces, such as wind, or by to anthropogenic causes, such as vehicular traffic. Residual contaminants bound to surficial soils may be transported as suspended particulates or dusts and, thus, may migrate from the source areas when environmental conditions are favorable.

Factors influencing the potential for dust entrainment into the atmosphere include surface roughness, surface soil moisture, soil particle sizes, kind and amount of vegetative cover, amount of soil surface exposed to the eroding wind force or vehicular traffic, physical and chemical properties of the soil, wind velocity, and other meteorological conditions (EPA, 1989). Current site-specific conditions at HPIA include the abundance of graveled lots, paved roads, or buildings over most of the areas of concern. All of these factors tend to decrease the potential for erosion and atmospheric suspension of particulates. Once in the atmosphere, contaminated dust or soil particles eroded from the site may be inhaled by receptors near the site. Volatilization of chemicals from

soils is not expected to be significant, due to the limited number of chemicals detected (acetone and methylene chloride). These may have been detected due to laboratory contamination.

3.2.3.3 Groundwater To Water Supply Wells

Since the MCB Camp Lejeune water supply wells tap the deep aquifer, the potential exists for direct migration of contaminants associated with the deep aquifer to the potable water supply. Water is pretreated before dispersal onsite, therefore, the volatile chemicals are most likely to be lost to the atmosphere through this process. The groundwater to water supply pathway is probably significant for persistent, mobile chemicals of concern.

3.2.3.4 Other Routes

Groundwater to surface water and groundwater to air were not considered significant migration routes due to the lack of surface water drainages onsite and the considerable depth (100 to 300 feet below ground surface) to reach the deep aquifer. It is unlikely that contaminants associated with this aquifer could readily transport to the surface.

3.3 IDENTIFICATION OF EXPOSURE PATHWAYS

An exposure pathway is the route that a chemical or physical agent takes from a source to an exposed population or individual (receptor). The exposure pathway describes a unique mechanism by which the receptor may be potentially exposed to chemicals or physical agents at/or originating from a site. For an exposure pathway to be complete, the following four elements must be present:

- A source or release from a source;
- A likely environmental migration route (i.e., leaching, volatilization, or partitioning from one medium to another) of a site-related chemical or physical agent;
- An exposure point where receptors may come in contact with site-related chemical or physical agents (i.e., a source area or environmental medium); and
- A pathway by which potential receptors may be exposed to a site-related chemical or physical agent (i.e., ingestion, direct contact, or inhalation of dusts or vapors).

If any of these components are not present, the exposure pathway is considered incomplete and is not expected to contribute to the total exposure from the site. A screening of current potential exposure pathways was conducted for each area of interest so that the risk characterization focuses only on the completed exposure pathways and eliminates from further consideration those pathways that are incomplete.

To perform a screening of completed human and nonhuman exposure pathways, each of the four elements listed above is identified and evaluated in detail. Routes of exposure (ingestion, direct contact, and inhalation) to the potentially contaminated media (soil and groundwater) are determined by careful examination of the current

extent of environmental contamination. The degree of exposure via each of the exposure pathways is determined by the following factors:

- Behavioral factors (i.e., the amount of time spent in contact with the contaminated medium, the amount of contaminated medium ingested, the amount of exposed skin);
- Chemical factors (i.e., the rate at which a chemical is absorbed through the skin, the degree to which a chemical is bioaccumulated in the body, the volatility of a chemical);
- Physical factors (i.e., soil particle size, ambient temperature, water body type, physical state of contaminant); and
- Physiological factors (i.e., age, skin condition, the ability of the body to metabolize and eliminate the chemical).

A summary of completed human exposure pathways is presented in Table 3-4. To quantify potential human exposures in the risk assessment process, it is necessary to make assumptions regarding each of the factors described previously in the absence of detailed site-, chemical-, or receptor-specific information. These assumptions, expressed as exposure factors and equations, are presented in Appendix B.

3.3.1 COMPLETED HUMAN EXPOSURE PATHWAYS

Groundwater and soil in all four areas were found to be contaminated with semivolatile organic chemicals (SVOCs), volatile organic chemicals (VOCs), and lead at the HPIA. The potential exposure pathways for the areas of concern include:

- Ingestion of VOCs, SVOCs, or lead contaminated groundwater or soil;
- Inhalation of volatilized VOCs and SVOCs from groundwater;
- Inhalation of dusts; and
- Dermal contact with VOCs, SVOCs, or lead in groundwater or soil

Exposure to chemicals associated with groundwater could occur through investigation, inhalation, and dermal contact (bathing, washing hands). Several pathways were excluded from the final pathway selection due to various reasons. For example, inhalation of dusts from the site is not considered significant due to the amount of paving, gravel, or presence of buildings in the areas of concern. It would be unlikely that contaminants associated with airborne particulates would create a significant exposure route. All other routes of exposure were considered significant, and thereby, quantitatively analyzed for chemical intake rates.

Current exposure to contaminants associated with groundwater cannot be accurately identified. The water for potable use onsite is supplied by a number of wells located throughout the entire base area. Water from these wells is pretreated at a central water treatment facility. The intermediate and deep groundwater monitoring wells are installed in the same aquifer that supplies the water supply wells. Thus, in the event that the water is not

Table 3-4. Summary of Completed Human (Corrected Worker) Exposure Pathways for Hadnot Point Areas of Concern.

Media	Exposure Pathway	Area 1	Area 2	Area 3
GROUNDWATER	Ingestion*	X	X	X
SOIL	Ingestion	X	X	X
	Direct Contact	X	X	X

Note: GW = groundwater.

* The current source of drinking water at Hadnot Point and nearby residential areas are from supply wells that draw water from the deep aquifer.

+ Includes adult and child exposure.

Source: ESE, 1991.

pretreated in the four areas of concern, the risks associated with exposure to the deep/intermediate groundwater can be estimated by summarizing the data from the monitoring wells. Although this pathway is unlikely due to the pretreatment of the water, the risks associated with groundwater exposure to workers was evaluated to represent the worst case scenario (i.e., water treatment was bypassed), and to determine the significance of groundwater contamination underlying the areas of concern.

Water supply wells were analyzed during the 1991 field efforts. The results of those analyses were not addressed in the risk assessment for the following reasons:

- Samples were collected directly from the well prior to treatment. Therefore, the concentrations observed would not be representative of concentrations to which receptors would be exposed.
- Water used for potable purposes is drawn from many wells located throughout the Hadnot Point Area. If potential contaminants of concern were detected, it would be difficult to determine the actual source of contamination.
- No defined exposure point exists.

3.3.1.1 Human Receptors

A worker was identified as the most representative human receptor at each area of concern. Though temporary residential facilities (less than 5 years) do exist within the HPIA, they are not directly associated with the areas of concern. It was therefore concluded the current worker would be the receptor to encounter the highest exposures (and subsequent, risks) associated with each area of concern.

A future residential scenario was not evaluated as a potential exposure pathway because future land uses of HPIA include further industrialization and enhancement of current uses (USMC, 1982).

3.3.2 COMPLETED NONHUMAN EXPOSURE PATHWAYS

For the evaluation of nonhuman exposure pathways, both terrestrial and aquatic receptors were evaluated. Aquatic species of animals, plants, and microorganisms are generally inescapably immersed in the water medium. Therefore, any chemicals associated with the water, sediment, or food sources can provide a direct exposure source to the organisms. In addition, recharge of the surface water by contaminated groundwater or runoff from contaminated soil can also provide an indirect source of contaminant exposure to aquatic life. Water soluble chemicals can enter an aquatic organism through the body surfaces (dermal and ocular), gills, and mouth.

Similarly, terrestrial organisms can become exposed through multiple routes due to their activity and proximity to the contaminated sites. Specific routes of potential exposure to terrestrial organisms include dermal, inhalation, and ingestion. The activity of terrestrial organisms onsite is unlimited. Some of the areas are fenced, though this does little to deter most organisms except possibly deer and other large mammals. Chemicals that are in food and soil can be ingested and absorbed through the digestive tract.

Quantification of dermal and inhalation exposure to terrestrial organisms is extremely difficult due to insufficient comparative laboratory data. Groundwater is not considered as an exposure medium for wildlife because points of contact were not identified (i.e., discharge).

Due to the extent of industrialization occurring onsite and specifically within the areas of concern, exposure to nonhuman organisms was considered to be negligible.

With the prevalence of industrialization, most of HPIA is covered by buildings, paved roads, or dirt/gravel lots. This does not provide adequate habitat to support many mammals. Possible ecological receptors at HPIA would include birds, vegetation, and possibly a few small mammals. Exposure pathways to nonhuman receptors identified at HPIA include:

- Incidental soil ingestion of metals, VOC, and/or SVOC contaminated soil; and
- Inhalation of dust borne contamination.

Most areas within HPIA are covered by asphalt, buildings, dirt/gravel lots, or lawn. Therefore, the likelihood of either of the above listed exposure pathways to occur is low.

Exposure to groundwater was not considered as a potential pathway since points of discharge were not located. Groundwater is not being utilized for any agricultural purposes onsite, therefore, direct access to water by ecological receptors is unlikely.

3.3.2.1 Nonhuman Receptors

Due to the limited habitat, it was determined that the ecological receptors potentially exposed at HPIA include vegetation, birds, and possibly some small mammals. During the 1991 field investigation, no species of small mammals were positively identified.

3.3.3 QUANTIFICATION OF EXPOSURE

3.3.3.1 Exposure Concentrations

An exposure concentration is the concentration of contaminant in an environmental medium (e.g. groundwater, surface soil, surface water, sediment, and air) that may reach a potential human or nonhuman receptor. Because the exposure concentration is the average concentration contacted at the exposure point or points over the exposure period, the objective is to provide a conservative estimate of this average concentration, such as the 95th percent upper confidence limit (UCL95), on the arithmetic mean chemical concentration (EPA, 1989). However, due to the limited data, UCL 95 values could not be calculated. The maximum detected concentration at each area of concern was used as the reasonable maximum exposure (RME) concentration. Onsite human and nonhuman exposure point concentrations have been estimated for the current exposure scenarios for the four areas of concern by using the maximum concentration observed for each COC (Table 3-5).

Table 3-5. Summary of Exposure Concentrations in Surface Soil and Groundwater (Deep and Intermediate) for Each Area of Concern at HPIA.

Chemical	Exposure Concentration ^a					
	Surface Soils (µg/kg)			Groundwater (µg/L)		
	902	1202	1602	902	1202	1602
1,2-Dichloroethene (Total)	BDL	BDL	BDL	12	1	11
Benzene	BDL	BDL	BDL	2	BDL	BDL
Lead ^b	56.90	84.80	36.60	13.50	8.90	27.10
Acenaphthene	42	72	BDL	1.00	5	BDL
Anthracene	180	15	BDL	BDL	BDL	BDL
Benzo(a)anthracene	280	140	BDL	BDL	BDL	BDL
Benzo(b)fluoranthene	250	140	BDL	BDL	BDL	BDL
Benzo(k)fluoranthene	210	150	BDL	BDL	BDL	BDL
Benzo(a)pyrene	240	140	BDL	BDL	BDL	BDL
Benzo(g,h,i)perylene	110	72	BDL	BDL	BDL	BDL
Chrysene	260	270	BDL	BDL	BDL	BDL
Flouranthene	690	370	BDL	BDL	BDL	BDL
Flourene	48	63	BDL	BDL	BDL	BDL
Indeno(1,2,3-cd)pyrene	130	82	BDL	BDL	BDL	BDL
2-Methylnaphthalene	BDL	BDL	300	9	2	BDL
Naphthalene	BDL	BDL	220	270	56	BDL
Phenanthrene	500	210	110	BDL	BDL	BDL
Pyrene	530	290	BDL	BDL	BDL	BDL

^a = Exposure concentrations were derived from maximum concentrations observed from each media at each area of concern.

^b = Units for Lead in Soils are mg/kg.

902 = Groundwater data for Area 902 was collected from wells HPGW24 and HPGW30. Soils data was collected from soil borings HPSB1 through 10.

1202 = Groundwater data for Area 1202 was collected from wells HPGW17 and HPGW31. Soils data was collected from soil borings HPSB11 through 20.

1602 = Groundwater data for Area 1602 was collected from wells HPGW9 and HPGW4. Soils data was collected from soil borings HPSB21 through 30.

Source: ESE, 1991.

Groundwater modeling to determine COC concentrations was not performed due to the limited amount of data. It was determined that concentrations of analytes in the deep groundwater were estimated quantities (J qualified data), meaning there is a limited confidence in the data value. Most of the data points were "J" qualified (e.g., the chemical was positively identified but the actual concentration was estimated). Most of these data points fall below detection limits and, therefore, the reported concentration was estimated by the laboratory by data extrapolation.

3.3.3.2 Estimation of Human Pathway-Specific Chemical Intakes

The chemical intake is the amount of contaminant entering the human receptor's body. Exposure pathway-specific chemical intakes are determined based on the exposure concentrations observed at the receptor area of concern, and on specific exposure factors. These exposure factors can be classified as chemical-specific (i.e., dermal absorption factors, skin permeability constants, volatilization factors) and nonchemical-specific (i.e., behavioral or physiological factors, exposure frequencies, exposed skin surface areas, exposure durations, body weights, intake rates). Nonchemical-specific exposure factors vary greatly according to the individual site and potential receptor.

To provide an estimate of a (RME) scenario, maximum concentrations for each COC in each matrix were used as the exposure concentrations. In addition, data from the intermediate and deep wells were combined for each site, since these wells draw from the same aquifer. The formulas used to calculate human pathway-specific chemical intakes were based on the generic chemical intake equation presented in EPA Risk Assessment Guidance (EPA, 1989) and illustrated below:

$$I = \frac{C \times CR \times EF \times ED}{BW \times AT}$$

- where: I = intake; the amount of chemical at the exchange boundary (mg/kg body-weight-day).
- C = chemical concentration; the average concentration contacted over the exposure period (e.g., mg/liter water).
- CR = contact rate; the amount of contaminated medium contacted per unit time or event (e.g., liters/day).
- EF = exposure frequency; describes how often exposure occurs (e.g., days/year).
- ED = exposure duration; describes how long exposure occurs (e.g., 1, 10, or 40 years).
- BW = body weight; the average body weight over the exposure period (kg).
- AT = averaging time; period over which exposure is averaged (days).

Because the exposure conditions differ for each exposure route, the site-specific exposure factors were incorporated into the generic formula for each exposure pathway to produce a set of chemical intake formulas specific for each exposure route. The site-specific chemical intakes are presented in Table 3-6. The site-specific chemical intake formulas and site-specific exposure factors used in each formula are discussed and presented in Appendix B, as are the chemical intakes.

3.3.3.3 Estimation of Nonhuman Pathway-Specific Chemical Intakes

For the purposes of quantifying exposure to ecological receptors, only exposure pathways involving soil were addressed since these receptors do not have direct access to the groundwater. Intake by terrestrial wildlife was quantified by applying estimated soil ingestion rates (Merck, 1979). Based on the variety of potential terrestrial species occurring within HPIA, soil ingestion is also estimated to be highly variable. Animals that burrow or prey on burrowing animals, such as earthworms or voles, are more likely to ingest higher quantities of soil than those that prey on species with little soil contact. Birds ingest grit and any accompanying soil for use in the gizzard. Very little research has been done to quantify soil ingestion by wildlife species. For this reason, soil ingestion was assumed to provide a contaminant uptake equivalent to five percent of the diet for all species (Merck, 1979), or:

<u>Animal</u>	<u>Dietary Dry Matter Intake (kg/kg bw/day)</u>	<u>Soil Intake (kg/kg bw/day)</u>
Poultry/Birds	4.80	0.24
Small Mammal (based on cats)	0.024	0.0012

The current and future land uses for HPIA are industrial, thereby limiting exposure to nonhuman receptors, such as birds, small mammals, and vegetation. Groundwater is not accessible, however, certain bare patches on contaminated soil may provide an exposure pathway.

Exposure concentrations used for the calculation of chemical intakes are equivalent to the maximum concentration of each COC identified in surface soils. The estimated intake rates for each nonhuman receptor type are shown in Table 3-7.

Table 3-6. Summarization of Exposure Assessment Results for the Current Land Use (Adult Worker) for each Area of Concern (Page 1 of 3)

Area	Population	Exposure Pathway	Chemical	Chronic Daily Intake (CDI) (mg/kg-day)		
				Carcinogenic Effects	Noncarcinogenic Effects	
Building 902	Adult Worker	Ingestion of contaminated GW from the AOC	Benzene	6.99 E-6	1.96 E-5	
			Naphthalene		2.64 E-3	
			Lead		1.32 E-4	
1,2-DCE (total)				1.17 E-4		
2-Methylnaphthalene				8.81 E-5		
Acenaphthalene				9.78 E-6		
Adult Worker			Dermal exposure of contaminated soil	Benzo(a)anthracene	4.08 E-7	1.14 E-6
	Chrysene	3.79 E-7		1.06 E-6		
	Benzo(b)fluoranthene	3.64 E-7		1.02 E-6		
	Benzo(a)pyrene	3.50 E-7		9.80 E-7		
	Benzo(k)fluoranthene	3.06 E-7		8.57 E-7		
	Indeno(1,2,3-cd)pyrene	1.90 E-7		5.31 E-7		
	Lead			2.32 E-5		
	Pyrene			2.16 E-6		
	Fluoranthene			2.82 E-6		
	Phenanthene			2.04 E-6		
	Benzo(g,h,i)perylene			4.49 E-7		
	Fluorene			1.96 E-7		
	Acenaphthene			1.71 E-7		
	Anthracene			7.35 E-7		
	Adult Worker	Ingestion of contaminated soil		Benzo(a)anthracene	4.89 E-8	1.37 E-7
				Chrysene	4.54 E-8	1.27 E-7
				Benzo(b)fluoranthene	4.37 E-8	1.22 E-7
Benzo(a)pyrene			4.19 E-8	1.17 E-7		
Benzo(k)fluoranthene			3.67 E-8	1.03 E-7		
Indeno(1,2,3-Cd)pyrene			2.27 E-8	6.36 E-8		
Lead				2.78 E-6		
Pyrene				2.59 E-7		
Fluoranthene				3.38 E-7		
Phenanthene				2.45 E-7		
Benzo(g,h,i)perylene				5.38 E-8		
Fluorene				2.35 E-8		
Acenaphthene				2.05 E-8		
Anthracene		8.81 E-8				

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Table 3-6. Summarization of Exposure Assessment Results for the Current Land Use (Adult Worker) for each Area of Concern (Page 2 of 3)

Area	Population	Exposure Pathway	Chemical	Chronic Daily Intake (CDI) (mg/kg-day)	
				Carcinogenic Effects	Noncarcinogenic Effects
Building 1202	Adult Worker	Dermal exposure to contaminated soil	Chrysene	3.94 E-7	1.10 E-6
			Benzo(k)fluoranthene	2.19 E-7	6.12 E-7
			Benzo(a)anthracene	2.04 E-7	5.71 E-7
			Benzo(a)pyrene	2.04 E-7	5.71 E-7
			Benzo(b)fluoranthene	2.04 E-7	5.71 E-7
			Indeno(1,2,3-Cd)pyrene	1.20 E-7	3.35 E-7
			Lead		3.46 E-5
			Pyrene		1.18 E-6
			Fluoranthene		1.51 E-6
			Phenanthene		8.57 E-7
			Benzo(g,h,i)perylene		2.94 E-7
			Fluorene		2.57 E-7
			Acenaphthene		2.94 E-7
			Anthracene		6.12 E-8
	Adult Worker	Ingestion of contaminated GW from the AOC	Lead		8.71 E-5
			Naphthalene		5.48 E-4
			1,2-DCE (total)		9.78 E-6
			Acenaphthalene		4.89 E-5
			2-Methylnaphthalene		1.96 E-5
	Adult Worker	Ingestion of contaminated soil	Chrysene	4.72 E-8	1.32 E-7
			Benzo(k)fluoranthene	2.62 E-8	7.34 E-8
			Benzo(a)anthracene	2.45 E-8	6.85 E-8
			Benzo(a)pyrene	2.45 E-8	6.85 E-8
			Benzo(b)fluoranthene	2.45 E-8	6.85 E-8
			Indeno(1,2,3-cd)pyrene	1.43 E-8	4.01 E-8
			Lead		4.15 E-6
			Pyrene		1.42 E-7
			Fluoranthene		1.81 E-7
			Phenanthene		1.03 E-7
			Benzo(g,h,i)perylene		3.52 E-8
			Fluorene		3.08 E-8
			Acenaphthene		3.52 E-8
			Anthracene		7.34 E-9

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Table 3-6. Summarization of Exposure Assessment Results for the Current Land Use (Adult Worker) for each Area of Concern (Page 3 of 3)

Area	Population	Exposure Pathway	Chemical	Chronic Daily Intake (CDI) (mg/kg-day)	
				Carcinogenic Effects	Noncarcinogenic Effects
Building 1602	Adult Worker	Ingestion of contaminated GW from the AOC	Lead 1,2-DCE (total)		2.65 E-4 1.08 E-4
	Adult Worker	Dermal exposure to contaminated soil	Lead Naphthalene 2-Methylnaphthalene		1.49 E-5 8.98 E-7 1.22 E-6
	Adult Worker	Ingestion of contaminated soil	Lead Naphthalene 2-Methylnaphthalene		1.79 E-6 1.08 E-7 1.47 E-7
Site 22	Adult Worker	Ingestion of contaminated GW from the AOC	Benzene	9.43 E-5	2.64 E-4
			Lead	2.20 E-5	6.36 E-5

AOC = Area of Concern
 GW = Groundwater
 1,2-DCE (total) = 1,2-Dichloroethylene

Table 3-7. Chemical Intake Concentrations for Nonhuman Receptors Exposed to COCs Associated with Soils From Each Area of Concern (mg/kg/bw-day) (Page 1 of 2)

Area of Concern	COC	Receptors		
		Vegetation*	Birds	Small Mammals
902	Lead	56.9	13.66	6.83 E-2
	Benzo(a)anthracene	2.80 E-1	6.72 E-2	3.36 E-4
	Chrysene	3.60 E-1	6.72 E-2	3.36 E-4
	Benzo(b)fluoranthene	2.50 E-1	6.24 E-2	3.12 E-4
	Benzo(a)pyrene	2.40 E-1	6.00 E-2	3.00 E-4
	Benzo(k)fluoranthene	2.10 E-1	5.04 E-2	2.52 E-4
	Indeno(1,2,3cd)pyrene	1.30 E-1	3.12 E-2	1.56 E-4
	Pyrene	5.30 E-1	1.27 E-1	6.36 E-4
	Fluoranthene	6.90 E-1	1.66 E-1	8.28 E-4
	Phenanthrene	5.00 E-1	1.20 E-1	6.00 E-4
	Benzo(ghi)perylene	1.10 E-1	2.64 E-2	1.32 E-4
	Acenaphthene	4.20 E-2	1.01 E-2	5.04 E-5
	Anthracene	1.80 E-1	4.32 E-2	2.16 E-4
	Fluorene	4.80 E-2	1.15 E-2	5.76 E-5
1202	Lead	84.8	20.35	1.02 E-1
	Benzo(a)anthracene	1.40 E-1	3.36 E-2	1.68 E-4
	Chrysene	2.70 E-1	6.48 E-2	3.24 E-4
	Benzo(b)fluoranthene	1.40 E-1	3.36 E-2	3.24 E-4
	Benzo(a)pyrene	1.40 E-1	3.36 E-2	1.68 E-4
	Benzo(k)fluoranthene	1.50 E-1	3.60 E-2	1.80 E-4
	Indeno(1,23cd)pyrene	8.20 E-2	1.97 E-2	9.94 E-5
	Pyrene	2.9 E-1	6.96 E-2	3.48 E-4
	Fluoranthene	3.70 E-1	8.88 E-2	4.44 E-4
	Phenanthrene	2.1 E-1	5.04 E-2	2.52 E-4
	Benzo(ghi)perylene	7.2 E-2	1.73 E-2	8.64 E-5
	Acenaphthene	7.2 E-2	1.73 E-2	8.64 E-5
	Anthracene	1.50 E-2	3.6 E-3	1.80 E-5
	Fluorene	6.30 E-2	1.5 E-2	7.56 E-5

Table 3-7.

Chemical Intake Concentrations for Nonhuman Receptors Exposed to COCs Associated with Soils From Each Area of Concern (mg/kg/bw-day) (Page 2 of 2)

Area of Concern	COC	Receptors		
		Vegetation*	Birds	Small Mammals
1602	Lead	36.6	8.78	4.39 E-2
	Phenanthrene	1.10 E-1	2.64 E-2	1.32 E-4
	Naphthalene	2.20 E-1	5.28 E-2	2.64 E-4
	2-Methylaphthalene	3.00 E-1	7.20 E-2	3.60 E-4
Site 22		NA		

NA = Soil samples were not collected from Site 22.

* = Value for intake is equivalent to direct exposure to maximum observed concentration.

Source: ESE, 1991

4.0 TOXICITY ASSESSMENT

4.0 TOXICITY ASSESSMENT

A toxicity assessment involves an in-depth examination of the physical and chemical properties and toxicity of the COCs. Toxicity assessments were performed for each indicator chemical or indicator chemical group (i.e., PAHs) by reviewing the available literature for information on acute and chronic health effects on human and nonhuman biota, as well as effects on the environment (Appendix A). Environmental fate is predicted for each of the indicator chemicals as data are available, including persistence, bioaccumulation, and breakdown products.

The objective of the toxicity assessment is to characterize the nature of the health effects associated with the COC at the four study areas within HPIA, including:

- Definition of terms commonly used in toxicity assessments for carcinogenic and noncarcinogenic effects;
- A summary profile of the available toxicological information for each COC to include pharmacokinetics, human health effects, environmental effects, and dose-response information; and
- A summary of the dose-response values used in the risk characterization (Section 5.0) for estimating acceptable intake levels and quantifying risks.

Terms relevant to the toxicity profiles and dose-response information are followed by a summary of the qualitative and quantitative toxicological information for each COC.

A total of four chemicals were identified as contaminants of concern for the HPIA study area. The following sections briefly describe the toxicity of each contaminant to human and nonhuman. Chronic and subchronic reference dose (RfD) values for most of the contaminants have been derived by the EPA and are presented in Table 2-5.

4.1 DEFINITION OF TERMS

A number of terms commonly used in toxicity assessments for carcinogenic and noncarcinogenic effects are defined in the following subsections.

4.1.1 CHRONIC REFERENCE DOSE

The chronic RfD estimates of daily exposure levels for the human population, including sensitive subpopulations, which is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA, 1989). Chronic RfDs are specifically developed to be protective for long-term exposure to a compound, Superfund program guidelines state seven years to a lifetime (EPA, 1989).

4.1.2 CHRONIC HAZARD INDEX

The chronic hazard index (HI) is a ratio of the lifetime average daily exposure of a noncarcinogenic chemical contaminant to the acceptable intake exposure level. If this ratio is greater than unity (<1), then the lifetime average daily exposure has exceeded the acceptable intake exposure level, indicating that there may be concern for potential noncancer effects (EPA, 1989).

4.1.3 CANCER SLOPE FACTOR

The cancer slope factor (CSF) is used to estimate an upper-bound probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen (EPA, 1989). The CSF is generally reported in mg/kg/day and is calculated using an assumed low-dosage linear relationship determined from animal studies (EPA, 1989). The value used in reporting the slope factor is an upper 95-percent confidence limit on the probability of response per unit intake of a chemical over a lifetime, converting estimated intakes directly to incremental risk (EPA, 1989).

4.1.4 CANCER RISK

For carcinogens, risks are estimated as the incremental probability of an individual developing cancer during a lifetime as a result of exposure to a potential carcinogen (i.e. incremental or excess individual lifetime cancer risk)(EPA, 1989). A cancer risk of $1.0E-06$ is the risk of one additional case of cancer per one million of exposed people.

4.2 TOXICITY PROFILES OF THE COCS

A summary of the toxicological information for each COC is presented in Appendix A to include discussions of chemical-specific pharmacokinetics, human health effects, environmental effects, and does-response information. Where human health effects data are limited, available animal data are presented.

4.3 QUANTITATIVE EVALUATION OF TOXICITY

A summary of the available noncarcinogenic and carcinogenic dose-response information for both the oral and inhalation routes of exposure for each COC are summarized in Table 2-5. The table lists the chemical name, oral and inhalation RfDs, oral and inhalation CSFs, and Weight of Evidence (WoE) categories for carcinogenic effects. The table also lists the federal acute and chronic ambient water quality criteria for the protection of freshwater and marine organisms. Where data was insufficient and/or unavailable to determine dose-response values for risk characterization, health-based values were developed using available regulatory references and resources for human health dose-response values.

5.0 RISK CHARACTERIZATION

5.0 RISK CHARACTERIZATION

The objectives of characterizing risk is to integrate information developed in the exposure assessment (Section 3.0) and the toxicity assessment (Section 4.0) into a complete evaluation of the current and future human health risks associated with contaminants detected at the four study areas at HPIA. The risk assessment evaluates the nature and degree of risk to potential receptor populations described in Section 3.0. Risk estimates are derived for individual contaminants, as well as for the total contaminant contribution from the identified sources, to identify the media and contaminants of most concern. Risk managers use these results to develop priorities for remedial action planning.

5.1 HUMAN RISK ASSESSMENT

The methods used in this risk analysis are those presented in the EPA Risk Assessment Guidance for Superfund: Human Health Evaluation Manual (1989), the Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors, (EPA, 1991), and other EPA exposure guidances. The main human exposure routes evaluated for the HPIA areas of concern were listed in Table 3-4.

Human health risks were determined for each exposure pathway. The health risks were evaluated separately for noncarcinogenic and carcinogenic effects, and carcinogenic compounds were also evaluated for their noncarcinogenic effects. The human health risks were evaluated for each area of concern based on the maximum exposure concentrations and exposure factors presented in Section 3.0.

Risk estimates relevant to uses of the deep aquifer are presented for current onsite water uses. Deep groundwater is used as a potable water source on the installation and is addressed in the individual site discussions with respect to current site exposure. Downgradient residential areas also draw water from the deep aquifer. However, risk estimates were not evaluated for current residential use located outside of HPIA (offsite) due to limitations of the data. The potential for future consumption of deep groundwater is addressed for the future residential land use scenario, which is found in Appendix D.

Risk estimates relevant to direct contact and incidental ingestion of surface soil are presented for onsite exposure scenarios. The current onsite risks are evaluated based on worker exposure.

Following the presentation of the risks associated with human and nonhuman exposure to site contaminants, the uncertainties associated with the risk analyses are presented in Section 5.3. These uncertainties may be attributable to lack of monitoring data, incomplete understanding of the mechanisms involved in contaminant transport, assumptions used in exposure assessment, or lack of toxicological information for a particular contaminant.

5.1.1 ASSUMPTIONS AND METHODS

Human health risks are discussed independently for carcinogenic and noncarcinogenic contaminants because of the different toxicological endpoints, relevant exposure durations, and methods employed in characterizing risk.

The COCs at HPIA considered by EPA as potential carcinogens include:

- Benzene
- Benzo(a)anthracene
- Benzo(a)pyrene
- Benzo(b,k)fluoranthene
- Chrysene
- Indeno(1,2,3-cd)pyrene
- Lead

Noncarcinogenic COCs are:

- 1,2-Dichloroethene
- 2-Methylnaphthalene
- Acenaphthene
- Anthracene
- Benzo(g,h,i)perylene
- Fluoranthene
- Fluorene
- Naphthalene
- Phenanthrene
- Pyrene

5.1.1.1 Carcinogenic Effects

Incidental human health risks associated with exposure to carcinogenic contaminants were calculated based on EPA's (1986a) Guidelines for Carcinogenic Risk Assessment, and EPA's (1986b) Guidelines for the Health Risk Assessment of Chemical Mixtures. Cancer risks were first calculated for individual contaminants by multiplying exposure levels of each contaminant by the appropriate carcinogenic slope factors (CSF) as follows:

$$\text{Risk} = I \times \text{CSF}$$

where: Risk = A unitless probability of an individual developing cancer,

I = Chronic daily chemical intake averaged over 70 years (mg/kg-day)(chemical intakes were calculated in Section 3.0 and presented in Table 3-6); and

CSF = Cancer potency slope factor, expressed in (mg/kg-day)⁻¹ (CSFs are presented in Table 2-5).

While estimating risk by considering one chemical at a time might significantly underestimate the risks associated with simultaneous exposures to several substances, the total combined health risk was also evaluated for each pathway by summing estimates derived for each compound in that pathway as presented below.

$$\text{Risk}_T = \sum \text{Risk}_i$$

where: Risk_T = the total cancer risk, expressed as a unitless probability; and
 Risk_i = the risk estimate for the i^{th} substance.

The additive approach is in accordance with EPA guidelines on chemical mixtures (EPA, 1986). This approach also assumes independence of action by the contaminants (i.e., that there are no synergistic or antagonistic chemical interactions and that all of the chemicals have the same toxicological endpoint of cancer). EPA (1986) also considers cancer risks from various exposure routes to be additive. Thus, risks from inhalation, dermal absorption, and oral exposures can be added to estimate total overall risk to human receptors as follows:

$$\text{Total Exposure Cancer Risk} = \text{Risk (pathway}_1) + \text{Risk (exposure pathway}_2) + \dots + \text{Risk (exposure pathway}_i)$$

The site-specific carcinogenic risk estimates were based on the reasonable maximum exposure concentrations and exposure factors presented in Section 3.0. In order to provide a perspective on the risks associated with the site, the magnitude of the cancer risks associated with the known or suspected carcinogens detected at the site were compared to the EPA acceptable cancer risk range of 1.0E-04 to 1.0E-06 (40 CFR 300.430:62). For known or suspected carcinogens, acceptable exposure levels are the residual concentration levels that represent an excess cancer risk to an individual of between 1.0E-04 to 1.0E-06 (40 CFR 300.430:62), based on the dose and response information for the particular COC. The NCP has identified an excess upper bound lifetime cancer risk of 1.0E-06 as the point of departure for determining the need for remediation of contaminants that do not have ARARs or for which an ARAR is not sufficiently protective because of the presence of multiple contaminants or multiple pathways of exposure (40 CFR 300.430:62).

5.1.1.2 Noncarcinogenic Effects

The measure used to describe the potential for noncarcinogenic toxicity to occur in an individual is not expressed as a probability, rather, the potential for noncarcinogenic effects is evaluated by comparing an exposure level over a specified time period (e.g., lifetime) with an RfD derived for a similar period (EPA, 1989). This ratio of exposure to toxicity is called an HI and is calculated as follows:

$$\text{Noncancer Hazard Index} = \frac{E}{\text{RfD}}$$

where: E = exposure level (or chemical intake);
 RfD = reference dose (RfDs are presented in Table 2-5).

The HI approach assumes that there is a level of exposure (i.e., RfD) below which it is unlikely for even sensitive populations to experience adverse health effects (EPA, 1989). If the exposure level exceeds the threshold level (i.e., if E/RfD exceeds unity), there may be a concern for potential noncancer effects (EPA, 1989). As with the carcinogenic contaminant evaluation, estimating noncancer hazard potential by considering one chemical at a time might significantly underestimate the risks associated with simultaneous exposures to several substances. Thus, the total combined HIs were also evaluated for each pathway by summing estimates derived for each compound for that pathway, as follows:

$$HI = E_1/RfDI_1 + E_2/RfDI_2 + \dots + E_i/RfDI_i$$

where: E_i = exposure level (dose) for the i^{th} contaminant,
 $RfDI_i$ = reference dose for the i^{th} contaminant.

This additive approach assumes that multiple exposures could result in an adverse effect and that the magnitude of the effect is proportional to the sum of the ratios of the exposures to acceptable exposures. The assumption of additivity is applicable to contaminants that induce the same type of effect. If the HI is greater than unity, contaminants are reevaluated by critical effect, and separate HIs are calculated by type of effect. The possible effects of multimedia exposures are evaluated by summing the HI values for inhalation and oral exposures for the relevant exposure routes.

Noncarcinogenic endpoints may be the result of chronic (e.g., seven years to a lifetime), subchronic (e.g., two weeks to seven years), and shorter-term exposures (e.g., less than two weeks) (EPA, 1989). As the exposure scenarios evaluated for HPIA are long-term in nature (year-round users of drinking water resources, working at HPIA as a career), the hazard assessment evaluated chronic long-term exposures using chronic RfD values.

5.1.2 SITE-SPECIFIC RISK CHARACTERIZATION

The carcinogenic risks and noncarcinogenic hazard indices were calculated for each of the four onsite study areas at Hadnot Point (Areas 902, 1202, 1602 and the Hadnot Point Fuel Tank Farm). Because the activities performed at each area differ and the areas are not in close proximity to each other, the risks were presented separately for each area. Carcinogenic risks and noncarcinogenic HI's are presented in Tables 5-1 through 5-5 and in Appendix C. More importantly, characterizing each study area separately allows for prioritization of remedial activities that may be required.

The risk characterization for each study area evaluated the risks associated with potential worker exposure to the COCs identified in the surface soil and the deep aquifer. As the Hadnot facility is currently used for industrial purposes and is expected to be further industrialized in the future (USMC, no date) the risks evaluated were based on an industrial worker exposure scenario. Assuming that the concentrations of chemicals do not

Table 5-1. Carcinogenic Risks Associated with Potential Exposure to Soil and Groundwater at Area 902.

Chemical	Oral WOE	Media	Risk		Total Risk (Dermal & Oral)
			Dermal	Oral	
Benzo (a) anthracene	B2	S	4.7E-06	5.6E-07	5.3E-06
Benzo (a) pyrene	B2	S	4.0E-06	4.8E-07	4.5E-06
Benzo (b) flouranth	B2	S	4.2E-06	5.0E-07	4.8E-06
Benzo (k) flouranth	B2	S	4.2E-07	4.2E-07	3.9E-06
Chrysene	B2	S	4.4E-06	5.2E-07	4.9E-06
Indeno (cd)	B2	S	2.2E-06	2.6E-07	2.4E-06
SUBTOTAL			2.3E-05	2.8E-06	2.6E-07
Benzene	A	GW	--	2.0E-07	2.0E-07
SUBTOTAL			--	2.0E-07	2.0E-07
GRAND TOTAL RISK			2.3E-05	3.0E-06	2.6E-05

Table 5-2. Noncarcinogenic Hazard Indices Associated with Potential Exposures to Groundwater and Soil at Area 902.

Chemical	Media	HI		Total HI (Dermal + Oral)
		Dermal	Oral	
Acenaphthene	S	2.9 E-06	3.4 E-07	3.2 E-06
Anthracene	S	2.4 E-06	2.9 E-07	2.7 E-06
Benzo(a)anthracene	S	3.8 E-05	4.6 E-06	4.3 E-05
Benzo(a)pyrene	S	3.3 E-05	3.9 E-06	3.7 E-05
Benzo(b)fluoranthene	S	3.4 E-05	4.1 E-06	3.8 E-05
Benz(ghi)pyrene	S	1.5 E-05	1.8 E-06	1.7 E-05
Benzo(k)fluoranthene	S	2.9 E-05	3.4 E-06	3.2 E-05
Chrysene	S	3.5 E-05	4.2 E-06	4.0 E-05
Fluoranthene	S	7.0 E-05	8.4 E-06	7.9 E-05
Fluorene	S	4.9 E-06	5.9 E-07	5.5 E-06
Indeno(1,2,3cd)pyrene	S	1.8 E-05	2.1 E-06	2.0 E-05
Phenanthrene	S	6.8 E-05	8.2 E-06	7.6 E-05
Pyrene	S	7.2 E-05	8.6 E-06	8.1 E-05
Lead	S	4.6 E-02	5.6 E-03	5.2 E-02
SUBTOTAL		4.7 E-02	5.6 E-03	5.3 E-02
1,2-DCE	GW	--	1.2 E-02	1.2 E-02
Lead	GW	--	2.6 E-01	2.6 E-01
2-Methylnaphthalene	GW	--	2.9 E-03	2.9 E-03
Acenaphthene	GW	--	1.6 E-04	1.6 E-04
Naphthalene	GW	--	6.6 E-01	6.6 E-01
SUBTOTAL		--	9.4 E-01	9.4 E-01
GRAND TOTAL		4.7 E-02	9.5 E-01	9.9 E-01

1,2-DCE = 1,2-Dichloroethene
S = Surface Soil

GW = Groundwater
HI = Hazard Index

Source: ESE, 1991

change over time the following noncarcinogenic and carcinogenic risks were determined to represent current and future site conditions.

5.1.2.1 Area 902

The analytical results indicate the presence of both noncarcinogenic and carcinogenic contaminants in groundwater and surface soil at area 902. Groundwater contaminants include benzene, naphthalene, lead, dichloroethenes, methylnaphthalene, and acenaphthene, while surface soil contaminants include PAHs and lead. As the exposure assessment indicated, exposure to contaminants in soil may occur by inadvertent ingestion of small quantities of soil and dermal absorption of contaminants from soil during work activity involving direct contact with soils in this area. Although the facility is supplied with potable water, which is drawn from the deep aquifer and pretreated prior to distribution, a current ingestion exposure potable use scenario at area 902 was also evaluated to determine if the detected concentrations of contaminants in the deep aquifer may pose health concerns in the unlikely event that the groundwater is not pretreated prior to potable uses.

The results of the carcinogenic and noncarcinogenic risk analyses are presented in Tables 5-1 and 5-2, respectively, and discussed below.

Carcinogenic--Groundwater

The only carcinogen detected in groundwater from this area was benzene, which resulted in a total carcinogenic health risk of 2×10^{-7} . This risk level is below the EPA acceptable cancer risk range of 10^{-4} to 10^{-6} (40 CFR 300.430:62) indicating that the concentration of benzene at this area is not expected to incur significant health risks based on the exposure assumptions evaluated (i.e., ingestion of the maximum concentration detected at the site).

Carcinogenic--Soil

The results of the soil risk assessment at this area indicate that direct contact with soils (dermal and ingestion) results in an overall potential risk of 2.57×10^{-5} . This risk level is within the EPA acceptable cancer risk range of 10^{-4} to 10^{-6} (40 CFR 300.430:62), but exceeds the excess upper bound lifetime cancer risk of 1×10^{-6} . Because the NCP has identified a cancer risk of 1×10^{-6} as the point of departure for determining the need for remediation of contaminants that do not have ARARs or for which a ARAR is not sufficiently protective because of the presence of multiple contaminants or multiple pathways of exposure [40 CFR 300.430:62], soils at this area are considered for further evaluation in the Feasibility Study.

Noncarcinogenic--Groundwater

Lead, naphthalene, methylnaphthalene, acenaphthene, dichloroethenes, and benzene are the noncarcinogenic COCs detected in the groundwater at area 902. The results of the HI calculations indicate that the individual COCs as well as the total sum of COCs do not result in HIs exceeding one. Therefore, these compounds are

not expected to incur toxic or systemic effects at the concentrations detected at the site based on the exposure assumptions evaluated at this area.

Noncarcinogenic--Soil

Lead and PAHs are the noncarcinogenic COCs detected in the surface soil at area 902. As with the groundwater results, the individual COCs as well as the total sum of COCs do not result in HIs exceeding one. Therefore, these compounds are not expected to incur toxic or systemic effects at the concentrations detected at the site, based on the exposure assumptions evaluated at this area.

5.1.2.2 Area 1202

The analytical results indicate the presence of both noncarcinogenic and carcinogenic contaminants in groundwater and soils at area 1202. Groundwater contaminants include naphthalene, lead, dichloroethenes, methylnaphthalene, and acenaphthene, while surface soil contaminants include PAHs and lead. Exposure to soil and groundwater at area 1202 can occur through pathways similar to those described for area 902 (Refer to Section 5.1.2.1). The results of the carcinogenic and noncarcinogenic risk analyses for soil and groundwater exposure at area 1202 are presented in Tables 5-3 and 5-4, and discussed below.

Carcinogenic-Groundwater

There were no carcinogens detected in the groundwater at this area.

Carcinogenic-Soil

The results of the soil risk assessment at this area indicate that direct contact with soils (dermal and ingestion) results in an overall potential risk of 1.73×10^{-5} . This risk level is within the EPA acceptable cancer risk range of 10^{-4} to 10^{-6} (40 CFR 300.430:62), but exceeds the excess upper bound lifetime cancer risk of 1×10^{-6} . Because the NCP has identified a cancer risk of 1×10^{-6} as the point of departure for determining the need for remediation of contaminants that do not have ARARs or for which a ARAR is not sufficiently protective because of the presence of multiple contaminants or multiple pathways of exposure [40 CFR 300.430:62], soils at this area are considered for further evaluation in the Feasibility Study.

Noncarcinogenic-Groundwater

Lead, naphthalene, 2-methylnaphthalene and 1,2-dichloroethenes were the noncarcinogenic COCs detected in the groundwater at area 1202. The results of the HI calculations indicate that the individual COCs as well as the total sum of COCs do not result in HIs exceeding one. Therefore, these compounds are not expected to incur toxic or systemic effects at the concentrations detected at the site based on the exposure assumptions evaluated at this area.

Table 5-3. Carcinogenic Risks Associated with Potential Exposures to Soil at Area 1202.

Chemical	Oral WoE	Risks		Total (Dermal + Oral)
		Dermal	Oral	
Benzo(a)anthracene	B2	2.3 E-06	2.8 E-07	2.6 E-06
Benzo(a)pyrene	B2	2.3 E-06	2.8 E-07	2.6 E-06
Benzo(b)fluoranthene	B2	2.3 E-06	2.8 E-07	2.6 E-06
Benzo(k)fluoranthene	B2	2.5 E-06	3.0 E-07	2.8 E-06
Chrysene	B2	4.5 E-06	5.4 E-07	5.1 E-06
Indeno(1,2,3cd)pyrene	B2	1.4 E-06	1.6 E-07	1.5 E-06
GRAND TOTAL		1.5 E-05	1.9 E-06	1.7 E-05

WOE = EPA Weight of Evidence Category.

Source: ESE, 1991

Table 5-4. Noncarcinogenic Hazard Indices Associated with Potential Exposures to Groundwater and Soil at Area 1202.

Chemical	Media	HI		Total HI (Dermal & Oral)
		Dermal	Oral	
Acenaphthene	S	4.9E-06	5.9E-07	5.5E-06
Anthracene	S	2.0E-07	2.4E-08	2.3E-07
Benzo(a)anthracene	S	1.9E-05	2.3E-06	2.1E-05
Benzo(a)pyrene	S	1.9E-05	2.3E-06	2.1E-05
Benzo(b)fluoranthene	S	1.9E-05	2.3E-06	2.1E-05
Benzo(ghi)pyrene	S	9.8E-06	1.2E-06	1.1E-05
Benzo(k)fluoranthene	S	2.0E-05	2.4E-06	2.3E-05
Chrysene	S	3.7E-05	4.4E-06	4.1E-05
Fluouranthene	S	3.8E-05	4.5E-06	4.2E-05
Fluorene	S	6.4E-06	7.7E-07	7.2E-06
Indeno(1,2,3cd)pyrene	S	1.1E-05	1.3E-06	1.2E-05
Phenanthrene	S	2.9E-05	3.4E-06	3.2E-05
Pyrene	S	3.9E-05	4.7E-06	4.4E-05
Lead	S	6.9E-02	8.3E-03	7.7E-02
SUBTOTAL		6.9E-02	8.3E-03	7.7E-02
Naphthalene	GW	--	1.4E-01	1.4E-01
Lead	GW	--	1.7E-01	1.7E-01
1,2-DCE	GW	--	9.8E-04	9.8E-04
2-Methynaphthalene	GW	--	6.5E-04	6.5E-04
Acenaphthene	GW	--	8.2E-04	8.2E-04
SUBTOTAL			3.1E-01	3.1E-01
GRAND TOTAL		6.9E-02	3.2E-01	3.9E-01

Noncarcinogenic-Soil

Lead and PAHs are the noncarcinogenic COCs detected in the surface soil at area 902. As with the groundwater results the individual COCs as well as the total sum of COCs do not result in HIs exceeding one. Therefore, these compounds are not expected to incur toxic or systemic effects at the concentrations detected at the site based on the exposure assumptions evaluated at this area.

5.1.2.3 Area 1602

The analytical results indicate the presence of only noncarcinogenic contaminants in groundwater and soil at area 1602. Groundwater contaminants include lead and 1,2-dichloroethenes, while surface soil contaminants include lead, naphthalene and methylnaphthalene. Exposure to soil and groundwater at area 1602 can occur through pathways similar to those described for area 902 (see section 5.1.2.1). The results of the noncarcinogenic risk analyses for soil and groundwater exposure at area 1602 are presented in Table 5-5 and discussed below.

Noncarcinogenic-Groundwater

Lead and 1,2-dichloroethenes are the noncarcinogenic COCs detected in the groundwater at area 1602. The results of the HI calculations indicate that the individual COCs as well as the total sum of COCs do not result in HIs exceeding one. Therefore, these compounds are not expected to incur toxic or systemic effects at the concentrations detected at the site based on the exposure assumptions evaluation at this area.

Noncarcinogenic-Soil

Lead, naphthalene and 2-methylnaphthalene are the noncarcinogenic COCs detected in the surface soil at area 1602. As with the groundwater results of the individual COCs as well as the total sum of COCs do not result in HIs exceeding one. Therefore, these compounds are not expected to incur toxic or systemic effects at the concentrations detected at the site based on the exposure assumptions evaluated at this area.

5.1.2.4 Site 22

The analytical results indicate the presence of two potentially carcinogenic COCs, benzene, and lead in the deep aquifer below Site 22. The observed level of benzene (27 µg/L) was elevated above the MCL of 5 µg/L, therefore, there is risk associated with exposure to groundwater from Site 22. Exposure to groundwater at Site 22 can occur through pathways similar to those already described for areas 902, 1202, and 1602. The risk associated with soil related contamination were not addressed since soil samples were not collected from this area. The results of the carcinogenic and noncarcinogenic risk analyses for Site 22 groundwater are presented in Table 5-6.

Carcinogenic-Groundwater

The results of the deep groundwater risk assessment indicate that contact with groundwater results in a risk of 2.8E-06. This level is at the 1.0E-06 point of departure identified by the NCP. Benzene was detected in one well at a level of 27 µg/L, which exceeds the established MCL of 5 µg/L for the protection of human health.

Table 5-5. Noncarcinogenic Hazard Indices Associated with Potential Exposures to Groundwater and Soil at Area 1602.

Chemical	Media	HI		Total HI (Dermal + Oral)
		Dermal	Oral	
2-methylnaphthalene	S	4.1 E-05	4.9 E-06	4.6 E-05
Naphthalene	S	2.2 E-04	2.7 E-05	2.5 E-04
Lead	S	3.0 E-02	3.6 E-03	3.3 E-02
SUBTOTAL		3.0 E-02	3.6 E-03	3.4 E-02
1,2-DCE	GW	--	1.1 E-02	1.1 E-02
Lead	GW	--	5.3 E-01	5.3 E-01
SUBTOTAL		--	5.4 E-01	5.4 E-01
GRAND TOTAL		3.0 E-02	5.4 E-01	5.7 E-01

1,2-DCE = 1,2-Dichloroethene
 HI = Hazard Index
 S = Surface Soil
 GW = Groundwater

Source: ESE, 1991

Table 5-6. Carcinogenic and Noncarcinogenic Risks Associated with Potential Exposures to Groundwater at Site 22.

Chemical	Media	Noncarcinogenic (Oral HI)	Carcinogenic Risk
Benzene	GW	2.64 E-06	2.80 E-06
Lead	GW	1.30 E-01	NA
GRAND TOTAL		1.30 E-01	2.80 E-06

GW = Groundwater
HI = Hazard Index

Source: ESE, 1991

Noncarcinogenic - Groundwater

Results of the risk characterization indicate an overall potential risk associated with noncarcinogenic exposure fall below an HI of one. The results of the HI calculations indicate that the individual COCs as well as the total sum of COCs do not exceed a value of one. Therefore, exposure to noncarcinogenic chemicals associated with Site 22 groundwater are not expected to cause toxic or systemic effects. In addition, the concentrations of lead fall below the final action level of 15 $\mu\text{g}/\text{L}$ for lead in groundwater (56 FR 26478, June 7, 1991).

5.2 NONHUMAN RISK ASSESSMENT

The methods used in the ecological risk analysis are those presented in the EPA Risk Assessment Guidance for Superfund: Environmental Evaluation Manual (1989). Ecological risks were only determined for the soil ingestion pathway. Exposure to groundwater within the HPIA was not considered a feasible pathway since points of groundwater discharge were not identified and there are no agricultural uses of groundwater within HPIA (i.e., irrigation of crops).

The nonhuman risk characterization was performed by comparison of calculated chemical intakes to literature derived toxicity values (i.e., lowest observed effect level [NOEL] or no observed effect level (NOEL)). If lowest or no observed effect levels were not given, toxicity data were reviewed and the lowest relative value reported as toxic was used.

The literature derived values used for the qualitative comparisons to establish risk are presented in Table 5-7. Often, data were not available for certain species groups. For instance, laboratory animal studies were common, but little data were available for wild animals. The toxicological data base is even more sparse for birds. For chemicals lacking toxicity information for a species group, quantification of risk was not made specifically for that group due to the uncertainty involved extrapolating between taxonomic kingdom, phyla, or class. For example, if toxicity data for plants were unavailable, risk for plants was not quantified from data derived from mammal studies. Although extrapolation within a class (i.e., rats to small wild mammals) is also uncertain, values for mammals or birds were considered to be representative within a class due to the lack of available toxicological data for most COCs. For related chemicals, such as the PAHs, if toxicity data were insufficient to represent the different chemical forms, then one toxicity value would be used from another chemical within the group.

Risk to ecological organisms is not quantified in the same manner as human health (i.e., hazard quotient or RfD). Instead, exposure point concentrations for direct contact or intakes were compared to the literature toxicity values and noted, whether in exceedence or not. When chemicals exceed the toxicity value this indicates a potential threat to ecological health may exist and further evaluation of ecological risk is appropriate for those areas.

Table 5-7. Literature Derived Toxicity Values Considered in the Exposure Assessment for Ecological Risk.

Chemical of Concern	Receptors		
	Vegetation	Birds	Small Mammals
Lead	125	72	294
Benzo(a)pyrene	NA	NA	0.002
Benzo(a)anthracene	NA	NA	2.00
Benzo(b)fluoranthene	NA	NA	40
Benzo(k)fluoranthene	NA	NA	72
Indeno(1,2,3cd)pyrene	NA	NA	72
Chrysene	NA	NA	99
Anthracene	NA	NA	3300

NA = Data for toxicity values is unavailable.

Sources: Eisler, 1987; ESE, 1991.

When the toxicity profile data was evaluated to develop appropriate references values, the following assumptions were made regarding the quality and applicability of the data:

- Chronic was preferred to acute,
- Similar species data were preferred to non related species,
- Only oral route of exposure data were considered to be consistent with exposure assessment, and
- Only nonhuman data were considered.

Each of the areas of concern was addressed qualitatively and then quantitatively. The quantitative assessment is expressed as less than one (exposure/toxicity value) or greater than one. Values less than one indicate exposure does not exceed the toxicity value. Although the exposure estimates are uncertain they are believed to be conservative because the maximum observed concentration was used to represent exposure point concentrations, and the contaminated source is assumed to be the total source of intake.

5.2.1 RISKS ASSOCIATED WITH THE AREAS OF CONCERN.

5.2.1.1 Area 902

The intakes due to ingestion exposure of contaminated soils at area 902 appear to be low for all receptors of concern. Due to lack of toxicological information for individual PAH compounds, the literature toxicity value for benzo(a)pyrene was used for quantitative comparisons to environmental concentrations. Results of the quantitative analysis indicate there is no risk to ecological receptors being exposed to soil contamination at area 902. Risks to nonhuman receptors are presented in Table 5-8.

5.2.1.2 Area 1202

The intake rates for receptors being exposed to area 1202 related soil contamination appear low. Intake rates of lead via ingestion are probably overconservative since the lead associated with soil is probably not completely available for uptake. Results of the quantitative risk analysis indicate there is no risk to ecological receptors at area 1202. Risks to nonhuman receptors are presented in Table 5-9.

5.2.1.3 Area 1602

Only four chemicals of concern were identified in area 1602 soil and the chemical intake rates of these chemicals are low for nonhuman receptors. Quantitative evaluation of risk reveals no potential risk is present at area 1602. Results of the quantitative risk calculations are presented in Table 5-10.

5.2.1.4 Site 22

With deep groundwater being the only media sampled at Site 22, risks to ecological receptors could not be addressed.

Table 5-8. Comparison of Nonhuman Chemical Intake Concentrations to Toxicity Values for Area 902.

Chemical of Concern	Receptors		
	Vegetation	Birds	Small Mammals
Lead	<1	<1	<1
Benzo(a)anthracene	NA	NA	<1
Chrysene	NA	NA	<1
Benzo(b)fluoranthene	NA	NA	<1
Benzo(a)pyrene	NA	NA	<1
Benzo(k)fluoranthene	NA	NA	<1
Indeno(1,2,3cd)pyrene	NA	NA	<1*
Pyrene	NA	NA	<1*
Fluoranthene	NA	NA	<1*
Phenanthrene	NA	NA	<1*
Benzo(ghi)perylene	NA	NA	<1*
Acenaphthene	NA	NA	<1*
Anthracene	NA	NA	<1
Fluorene	NA	NA	<1*

NA = Data for Toxicity values is unavailable.

* = Quantitative comparison based upon evaluation using literature derived toxicity value for benzo(a)pyrene.

Source: ESE, 1991

Table 5-9. Comparison of Nonhuman Chemical Intake Concentrations to Toxicity Values for Area 1202.

Chemical of Concern	Receptors		
	Vegetation	Birds	Small Mammals
Lead	<1	<1	<1
Benzo(a)anthracene	NA	NA	<1
Chrysene	NA	NA	<1
Benzo(b)fluoranthene	NA	NA	<1
Benzo(a)pyrene	NA	NA	<1
Benzo(k)fluoranthene	NA	NA	<1
Indeno(1,2,3cd)pyrene	NA	NA	<1
Pyrene	NA	NA	<1*
Fluoranthene	NA	NA	<1*
Phenanthrene	NA	NA	<1*
Acenaphthene	NA	NA	<1*
Anthracene	NA	NA	<1
Fluorene	NA	NA	<1*

NA = Data for Toxicity values is unavailable.

* = Quantitative comparison based upon evaluation using literature derived toxicity value for benzo(a)pyrene.

Source: ESE, 1991.

Table 5-10. Comparison of Nonhuman Chemical Intake Concentrations to Toxicity Values for Area 1602.

Chemical of Concern	Receptors		
	Vegetation	Birds	Small Mammals
Lead	<1	<1	<1
Phenanthrene	NA	NA	<1*
Naphthalene	NA	NA	<1*
2-methylnaphthalene	NA	NA	<1*

NA = Data for Toxicity values is unavailable.

* = Quantitative comparison based upon evaluation using literature derived toxicity value for benzo(a)pyrene.

Source: ESE, 1991.

5.3 UNCERTAINTY ANALYSIS

The risk calculations conducted for Superfund site risk assessments are not fully probable estimates of risk, given the use of conditional exposure assumptions and toxicity assumptions. There is uncertainty associated with the exposure assessment due to data limitations, exposure assumptions, and intake variable assumptions. In addition, there are uncertainties inherent in the calculations of risk as the dose-response information are estimates derived from animal studies extrapolated to human exposure. A summary of the assumptions used to assess exposure and risk, and the uncertainty associated with each, are discussed in the following sections.

5.3.1 EXPOSURE ASSESSMENT

5.3.1.1 Data Limitations

A limited number of samples were collected during the 1991 field activities. The resulting data did not justify the completion of UCL95 values for use as exposure concentrations. Instead, maximum concentrations were chosen for the exposure concentrations because this would represent of the worst case scenario.

There is uncertainty associated with the quantifiable quality of the data. Data for most of the inorganic chemicals were detected at concentrations that fell within a range above the instrument detection limit, but below the contract required detection limit (B qualified). Data of this kind has reasonable certainty in the reported concentration.

Data for most of the organic chemicals fell below the detection limits of the instrument. A number of data points were reported as J qualified data (i.e., semivolatile chemicals), meaning the value was estimated either for a tentatively identified compound or when a compound is present (spectral identification criteria are met, but the value is less than the contract required detection limit). Data of this nature are of a quality that is usable for risk assessments, however, there is uncertainty associated with the reported concentration of the chemical (but not in its assigned identity). Results of the 1991 chemical analyses yielded detections of semivolatiles below reported instrument detection limits. These concentrations were J Qualified and, therefore, were used in the quantitation of this risk assessment. As a result, uncertainty was introduced by the use of this data, because the certainty of the data values are unknown and, therefore, the monitoring data may or may not be representative of conditions at the site.

5.3.1.2 Exposure Assumptions

The pathways of exposures chosen for the areas of concern may be overconservative as compared to real conditions. A potential pathway of exposure identified in this report was the ingestion of groundwater from the deep aquifer. Concentrations of COCs identified in monitoring wells were used to assess intake rates for the receptors onsite. In reality, the workers within the areas of concern will ingest water drawn from composited well water that was pretreated before dispersal onsite. The resulting risks that were calculated from the

groundwater ingestion exposure are, therefore, representative of a worst case scenario, when pretreatment is avoided, or somehow a well is installed within the area of concern and used for potable purposes directly.

Another pathway of exposure identified in this report includes the incidental ingestion of soil. Most of the areas within the immediate vicinity of the areas of concern are paved or covered by buildings. The actual occurrence of soil ingestion would therefore be minimal. It is concluded that the risks associated with soil ingestion are, therefore, overconservative of actual conditions onsite.

5.3.1.3 Intake Variable Assumptions

The actual likelihood of exposure pathway completion using the intake variables (as described in Appendix B) is probably low. For instance, for the purpose of establishing a current worker exposure, we assumed the worker stayed within the area of highest possible contamination for 8 hours a day, 250 days per year. Several of the buildings within the areas of concern are rather large facilities enabling the movement of workers over a large area. Therefore, the assumption of a worker being exposed to the highest source of contamination continually is unlikely.

We also assumed that the concentrations observed are going to be continuous over time. In actuality, the concentrations could either increase from continual contaminant use and disposal, or decrease from natural degradation processes. The uncertainty associated with the intake rate calculations is probably high due to all the factors discussed in these sections.

5.3.2 RISK CHARACTERIZATION

Another source of uncertainty may be due to the initial selection of substances used to characterize exposures and risk. Toxicity values for benzo(a)pyrene were used for intake rate calculations for all carcinogenic PAHs. This may be overconservative since the toxicity of these chemicals may or may not be as severe as the toxicity of BaP.

The toxicity values used for risk characterization (CSF and RfD values) are derived from animal studies, therefore, the direct use for human risk will introduce significant uncertainty. Extrapolation of animal data to human health toxicity has long been a standard practice in toxicology studies. With the use of animal data, the comparisons can be either over or underconservative because the correlation of animal toxicity to human toxicity is unknown.

For the calculation of risks, this report followed the procedures as described by EPA (1989), which states that, in the absence of adequate information, the carcinogenic risks should be treated as additive and that noncarcinogenic risks should also be treated as additive.

Available literature has not evaluated multiple chemical interactions for all the chemicals evaluated. In addition, laboratory studies have a limited direct application due to site-specific environmental factors.

Additional uncertainties are introduced into the risk characterization by assuming additivity for the calculation of hazard indexes and risk. Additional chemical interactions could be occurring that would result in a more severe toxicity to the organism (i.e., synergism). In addition, the additive approach also ignores possible antagonistic interactions that would lessen the toxicity.

6.0 SUMMARY AND CONCLUSIONS

6.0 SUMMARY AND CONCLUSIONS

The quantitative baseline risk assessment for HPIA was performed to determine if the residual contamination associated with the past disposal practices at the four areas of concern pose health risks to human and environmental receptors. The results of the risk assessment are used to identify those media and/or areas that require further evaluation in the feasibility study.

The risk assessment for HPIA evaluated the human and nonhuman health risks associated with potential exposures to contaminants identified in the surface soil and deep intermediate groundwater in the vicinity of Buildings 902, 1202, and 1602. As described in the exposure assessment, the significant current exposure pathways of concern were worker exposure to soils via direct contact (i.e., ingestion and dermal absorption) and ingestion of groundwater. Current residential populations were not addressed because the current worker receptor is more likely to be at risk due to prolonged exposure. Residents onsite occupy housing for only short periods of time (about 2 years). Because future land management plans at the site are to develop military barracks in the area, residential exposures were performed and are found in Appendix D. Risks are orders of magnitude below the EPA departure point of $1.0E-04$.

6.1 SUMMARY OF HUMAN RISKS

Based on the results of the RA for the HPIA, none of the areas evaluated resulted in an exceedance of the cancer risk range of $1.0E-04$ or an HI of one for either the groundwater or soil exposure pathways (Tables 6-1 and 6-2). Therefore, these media are excluded from further consideration, and the PAHs in the soil are excluded from consideration in the feasibility study.

The results of the cancer risk evaluation for the groundwater exposure pathway indicates that Site 22 presents a risk of $2.64E-06$, a risk that does not exceed the point of departure risk of $1.0E-04$.

6.2 SUMMARY OF NONHUMAN RISKS

Results of ecological risk characterization indicate that there is no risk associated with nonhuman receptor exposure to contaminated soil from areas 902, 1202, and 1602. Risk associated with groundwater was not evaluated since this exposure pathway was not considered feasible for any of the areas of concern.

6.3 REMEDIAL ACTION OBJECTIVES FOR PAHS IN SOIL

To determine the point at which the overall remedial goal for the study area is achieved, site-specific remedial action objectives must be identified. Remedial action objectives are media-specific goals for protecting human health and the environment; consequently, the remedial alternatives must meet these goals. These goals may be contaminant-specific levels or health-and risk-based guidelines to be followed in conducting remedial actions

Table 6-1. Summary of Carcinogenic Risks Associated With Potential Worker Exposure to Hadnot Point.

COC	Area 902			Area 1202		Site 22	
	Soil	GW	Total	Soil	GW	GW	Total
PAHs	2.6E-05	--	2.6E-05	1.7E-05	--	--	1.7E-05
Benzene	--	2.0E-07	2.0E-07	--	--	2.80E-6	2.80E-6
1,2-DCE (total)	--	--	--	--	--	--	--
Lead	--	--	--	--	--	--	--
GRAND TOTAL			2.6E-05 ^(a)			2.80E-6	1.98E-5 ^(b)

1,2-DCE = 1,2-Dichloroethene
 -- = Below detection limit

^a 99 percent of total risk due to PAHs in soil

^b 86 percent of total risk due to PAHs in soil

Source: ESE, 1991

at the site. Thus, prior to evaluating remedial alternatives for PAHs in soil during the feasibility study, potential applicable or relevant and appropriate requirements (ARARs), and development of health- and risk-based guidelines for those contaminants having no ARARs, must be identified.

Based on the guidance in OSWER Directive 9355.0-3 (dated April 22, 1991), which states in part "...(w)here the cumulative carcinogenic site risk to an individual based on reasonable maximum exposure for both current and future land use is less than $1.0E-04$, and the non-carcinogenic hazard quotient is less than 1, action generally is not warranted unless there are adverse environmental impacts...". With these in mind, and considering the HPIA sites, no further remedial action is necessary, and the soils contaminated by PAH's will not be addressed in the feasibility study.

7.0 REFERENCES

7.0 REFERENCES

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APPENDIX A
TOXICITY PROFILES FOR CHEMICALS OF CONCERN

BENZENE

Benzene is a major industrial chemical made from coal and oil. It is used to make other intermediate chemicals, as well as types of plastics, detergents and pesticides. Benzene is a component of gasoline and can be found naturally in the environment produced by volcanoes and forest fires (EPA, 1984). Benzene has a vapor pressure of 95.2 mm Hg at 25°C and readily volatilizes from water and air (EPA, 1979b). Photooxidation of benzene is the most likely chemical fate process following its release to air. The half-life of benzene for air and water is approximately 6 and 1 to 6 days respectively (EPA, 1979b).

A range of soil-water partition coefficients (94 - 343) have been reported (Rogers *et al.*, 1980; Lyman *et al.*, 1982; Lyman and Loreti, 1986; and Kadeg *et al.*, 1986). Benzene has a water solubility of 1,750 mg/L at 25°C which would indicate that benzene will exhibit some environmental mobility (EPA, 1979b).

HEALTH EFFECTS ASSESSMENT

Benzene is a recognized human carcinogen (IARC, 1982). Several studies have provided sufficient evidence to indicate that benzene exposure is correlated to the incidence of leukemia in humans. Benzene is known to induce aplastic anemia in humans with a latency period of up to 10 years. In both animals and humans benzene exposure is correlated to chromosomal damage and is fetotoxic (IARC, 1982; EPA, 1979b).

Exposure to high concentrations (20,000 ppm) of benzene in air can be fatal. The most prominent symptoms of exposure include; central nervous system depression and convulsions, vertigo, drowsiness, headache, nausea and eventual unconsciousness. Death is usually the result of respiratory or cardiac failure (Holvey, 1972). Dermal exposure to benzene can cause blistering erythema and scaly dermatitis (IARC, 1982).

Animal lethality data indicate that benzene has a low oral acute toxicity (O'Bryan and Ross, 1986). The acute oral LD50 value of benzene in rats ranges from 3.4 g/kg to 5.6 g/kg depending upon the age of the animal (EPA, 1980a). The acute oral LD50 for the mouse was reported as 4.7 g/kg (EPA, 1980a).

ECOTOXICITY

Aquatic Organisms

A range of EC50 values have been reported for benzene for freshwater invertebrate and vertebrate species. Six fresh water species of fish have been tested with benzene. The resulting LC50 values ranged from 5,300 ug/L for rainbow trout to 386,000 ug/L for the mosquito fish (*Gambusia affinis*). However, only values for the rainbow trout (5,300 µg/L) were derived using a flow through system in which the toxicant concentration was

measured (EPA, 1980a). Results of acute exposure tests with species of freshwater invertebrates and vertebrates are presented in Table 1-1.

Several saltwater invertebrate and fish species have also been tested with benzene. Results indicate that the invertebrate EC50 values were considerably variable, with a range of values of 17,600 to 964,000 ug/L. The striped bass had 96-hour LC50 values of 10,900 and 5,100 ug/L (EPA, 1980a). A variety of exposure studies conducted on grass shrimp (*Palaemonetes pugio*) by Potera (1975) revealed LC50 values ranging from 33,500 to 90,800 ug/L, depending upon temperature, salinity and life-stage of the test organism.

Terrestrial Organisms

Information in the literature concerning exposure to wildlife could not be located.

CRITERIA AND STANDARDS

Due to the carcinogenicity of Benzene (weight of evidence = A) the ambient water criterion for the protection of human health is set at zero. Estimates of the carcinogenic risks associated with a life-time exposure from ingestion of contaminated water and aquatic organisms are 6.6, 0.66 and 0.066 ng/L for a risk of 10⁻⁵, 10⁻⁶ and 10⁻⁷ respectively (EPA, 1987b).

Existing standards for benzene in air for occupational exposure include 10 ppm (32mg/m³) and an emergency temporary level of 1 ppm by the Occupational Safety and Health Administration (NIOSH, 1974; 1977).

Table 1-1. Acute Toxicity of Benzene to Freshwater and Saltwater Organisms (page 1 of 2).

Species	LC50/EC50 (µg/l)	Species Acute Value (µg/l)	Reference
<u>FRESHWATER SPECIES:</u>			
Cladoceran <u>Daphnia magna</u>	203,000	---	U.S. EPA, 1978b
Cladoceran <u>Daphnia magna</u>	400,000	---	Canton & Adema, 1978
Cladoceran <u>Daphnia magna</u>	620,000	---	Canton & Adema, 1978
Cladoceran <u>Daphnia magna</u>	412,000	---	Canton & Adema, 1978
Cladoceran <u>Daphnia magna</u>	412,000	---	Canton & Adema, 1978
Cladoceran <u>Daphnia magna</u>	356,000	---	Canton & Adema, 1978
Cladoceran <u>Daphnia magna</u>	356,000	380,000	Canton & Adema, 1978
Cladoceran <u>Daphnia pulex</u>	345,000	---	Canton & Adema, 1978
Cladoceran <u>Daphnia pulex</u>	265,000	300,000	Canton & Adema, 1978
Rainbow trout (juvenile) <u>Salmo gairdneri</u>	5,300	5,300	DeGraeve <u>et al.</u> , 1980
Goldfish <u>Carassius auratus</u>	34,420	34,000	Pickering & Henderson, 1966
Fathead minnow <u>Pimephales promelas</u>	33,470	---	Pickering & Henderson, 1966
Fathead minnow <u>Pimephales promelas</u>	32,000	33,000	Pickering & Henderson, 1966
Guppy <u>Poecilla reticulata</u>	36,600	36,600	Pickering & Henderson, 1966
Mosquitofish <u>Gambusia affinis</u>	386,000	386,000	Wallen <u>et al.</u> , 1957

Table 1-1. Acute Toxicity of Benzene to Freshwater and Saltwater Organisms (page 2 of 2).

Species	LC50/EC50 ($\mu\text{g/l}$)	Species Acute Value ($\mu\text{g/l}$)	Reference
Bluegill <u>Lepomis macrochirus</u>	22,490	22,000	Pickering & Henderson, 1966
<u>SALTWATER SPECIES:</u>			
Pacific oyster <u>Crassostrea gigas</u>	924,000	924,000	LeGore, 1974
Copepod <u>Tigriopus californicus</u>	450,000	450,000	Korn et al., 1976
Bay shrimp <u>Crago franciscorum</u>	17,600	17,600	Benville & Korn, 1977
Grass shrimp <u>Palaeomonetes pugio</u>	27,000	27,000	Tatem, 1975
Dungeness crab (larva) <u>Cancer magister</u>	108,000	108,000	Caldwell et al., 1977
Striped bass <u>Morone saxatilis</u>	10,900	---	Meyerhoff, 1975
Striped bass <u>Morone saxatilis</u>	5,100	10,900	Benville & Korn, 1977

Source: EPA, 1978a

LEAD

Lead is used primarily in the production of storage batteries, oxides, and chemicals (including gasoline additives), as well as ammunition and various metal products, i.e., sheet lead, solder, and pipes (ATSDR, 1988). Lead, which is a heavy metal, exists in three oxidation states: 0, +2, and +4. Although lead compounds produced industrially are quite soluble, the lead compounds found in the environment are not usually mobile in normal groundwater and surface water because the lead leached from ores either becomes adsorbed to oxides or combines with carbonate or sulfate ions to form insoluble compounds (EPA, 1979). In aquatic environments, lead exists mainly as the divalent cation and becomes adsorbed onto particulate phases; however, in polluted waters, organic complexation is important (EPA, 1985). Sorption processes, whereby lead adsorbs to inorganic solids, organic materials, and hydrous iron and manganese oxides, usually control the mobility of lead, resulting in a strong partitioning of lead to bed sediments in aquatic systems (EPA, 1985). Benthic microbes can methylate lead to form tetramethyl lead, which is volatile and more toxic than inorganic lead (EPA, 1979).

HEALTH EFFECTS ASSESSMENT

Because dose-effect relationships for these low-level effects down to the lowest levels of internal exposure [blood lead levels <10 microgram per deciliter ($\mu\text{g}/\text{dL}$)] show no indication of a threshold, EPA's Reference Dose Work Group has considered it inappropriate to develop an acceptable RfD for subchronic or chronic exposures to inorganic lead (IRIS, 1990). For quantitative purposes and to provide a point of perspective in evaluating levels of health risks associated with lead exposure (a conservative estimate of an acceptable exposure level), a chronic oral daily human intake can be developed from the proposed MCL of 0.005 mg/L (53 FR 31516-31578). However, the derived daily human intake of 0.0005 mg/kg/day, assuming that the most sensitive receptor is a 10-kg child who ingests 1 L of water per day, is used only to provide a toxicity benchmark and is not applicable to less sensitive receptor populations, such as adults or less sensitive children.

EPA has classified lead as a group B2 (probable human) carcinogen by the oral and inhalation routes of exposure (IRIS, 1990). This classification indicates sufficient evidence exists to support carcinogenicity in animals but inadequate evidence of carcinogenicity in humans is available. No cancer slope factor has been calculated because, according to EPA, the current knowledge of lead pharmacology indicates that the derivation of either an oral or inhalation cancer slope factor by traditional means would not truly describe the potential risk. EPA determined the B2 classification based on the increased incidence of renal tumors observed following dietary and subcutaneous exposure of rats and mice to several soluble lead salts (IRIS, 1990). Although the most characteristic cancer response observed was bilateral renal carcinoma, other cancers such as lung tumors and gliomas (tumors formed from the interstitial tissue of the brain, spinal cord, pineal gland, posterior pituitary gland, and retina) were also observed (IRIS, 1990). Metallic lead, lead oxide, and lead tetra-alkyls have not been tested adequately to determine the weight-of-evidence classification (IRIS, 1990).

Humans are usually exposed to lead by the inhalation (occupational exposures) and oral routes (residential exposures). The noncarcinogenic effects of lead exposure do not appear to depend on the route of entry, but rather are correlated with internal exposure, usually measured as blood lead levels (ATSDR, 1988). Infants and young children are much more sensitive to lead poisoning than adults because they have greater gastrointestinal absorption efficiencies (approximately 53 percent for infants and young children versus 10 percent for adults) (Hammond, 1982; Chamberlain *et al.*, 1978). At high human exposure levels, lead produces encephalopathy, gastrointestinal effects (colic), anemia, kidney damage, electrocardiogram abnormalities, spontaneous abortion, and decreased fertility in men (ATSDR, 1988).

The effects of greatest concern from low-level exposure to lead are neurobehavioral effects, growth retardation in infants exposed prenatally and postnatally, and elevation of blood pressure in middle-aged men (ATSDR, 1988). Low-level chronic exposure to lead can also affect the synthesis of heme, a constituent of hemoglobin, which can have profound effects on fundamental metabolic and energy-transfer processes (ATSDR, 1988). In addition, low-level exposures can decrease the circulating levels of an active form of vitamin D that is responsible for the maintenance of calcium homeostasis in the body (ATSDR, 1988).

Lead has been shown to have teratogenic effects in experimental animals; however, there is little evidence that it supports teratogenicity in humans (IRIS, 1990). Results of *in vitro* and *in vivo* tests are contradictory, but tests suggest that lead is genotoxic (ATSDR, 1988).

ECOTOXICITY

Most forms of lead are toxic and can be incorporated into the body through inhalation, ingestion, dermal absorption, and placental transfer to the fetus. In general, organolead compounds are more toxic than inorganic lead compounds, biomagnification up the food chain is negligible, and young organisms are more susceptible than mature organisms (USFWS, 1988). Lead is not essential for plant growth, and excessive amounts can inhibit growth and reduce photosynthesis, mitosis, and water absorption (USFWS, 1988).

Aquatic Organisms

The toxicity of lead to aquatic species depends on the form of lead to which they are exposed and is a function of water pH and hardness. When in the presence of minerals, clays, or sand, lead is converted to a form less toxic to aquatic life and is not likely to be converted to the more toxic form under natural conditions. Free ion forms such as hydroxide, carbonate, and sulfate salts are more toxic to aquatic life or can be converted to the more toxic forms under natural conditions (EPA, 1985).

Laboratory studies performed by Everard and Denny (1985) on freshwater angiosperms, mosses, and benthic algae showed that aquatic mosses are extremely efficient at sorbing lead in solutions containing <1.0 mg/L of lead. In an algal/angiosperm association, algae takes up most of the lead, thus decreasing the concentration of lead reaching the angiosperm (Everard and Denney, 1985). Because algae are continually grazed and replaced by new growth, lead is easily transferred into the food web.

Some studies have shown that uptake of lead by submerged angiosperms, bryophytes, and algae is mainly passive and occurs when sediments disturbed by turbulence release lead (Welsh and Denny, 1980). Behan *et al.* (1979) found that the roots of rooted aquatic plants contain more lead than shoots.

Lead in the form of free ions of hydroxide, carbonate, and sulfate salts, which are commonly found in waterbodies, are more toxic to aquatic life or can be converted to more toxic forms under natural conditions (EPA, 1985). Water hardness and pH also affect lead toxicity. Data compiled by EPA (1985) indicate that lead is more toxic to organisms such as rainbow trout (*Oncorhynchus mykiss*), fathead minnow (*Pimephales promelas*), bluegill (*Lepomis macrochirus*), and water flea (*Daphnia magna*) in soft water than in hard water. Acute LC50 and EC50 toxicity values of lead to freshwater invertebrate species range from 124 $\mu\text{g/L}$ for an amphipod (*Gammarus pseudolimnaeus*) to $224,900$ $\mu\text{g/L}$ for a midge (*Tanytarsus dissimilis*). LC50 values for fish range from 300 $\mu\text{g/L}$ to $56,000,000$ $\mu\text{g/L}$ (Table 1-2). Different species exhibit different sensitivities to lead; amphipods were reported to be more sensitive to lead than any other freshwater animals in acute and chronic tests (EPA, 1985).

Borgmann *et al.* (1978) conducted a chronic bioassay test to observe the effects of lead on rates of mortality, growth, and biomass production of snails (*Lymnaea palustris*) when exposed to low levels of lead throughout their life cycle. Concentrations of lead as low as 19 $\mu\text{g/L}$ significantly decreased survival but not growth or reproduction. The NOEL for survival was reported at 12 $\mu\text{g/L}$, and almost complete mortality was observed at 54 $\mu\text{g/L}$ (Table 1-3).

Birdsall *et al.* (1986) reported elevated lead concentrations (up to 270 mg/kg dry weight) in tadpoles collected near heavily traveled highways, which may affect lead levels in wildlife that eat tadpoles. Fish tend to accumulate little lead in edible tissues; however, invertebrates can accumulate high levels. Demayo *et al.* (1982) report that concentrations of waterborne lead >10 $\mu\text{g/L}$ are expected to produce long-term effects on fish and fisheries. Mosquito larvae also accumulate lead. BCF values derived for aquatic life are presented in Table 1-4.

Terrestrial Organisms

Table 1-2. Acute Toxicity of Lead to Freshwater Aquatic Organisms (page 1 of 2).

Species	Range of Water Hardness (mg/L as CaCO ₃)	n*	Test Duration	Range of LC50 or EC50 (µg/L)	Reference
ALGAE					
<u>Chlamydomonas reinhardtii</u>	--	2	24 hours	4,140 to 17,000	Irmer <u>et al.</u> , 1986; Malanchuk and Gruending, 1973
<u>Microcystis aeruginosa</u>	--	1	8 days	450	EPA, 1985
<u>Scenedesmus quadricauda</u>	--	1	96 hours	2,500	Bringmann and Kuhn, 1959
INVERTEBRATES					
<u>Daphnia magna</u>	54 to 152	4	96 hours	612 to 1,910	EPA, 1985; Anderson, 1948
<u>Gammarus pseudolimnaeus</u>	46 to 48	2	96 hours	124 to 140	Spehar <u>et al.</u> , 1978; Call <u>et al.</u> , 1983
<u>Tanytarsus dissimilis</u>	48	1	96 hours	224,000	Call <u>et al.</u> , 1983
<u>Asellus meridianus</u>	--	2	48 hours	280 to 3,500	Demayo <u>et al.</u> , 1982
<u>Orconectes limosus</u>	--	1	96 hours	3,300	Boutet and Chaisemartin, 1973

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Table 1-2. Acute Toxicity of Lead to Freshwater Aquatic Organisms (page 2 of 2)

Species	Range of Water Hardness (mg/L as CaCO ₃)	n*	Test Duration	Range of LC50 or EC50 (µg/L)	Reference
FISH					
<u>Pimehales promelas</u>	20 to 360	8	96 hours	5,580 to 482,000	Pickering and Henderson, 1966; NRCC, 1973
<u>Oncorhynchus mykiss</u>	28 to 353	7	96 hours	1,170 to 542,000	Demayo <u>et al.</u> , 1982; Goettl <u>et al.</u> , 1972; Davies & Everhart, 1973; Davies <u>et al.</u> , 1976
<u>Lepomis macrochirus</u>	20 to 360	2	96 hours	23,800 to 442,000	Pickering and Henderson, 1966
<u>Micropterus dolomieu</u>	152	2	96 hours	2,800 to 28,000	Coughlan <u>et al.</u> , 1986
<u>Salvelinus fontinalis</u>	44	2	96 hours	3,362 to 4,100	Holcombe <u>et al.</u> , 1976
<u>Gasterosteus aculeatus</u>	--	1	96 hours	300	Wong <u>et al.</u> , 1978
<u>Puntius conchoni</u>	--	1	96 hours	379	Kumar and Pant, 1984
<u>Gambusia affinis</u>	--	1	96 hours	56,000,000	Wallen <u>et al.</u> , 1957

Note: -- = value not available.

*Number of tests used to calculate range values.

Source: EPA, 1985

Table 1-3. Chronic Toxicity of Lead to Freshwater Aquatic Organisms (page 1 of 2).

Species	Range of Water Hardness (mg/L as CaCO ₃)	n*	Test Duration	Effect	Effect Concentration (µg/L)	Reference
ALGAE						
<u>Microcystis aeruginosa</u>	--	1	8 days	Incipient inhabition	450	Bringmann and Kuhn, 1959
<u>Scenedesmus quadricauda</u>	--	1	8 days	Incipient inhabition	3,700	Bringmann and Kuhn, 1959
INVERTEBRATES						
<u>Daphnia magna</u>	45	1	21 days	LC50	300	Biesenger and Christensen, 1972
<u>Gammarus pseudolimnaeus</u>	46	1	28 days	LC50	28.4	Spehar <u>et al.</u> , 1978
<u>Tanytarsus dissimilis</u>	47	1	10 days	LC50	258	Anderson <u>et al.</u> , 1980
<u>Ephemerella grandis</u>	50	1	14 days	LC50	3,500	Nehring, 1976
<u>Daphnia magna</u>	52 to 151	3	Lifetime	MATC	9 to 193	Chapman <u>et al.</u> , 1985
<u>Lymnaea palustris</u>	--	1	Lifetime	MATC	12 to 54	Borgmann <u>et al.</u> , 1978

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Table 1-3. Chronic Toxicity of Lead to Freshwater Aquatic Organisms (page 2 of 2).

Species	Range of Water Hardness (mg/L as CaCO ₃)	n*	Test Duration	Effect	Effect Concentration (µg/L)	Reference
FISH						
<u>Oncorhynchus mykiss</u>	28 to 353	7	Lifetime	MATC	4 to 360	Davies <u>et al.</u> , 1976; Demayo <u>et al.</u> , 1982
<u>Salvelinus fontinalis</u>	44	1	Lifetime	MATC	58 to 119	Holcombe <u>et al.</u> , 1976
<u>Lepomis macrochirus</u>	41	1	Lifetime	MATC	70 to 120	EPA, 1985
<u>Ictalurus punctatus</u>	36	1	Lifetime	MATC	75 to 126	EPA, 1985
<u>Catostomus commersoni</u>	34	1	Lifetime	MATC	119 to 253	Demayo <u>et al.</u> , 1982
<u>Salvelinus namaycush</u>	33	1	Lifetime	MATC	48 to 83	EPA, 1985
<u>Esox lucius</u>	34	1	Lifetime	MATC	253 to 483	Demayo <u>et al.</u> , 1982

Note: MATC = maximum acceptable toxicant concentration. Lower value in each MATC pair indicates highest concentration tested producing no measurable effect on growth, survival, reproduction, and metabolic upset during chronic exposure; higher value indicates lowest concentration tested producing a measurable effect.

-- = value not available.

*Number of tests used to calculate range values.

Source: EPA, 1985

Table 1-4. Bioaccumulation of Lead in Freshwater Aquatic Organisms (page 1 of 2).

Species	Tissue	n*	Test Duration	BCF	Reference
ALGAE					
<u>Selenastrum capricornutum</u>	Whole body	2	28 days	26,000 to 92,000	Vighi, 1981
<u>Chlamydomonas reinhardtii</u>	Whole body	2	3 hours	20 to 26	Irmer <u>et al.</u> , 1986
INVERTEBRATES					
Aquatic invertebrates	Whole body	1	28 days	1,000 to 9,000	Demayo <u>et al.</u> , 1982
<u>Lymnaea palustris</u>	Whole body	1	120 days	1,700	Borgmann <u>et al.</u> , 1978
<u>Physa integra</u>	Whole body	1	28 days	738	Spehar <u>et al.</u> , 1978
<u>Pteronarcys</u> sp.	Whole body	2	14 to 28 days	86 to 1,120	Spehar <u>et al.</u> , 1978; Nehring, 1976
<u>Bachycentrus</u> sp.	Whole body	1	28 days	499	Spehar <u>et al.</u> , 1978
<u>Ephemerella grandis</u>	Whole body	1	14 days	2,366	Nehring, 1976

Table 1-4. Bioaccumulation of Lead in Freshwater Aquatic Organisms (page 2 of 2).

Species	Tissue	n*	Test Duration	BCF	Reference
FISH					
<u>Oncorhynchus mykiss</u>	Whole body	1	7 days	726	Wong <u>et al.</u> , 1978
	Intestinal lipids	1	10 days	17,300	Wong <u>et al.</u> , 1978
	Intestinal lipids	1	14 days	12,540	Wong <u>et al.</u> , 1978
<u>Salvelinus fontinalis</u>	Whole body	1	140 days	42	Holcombe <u>et al.</u> , 1976
	Liver	2	2 generations	420 to 571	Wong <u>et al.</u> , 1978
	Kidney	2	2 generations	1,504 to 1,806	Wong <u>et al.</u> , 1978
<u>Lepomis macrochirus</u>	Whole body	1	--	45	Atchison <u>et al.</u> , 1977

Note: -- = value not available.

*Number of tests used to calculate range values.

Source: EPA, 1985.

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The phytotoxicity of lead to plants is low compared with other trace elements, such as zinc and copper (Adriano, 1986). Lead uptake in plants is limited by the low bioavailability of lead from the soil. NRCC (1973) and Boggess and Wilson (1977) found that low soil pH and reduced amounts of organic matter, inorganic colloids, iron oxide, and phosphorus enhance the bioavailability of lead.

Phytotoxicity results from interference with leaf stomatal diffusion, mitochondrial respiration, photosynthesis, and ion uptake and translocation (Adriano, 1986). Plant mortality was reported at a soil solution concentration of 50 mg/L; plant toxicity was reported at 25 mg/L and at soil concentrations of 400 to 500 mg/kg (Adriano, 1986). EPA (1986) reports that a tolerable level of 250 mg/kg for total soil lead is based on "no effect" to alfalfa, oats, and rye grass at this level; one exception was corn seedlings, which evidenced stunted seedlings at 125 mg/kg. From this information, a total soil lead concentration of 125 mg/kg is recommended as protective of vegetation.

Several studies have been conducted on lead accumulation in agricultural crops. Sadiz (1985) found that corn grown in soils containing 786 mg/kg of lead accumulated 17 mg/kg, and corn grown in soils containing 924 mg/kg of lead accumulated 30 mg/kg. Soil levels of 12 mg/kg reduced reproduction in corn (Krishnayya and Bedi, 1986).

Beyer and Anderson (1985) found that survival and reproduction were reduced in woodlice (Porcellio scaber) fed soil litter treated with 12,800 mg/kg of lead oxide for 64 weeks, or two generations. Lead at similar concentrations can eliminate or reduce populations of bacteria and fungi on leaf surfaces and in soil. Many of these microorganisms are important decomposers (USFWS, 1988). EPA (1985) reported that in soft water (99 mg/L as CaCO₃), some marbled salamanders (Ambystoma opacum) exposed to 1.4 mg/L of lead died within 8 days.

Reports of lead poisoning in wild animals usually involve waterfowl. Single oral doses of lead shot (200 to 1,400 mg) can cause acute or chronic effects in mallard ducks. In studies with mourning doves (Zenaida macroura) conducted by Buerger *et al.* (1986), an oral dose of 72 mg produced 24-percent mortality within 1 month as well as significantly reduced egg hatching. Cases of lead poisoning have been reported for a variety of domestic animals, including cattle, horses, dogs, and cats. Anthropogenic sources such as stack emissions are cited as the lead source. Relatively low levels of exposure in food can cause fatalities when organisms forage in contaminated areas. As its potential toxicity in the food web increases, lead accumulates in tissues. Results of acute and chronic studies on terrestrial vertebrates are presented in Tables 1-5 and 1-6, respectively.

CRITERIA AND STANDARDS

Table 1-5. Acute Toxicity of Lead to Terrestrial Vertebrates (page 1 of 3).

Species	Dose	Chemical	How Administered	Effect	Reference
<u>Bovine</u> sp.	220 to 400 mg/kg-bw	Lead-acetate	Single oral dose	LD50	Zmudski <i>et al.</i> , 1983
	50 to 100 g	Lead	--	Toxic	Zmudski <i>et al.</i> , 1983
<u>Canis familiaris</u>	10 to 25 g	Lead	--	Toxic	Zmudski <i>et al.</i> , 1983
<u>Cavia cabaya</u>	25 mg/kg-bw	Lead-acetate	Intraperitoneal injection	Reduced brain weight of young	Edwards and Beatson, 1984
<u>Equus caballus</u>	500 to 700 g	Lead	--	Toxic	Zmudski <i>et al.</i> , 1978
<u>Mus</u> . sp. (pregnant)	20 mg/kg-bw	Lead	Intrauterine injection	Small litters, increased fetal deaths	Wide, 1985
<u>Rattus</u> sp.	80 mg/kg-bw	Tetramethyl lead	Intravenous injection	LD50	Branica and Konrad, 1980
	8 mg/kg-bw	Triethyl lead	Intravenous injection	LD50	Branica and Konrad, 1980
	108 mg/kg-bw	Tetramethyl lead	Single oral dose	LD50	Branica and Konrad, 1980
	12 mg/kg-bw	Tetraethyl lead	Single oral dose	LD50	Branica and Konrad, 1980
	5 mg/kg-bw	Triethyl lead	Intraperitoneal injection	LD50	Branica and Konrad, 1980

Table 5 Acute Toxicity of Lead to Terrestrial Vertebrates (page 2 of 3).

Species	Dose	Chemical	How Administered	Effect	Reference
<u>Anas platyrhynchos</u>	1 g	No. 6 lead shot	Single oral dose	9-percent mortality in 20 days	Longcore <u>et al.</u> , 1974
	6 g	No. 6 lead shot	Single oral dose	50-percent mortality in 20 days	Longcore <u>et al.</u> , 1974
	8 g	No. 6 lead shot	Single oral dose	100-percent mortality in 80 days	Longcore <u>et al.</u> , 1974
	107 mg/kg	Tetraethyl lead	Single oral dose	LD50	Hudson <u>et al.</u> , 1984
<u>Coturnix japonica</u>	24.6 mg/kg	Tetraethyl lead	Single oral dose	LD50	Hudson <u>et al.</u> , 1984
<u>Haliaeetus leucocephalus</u>	2 g	No. 4 lead shot	Oral	Death from 10 to 133 days posttreatment	Pattee <u>et al.</u> , 1981
<u>Streptopelia risoria</u>	440 to 488 mg	No. 4 lead shot	Single oral dose	Mortality in cold (<10°C) stressed birds	Kendall and Scanlon, 1984

Table 1-5. Acute Toxicity of Lead to Terrestrial Vertebrates (page 3 of 3).

Species	Dose	Chemical	How Administered	Effect	Reference*
<u>Zenaida macroura</u>	144 to 288 mg	No. 8 lead shot	Single oral dose	52- to 60-percent mortality in 4 weeks	Buerger <u>et al.</u> , 1986
	72 mg	No. 8 lead shot	Single oral dose	24-percent mortality in 4 weeks	Buerger <u>et al.</u> , 1986

Note: mg/kg-bw = milligrams per kilogram-body weight.
 -- = value not available.

Source: USFWS, 1988

Table 1-6. Chronic Toxicity of Lead to Terrestrial Vertebrates (page 1 of 2).

Species	Exposure (days)	Total Dose (mg/kg-bw)	How Administered	Chemical	Effect	Reference
<u>Bovine</u> sp. (calves)	7 to 20	35 to 54	Milk	Lead-acetate	Death	Zmudzki <u>et al.</u> , 1983
	7 to 90	32 to 315	Grain or hay	Lead	0 to 1.5-percent mortality	Zmudzki <u>et al.</u> , 1983
	105	630 to 735	Oral	Lead-acetate	Death	Zmudzki <u>et al.</u> , 1983
	8 to 60	160 to 440	--	Lead	Death	Zmudzki <u>et al.</u> , 1983
	10 to 20	50 to 100	--	Lead	16-percent mortality	Demayo <u>et al.</u> , 1982
	1,095	5,475 to 6,570	--	Lead	Chronic toxicity	Zmudzki <u>et al.</u> , 1983
<u>Bovine</u> sp. (adults)	1,095	6,570	--	Lead	No effect	NRCC, 1973
<u>Canis familiaris</u>	84 to 180	294 to 540	Oral	Lead-carbonate	Anorexia, anemia, renal necrosis	Clark, 1979
<u>Equus caballus</u>	105	656 to 1,029	Oral	Lead-acetate	No deaths	Zmudzki <u>et al.</u> , 1983
	84 to 100	621 to 740	Contaminated hay	Lead	100-percent mortality	Burrows and Borchard, 1982
	113 to 304	1,130 to 3,040	Oral	Lead-acetate	100-percent mortality	Burrows and Borchard, 1982

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Table 1-6. Chronic Toxicity of Lead to Terrestrial Vertebrates (page 2 of 2).

Species	Exposure (days)	Total Dose (mg/kg-bw)	How Administered	Chemical	Effect	Reference
<u>Sturnus vulgaris</u>	6	168	Capsule	Triethyl lead chloride	100-percent mortality	Osborn <u>et al.</u> , 1983
	11	30.8	Capsule	Triethyl lead chloride	Reduced growth	Osborn <u>et al.</u> , 1983

Note: ALAD = δ -amino levulinic acid dehydratase.
 -- = value not available.

Source: USFWS, 1988

AWQC are available for lead in freshwater systems. The 4-day (chronic) average concentration in $\mu\text{g/L}$ of lead should not exceed more than once every 3 years on the average the numerical value given by:

$$e^{(1.273(\ln(\text{hardness}))-4.705)}$$

The 1-hour (acute) average concentration in $\mu\text{g/L}$ of lead should not exceed more than once every 3 years on the average the numerical value given by:

$$e^{(1.273(\ln(\text{hardness}))-1.460)}$$

At a hardness of 100 mg/L as CaCO_3 , the chronic and acute criteria are 3.2 $\mu\text{g/L}$ and 82 $\mu\text{g/L}$, respectively.

1,2 DICHLOROETHENE

Dichloroethenes consist of three isomers: 1,2-dichloroethene (1,1-DCE), cis-1,2-dichloroethene (cis-1,2-DCE) and trans-1,2-dichloroethene (trans-1,2-DCE). The trans isomer widely used in industry, the trans- isomer is used more often than either the cis- isomer or a commonly available mixture. It is primarily used as a low temperature extraction solvent and as a solvent in a select number of manufacturing processes.

Dichloroethenes are clear colorless liquids with the molecular formula of $C_2H_2Cl_2$. The cis isomer of 1,2 DCE has a water solubility of 3,500 ug/ml, a vapor pressure of 208 mm Hg and a melting point of -80.5 C; trans 1,2-DCE has a water solubility of 6,300 ug/ml, a vapor pressure of 324 mm Hg and a melting point of -50 C (Wessling and Edwards, 1970). 1,2 DCE is not known to occur in nature and ambient levels have not been well researched.

HEALTH EFFECTS ASSESSMENT

Like other members of the chlorinated ethylene chemicals, the DCEs have anesthetic properties. Studies by Jenkins et al. 1972. indicate that the 1,2-DCE isomers are less potent than 1,1-DCE as a hepatotoxin. Freundt, et al. (1977) indicated that repeated inhalation exposures of 800 mg/m³ for 16 weeks to the trans 1,2-DCE isomer produces fatty degeneration of the liver.

The ability of 1,2 DCE to be absorbed by the human body has not been well documented. However in a study by McKenna, implies up to 35 to 50 % of inhaled DCEs and up to 100 % ingested DCEs may be absorbed systemically (1977 a and b). DCEs are metabolized through epoxide intermediates which are reactive and may form covalent bonds with tissue macromolecules (Henschler, 1977). Metabolism of the cis 1,2-DCE isomer occurs at a more rapid rate than the trans 1,2-DCE isomer. There is relatively little literature information regarding the rate at which any of the DCEs are excreted from the body (EPA, 1980b).

EPA lists a chronic oral reference dose of 0.02 mg/kg/day for the trans- isomer (IRIS, 1990) and a value of 0.2 mg/kg/day as the interim subchronic oral reference dose for the trans- isomer (HEAST, 1990). No inhalation reference dose is provided by EPA for the trans- isomer (IRIS, 1990; HEAST, 1990). No reference doses are presented for the cis- isomer, as EPA states that the data are inadequate for quantitative risk assessment (HEAST, 1990).

EPA (IRIS, 1990) has not classified the carcinogenicity of 1,2-dichloroethylene; however, EPA (1980b) had previously classified it as a group D (not classifiable) carcinogen, acknowledging that the compound cannot be reliably grouped.

The oral reference doses were derived from a 90-day study in which mice were exposed to concentrations of 100, 1,000, and 2,000 mg/L in drinking water (IRIS, 1990). Increased serum alkaline phosphatase levels were observed in male mice at the two highest levels, which are equivalent to 175 and 387 mg/kg-bw/day. Based on this study, the NOAEL was determined to be 17 mg/kg-bw/day (100 mg/L) and the LOAEL to be 175 mg/kg-bw/day (1,000 mg/L).

Rats orally exposed subchronically to a mixture of the isomers at doses as high as 1,000 mg/kg evidenced no effects following 7 weeks of exposure. A single dose of the cis- isomer at a level as low as 400 mg/kg-bw resulted in liver damage, with the authors stating that the trans- isomer is slightly less toxic than the cis- isomer (EPA, 1980b). When exposed to air concentrations of 200 ppm for up to 8 hours, inhibition of the mixed-function oxidase (MFO) system was reported, with the cis- isomer reported as more potent than the trans- isomer (EPA, 1980b). No chronic inhalation data were identified in the literature; an unpublished study indicated no effects in several species of experimental animals exposed up to 1,000 ppm of mixed isomers for 6 months (EPA, 1980b).

ECOTOXICITY

Aquatic Organisms

Most of the available data for dichloroethenes are for the 1,1 DCE isomer. The bluegill was tested with both 1,1-DCE and 1,2- DCE under similar conditions. Results of the 96 hour static exposure yielded LC50 values of 73,900 and 135,000 ug/L for 1,1-DCE and 1,2-DCE respectively. It was concluded that the location of the chlorine atoms on the molecule does not affect the acute toxicity of dichloroethenes very much.

1,2 DCE has an estimated steady-state bioconcentration factor of 4.0 in fish and shell fish. This value was estimated using the equation:

$$\text{Log BCF} = (0.85 \text{ Log } P) - 0.70$$

A calculated log P value of 1.53 was used with an adjustment factor of 0.395 (EPA, 1980b).

Terrestrial Organisms

No information was found in the available literature concerning the toxicity of 1,2-dichloroethylene to vegetation. No toxicity information was identified concerning livestock or terrestrial wildlife.

CRITERIA AND STANDARDS

The available data for dichloroethenes indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 11,600 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available for the chronic toxicity of DCEs to aquatic life.

A criterion cannot be derived at this time due to the insufficiency in the available data for 1,2 DCE.

PAHs

PAHs are a class of compounds consisting of substituted and unsubstituted polycyclic and heterocyclic aromatic rings. PAHs are formed by the incomplete combustion of organic compounds in the presence of insufficient oxygen. PAHs occur from both natural and anthropogenic sources. As a group, they are widely distributed in the environment and found in animal and plant tissue, sediments, air and surface water (Radding et al., 1976). Formed during the incomplete combustion of organic matter, PAHs are common constituents of tar, soot, petroleum products, engine lubricant wastes, tobacco smoke, automotive exhaust, smoked meats, fried foods, and creosote-treated wood (EPA, 1980c).

Anthracene group compounds are 3- and 4-ring PAHs (Table 1-7). On a commercial basis, these compounds have been imported in small quantities for special uses and are common constituents of coal tars, typically used in creosotes. Anthracene group compounds have possible carcinogenicity that might require careful reviews. They have moderately low volatility and water solubility. Due to their relatively low water solubilities and fairly high K_{ow} values, adsorption unto both organic and inorganic matter is a primary removal pathway for these compounds in the water column. The fraction of these PAHs that remains in the water column is expected to undergo photolytic degradation (Zepp and Schlotzhauer, 1979), the extent of which is affected by turbidity and light penetration. Volatilization from the aqueous phase is not expected to be a major fate process.

Benzo(a)pyrene (BaP) group chemicals (Table 1-7), in pure forms, are typically used as research laboratory standards. They have no commercial production or use other than as constituents of coal tars and coal tar containing creosotes. They generally have low vapor pressures and water solubilities, with the exception of acenaphthylene, which is soluble and volatile. Some of the PAHs in this group have been identified as carcinogens.

Adsorption to organic matter, with subsequent transport away from the water column, and photolysis are the more significant fate processes for these compounds in the aquatic environment. Volatilization and biodegradation are expected to be slow for PAHs.

Few data are available specific to individual PAH compounds; therefore, their aquatic fate is inferred from data summarized for PAH compounds in general. Most PAHs absorb solar radiation strongly and may, therefore, undergo direct photolysis or photooxidation (Radding et al., 1976). In the aqueous environment, photolysis is rapid for BaP and benzo(a)anthracene. Smith *et al.* (1978) reported half-lives in water of 1.2 hours and 1 to 2 hours, respectively. In contrast, hydrolysis is not thought to be a significant fate process because PAH compounds do not contain groups amenable to hydrolysis (Radding *et al.*, 1976).

Table 1-7. Physical and Chemical Properties of PAHs (page 1 of 2).

PAH	Molecular Weight (g/mole)	Water Solubility (mg/L, 25°C)	Octanol/Water Partition Coefficient (log K_{ow})	Vapor Pressure (torr)	Henry's Law Constant (atm m ³ /mole)	Organic Carbon Partition Coefficient, K_{oc} * (mL/g)
<u>Naphthalene Group</u>						
Naphthalene	128.19	31.7	3.29	0.09	4.8×10^{-4}	8.51×10^2
<u>Anthracene Group</u>						
Anthracene	178.24	0.045	4.45	2.4×10^{-4}	1.25×10^{-3}	1.4×10^4
Acenaphthene	154.21	3.42	4.33	1.6×10^{-3}	9.33×10^{-5}	4.6×10^3
Fluoranthene	202.26	0.26	5.33	5×10^{-6}	5.12×10^{-6}	3.8×10^4
Fluorene	166.23	1.69	4.18	1×10^{-2}	1.29×10^{-3}	7.3×10^3
Phenanthrene	178.24	1.00	4.46	9.6×10^{-4}	2.25×10^{-4}	1.4×10^4
Pyrene	202.26	0.14	5.32	2.5×10^{-6}	4.75×10^{-6}	3.8×10^4
<u>BaP Group</u>						
BaP	252.32	0.0038	6.08	5.6×10^{-9}	4.89×10^{-7}	5.5×10^6
Acenaphthylene	152.21	3.93	3.72	0.030	1.52×10^{-3}	2.5×10^3

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Table 1-7. Physical and Chemical Properties of PAHs (page 2 of 2).

PAH	Molecular Weight (g/mole)	Water Solubility (mg/L, 25°C)	Octanol/Water Partition Coefficient (log K_{ow})	Vapor Pressure (torr)	Henry's Law Constant (atm m ³ /mole)	Organic Carbon Partition Coefficient, K_{oc} * (mL/g)
Benz(a)anthracene	228.28	0.009	5.61	2.2×10^{-8}	7.34×10^{-7}	1.38×10^6
Benzo(b)fluoranthene	252.32	0.001	6.08	5.0×10^{-7}	1.66×10^{-4}	5.5×10^{-5}
Benzo(k)fluoranthene	252.32	0.00055	6.08	5.0×10^{-7}	3.02×10^{-4}	5.5×10^5
Benzo perylene	276.34	0.0003	6.51	1.0×10^{-10}	1.21×10^{-7}	1.6×10^6
Chrysene	228.28	0.0018	5.61	6.3×10^{-9}	1.05×10^{-6}	2.00×10^5
Dibenzo(a,h)-anthracene	278.36	0.0005	6.84	1.0×10^{-10}	7.3×10^{-8}	3.3×10^6
Indeno(1,2,3-cd)pyrene	276.34	0.0005	6.51	1.0×10^{-10}	6×10^{-10}	1.6×10^6

Note: atm m³/mole = atmospheres per cubic meter per mole.
g/mole = grams per mole.
mL/g = milliliters per gram.

Sources: EPAc and d

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Measured volatilization rates for PAHs have not been reported in the literature, so the assessment of volatilization as a transport process is only speculative. In general, the volatilization rate decreases as the vapor pressure decreases, both of which are inversely related to the number of aromatic rings. Southworth (1979) concluded that the rate of volatilization of PAHs with four or more rings is insignificant under all conditions.

Most PAH compounds adsorb onto particulate matter and are transported in water (Radding *et al.*, 1976). BaP and benzo(a)anthracene show rapid partitioning onto suspended matter, and sorption onto sediments is strongly correlated with organic carbon levels in sediments.

Bacteria have been shown to use some PAHs as a sole source of carbon for growth (Radding *et al.*, 1976). Evidence for bacterial degradation is limited, and no compound-specific information is available. Because no organisms have been isolated that are capable of using 4- or 5-ringed compounds as a sole carbon source, it is assumed that they are co-metabolized with simpler compounds.

The extent of migration of PAHs in the environment is a function of the log octanol-water and organic carbon partition coefficients (K_{ow} and K_{oc}). With comparably high K_{ow} and K_{oc} values and low water solubilities, PAHs are expected to strongly adsorb to particulate matter, particularly those high in organic content (EPA, 1982a). The low vapor pressures and Henry's law constants for these PAHs suggest that volatilization is not a primary removal mechanism from either soil or aquatic systems. In aquatic systems, the removal mechanisms for most PAHs are photochemical reactions, sorption onto particulate matter, and subsequent sedimentation and microbial degradation (EPA, 1982a). PAHs do not contain groups amenable to hydrolysis; however, direct photolysis may be an important fate process for PAHs dissolved in an aqueous environment (EPA, 1980d).

The mobility of naphthalene in soil and groundwater is strongly affected by the extent of soil sorption, which is a reversible process for this particular PAH. The potential for soil desorption and the relatively high water solubility of naphthalene suggest that migration from soil into groundwater and surface water may be an important transport process for naphthalene (EPA, 1982b). In addition, volatilization of naphthalene from aqueous solutions is reported as a significant removal process. Volatilization from soils may also be an important transport mechanism, although sorption of naphthalene vapors onto soil materials may slow vapor phase transport (EPA, 1982b). Once in the atmosphere, naphthalene is photooxidized; however, under normal soil and aqueous conditions, naphthalene is not expected to undergo hydrolysis or oxidation/reduction reactions. Although some aerobic microbial degradation is likely, this process is not expected to be a predominant fate mechanism due to the low concentration of microorganisms (at depth) and the low dissolved oxygen (EPA, 1982b).

HEALTH EFFECTS ASSESSMENT

PAHs may be separated into two specific groups: the potentially carcinogenic PAHs, which include benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, BaP, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene; and the noncarcinogenic PAHs, which include the remaining PAHs detected at the site.

Carcinogenic PAHs

EPA has not developed any RfDs for the carcinogenic PAHs (IRIS, 1990; HEAST, 1990). EPA has classified the carcinogenic PAHs as group B2 (probable human) carcinogens (HEAST, 1990), which indicates that insufficient human data are available to determine the potential carcinogenicity of the PAHs but that sufficient experimental animal data are available. EPA is currently finalizing the cancer slope factors for oral and inhalation exposures; the interim slope factors have been removed from the database (HEAST, 1990) pending final approval. Interim guidance is available from HEAST (1990) indicating that the oral and inhalation slope factors developed several years ago by EPA (1984) for BaP be used until the revised values are made available. The interim oral and inhalation values are $11.5 \text{ (mg/kg/day)}^{-1}$ and $6.1 \text{ (mg/kg/day)}^{-1}$, respectively.

The potential for carcinogenic PAHs to induce malignant tumors, both at the site of application and systemically, dominates the consideration of health hazards resulting from exposure (ICF, 1987a-e). BaP is a moderately potent experimental carcinogen in many species by various routes of exposure (ICF, 1987b). No reports directly correlate human BaP exposures and tumor development, although humans are likely to be exposed via numerous routes (ICF, 1987b). Oral administration of carcinogenic PAHs produced tumors of the forestomach in mice, and laryngeal and tracheal tumors were observed in laboratory animals following inhalation, intratracheal, and intravenous administration (ICF, 1987a-e; Clement, 1985). Dermal studies with mice indicate that carcinogenic PAHs produce skin carcinomas and lung adenomas (ICF, 1987a-e; Clement, 1985).

Generally, carcinogenic PAHs are active in mutagenic assays and have been shown to be teratogens and reproductive toxins (ICF, 1987a-e; Clement, 1985). Pertinent data regarding the reproductive and developmental toxicity of carcinogenic PAHs in humans and experimental animals following inhalation, oral, or dermal exposure could not be located in the available literature. Daily subcutaneous administration of dibenz(a,h)anthracene in rats from the first day of pregnancy, however, did result in fetal death and resorption and may have affected the fertility of the dams (ICF, 1987e). Nonmalignant effects associated with exposure to carcinogenic PAHs include liver and kidney damage, various skin disorders, and immunosuppressive effects (Clement, 1985; ICF, 1987a-e). Several dermal studies indicate that some PAHs can cause hemolymphatic changes in the lymph nodes in rats, and human dermal exposure to high concentrations of PAHs has resulted in burning, itching, dermatitis,

hyperkeratosis, and papular and vesicular eruptions, as well as phototoxic and photoallergic effects [Clement, 1985; Clayton and Clayton, 1981; International Agency for Research on Cancer (IARC), 1983].

Noncarcinogenic PAHs

Although toxicological data are not available for complete characterization of each noncarcinogenic PAH, EPA has derived oral chronic and subchronic RfDs for six noncarcinogenic PAHs. These values are (in mg/kg/day) (HEAST, 1990):

<u>Compound</u>	<u>Chronic</u>	<u>Subchronic</u>
acenaphthene	0.06	0.6
anthracene	0.3	3.0
fluoranthene	0.04	0.4
fluorene	0.04	0.4
naphthalene	0.004	0.004
pyrene	0.03	0.3

Except for naphthalene, the RfDs were based on critical oral mouse studies; the oral RfD for naphthalene is based on a rat study. For purposes of deriving a baseline risk assessment, the lowest RfD other than naphthalene (0.03 mg/kg/day for pyrene) may be used for deriving the noncarcinogenic risks associated with exposure to all PAHs, carcinogenic and noncarcinogenic, that do not have an RfD. It is not appropriate to apply the RfD for naphthalene to other PAHs because of the differences in the physicochemical and biological properties.

ECOTOXICITY

Aquatic Organisms

Little aquatic testing has been conducted with fluoranthene. Freshwater acute toxicity for fish and invertebrates is estimated to range from an LC50 value of 3.98 mg/L for bluegill sunfish (Lepomis macrochirus) to 325 mg/L for the cladoceran (Daphnia magna). The freshwater alga Selenastrum capricornutum was exposed for 96 hours to fluoranthene, and an EC50 value of approximately 54.5 mg/L was determined based on the reported reduction in cell numbers and chlorophyll a (EPA, 1980c).

The saltwater sandworm (Neanthes arenaceodentata) was the representative invertebrate tested and was most sensitive to phenanthrene (LC50 of 370 µg/L) (see Table 1-8). The LC50 values for benzo(a)anthracene and chrysene were both >1000 µg/L.

Table 1-8. Acute Toxicity of PAHs to Freshwater Aquatic Organisms

Species and Chemical	Concentration ($\mu\text{g/L}$)	Effect	Reference
<u>Lepomis macrochirus</u> (Bluegill)			
Benz(a)anthracene	1,000	LC ₈₇ (6 months)	EPA, 1980d
Fluorene	500	LC ₁₂ (30 days)	Finger <u>et al.</u> , 1985
Fluorene	910	LC ₅₀ (96 hours)	Finger <u>et al.</u> , 1985
<u>Gammarus pseudolimnaeus</u> (Amphipod)			
Fluorene	600	LC ₅₀ (96 hours)	Finger <u>et al.</u> , 1985
<u>Oncorhynchus mykiss</u> (Rainbow trout)			
Fluorene	820	LC ₅₀ (96 hours)	Finger <u>et al.</u> , 1985
<u>Pimephales promelas</u> (Fathead minnow)			
Fluorene	>100,000	LC ₅₀ (96 hours)	Finger <u>et al.</u> , 1985

Source: EPA, 1980d

A variety of species were tested using fluorene, and the data indicate that the fathead minnow (Pimephales promelas) is least sensitive (LC50 > 100,000 $\mu\text{g/L}$) and the bluegill (Lepomis macrochirus) was most sensitive (LC50 of 500 $\mu\text{g/L}$).

No chronic toxicity data for fluoranthene were found for freshwater fish or invertebrates. Existing data on a saltwater invertebrate, Mysidopsis bahia, report an acute-to-chronic ratio of 2.5 (EPA, 1980c). Based on this ratio, the estimated chronic value for the most sensitive freshwater species tested would be 1.6 mg/L.

Only phenanthrene and fluorene were used in chronic tests, and the midge (Chironomus plumosus) was more resistant to fluorene toxicity than Daphnia magna (Table 1-9). Two species were used for phenanthrene tests, and Daphnia magna (95 percent chronic index of 590 to 840 $\mu\text{g/L}$) was more resistant than the rainbow trout (95 percent chronic index of 10 to 90 $\mu\text{g/L}$).

Measured BCFs for naphthalene in rainbow trout and bluegill sunfish range from 40 to 300 (Rogers et al., 1983). Studies in fish have shown that uptake and depuration of naphthalene is rapid; therefore, once external contamination is removed, accumulated burdens would be cleared rapidly (Rogers et al., 1983). Studies with the freshwater invertebrate D. pulex report a rapid and greater accumulation from food than from water, which suggests the potential for bioaccumulation of naphthalene in secondary consumers (e.g., fish) through ingestion of zooplankton. Available data indicate that BaP will bioaccumulate. In Daphnia magna the BCF is 134,248 (Table 1-10).

Terrestrial Organisms

Plants can adsorb PAHs from soils through their roots and translocate them to other plant parts, such as developing shoots. Uptake rates were governed, in part, by PAH concentrations, PAH water solubility, soil type, and PAH physicochemical state (vapor and particulate). Lower molecular weight PAHs were absorbed by plants more readily than higher molecular weight PAHs [U.S. Fish and Wildlife Service (USFWS), 1987].

Aboveground parts of vegetables contain more PAHs than underground parts, which is attributed to airborne deposition and subsequent adsorption. The limited information available on PAH-induced phytotoxic effects indicates that these responses are rare (USFWS, 1987b). Certain plants contain chemicals (ellagic acid) known to protect against PAH effects. Ellagic acid can destroy the diol epoxide form of benzo(a)pyrene, inactivating its carcinogenic and mutagenic potential (Edwards, 1983).

Data on the biological effects of BaP, 3-methylcholanthrene, and perylene to reptiles and amphibians are limited. Data are even more limited on the effects of PAHs on avian wildlife. Two articles have discussed PAH effects

Table 1-9. Chronic Toxicity of PAHs to Freshwater Aquatic Organisms

Species and Chemical	95 Percent Chronic Index ($\mu\text{g/L}$)	Reference
<u>Chironomus plumosus</u> (Midge)		
Fluorene	1,900 to 3,000	EPA, 1980d
<u>Daphnia magna</u> (Cladoceran)		
Fluorene	330 to 550	EPA, 1980d
Phenanthrene	590 to 840	EPA, 1980d
<u>Oncorhynchus mykiss</u> (Rainbow trout)		
Phenanthrene	10 to 90	EPA, 1980d

Source: EPA, 1980d

Table 1-10. BCFs for PAHs (page 1 of 2).

Species and Chemical	Duration	BCF	Reference
<u>Daphnia magna</u> (Cladoceran)			
BaP	3 days	134,248	Lu <u>et al.</u> , 1977
<u>Daphnia pulex</u> (Cladoceran)			
Anthracene	60 minutes	200	EPA, 1980
Anthracene	24 hours	760 to 1,200	Southworth <u>et al.</u> , 1978
Benz(a)anthracene	24 hours	10,109	Southworth <u>et al.</u> , 1978
9-Methylanthracene	24 hours	4,583	Neff, 1985
BaP	3 days	134,248	Lu <u>et al.</u> , 1977
Naphthalene	24 hours	131	Neff, 1985
Pyrene	24 hours	2,702	Neff, 1985
<u>Crassostrea virginica</u> (Eastern oyster)			
BaP	14 days	242	Couch <u>et al.</u> , 1983
<u>Pimephales promelas</u> (Fathead minnow)			
Anthracene	2 to 3 days	485	Southworth, 1979
<u>Hexagenia sp.</u> (Mayfly)			
Anthracene	28 hours	3,500	EPA, 1980

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Table 1-10. BCFs for PAHs (page 2 of 2).

Species and Chemical	Duration	BCF	Reference*
<u>Oncorhynchus mykiss</u> (Rainbow trout)			
Anthracene	72 hours	4,400 to 9,200	Linder <u>et al.</u> , 1985
BaP	10 days	182 to 920	Gerhart and Carlson, 1978
Fluoranthene	21 days	379	Gerhart and Carlson, 1978
Pyrene	21 days	69	Gerhart and Carlson, 1978
<u>Physa p.</u> (Snail)			
BaP	3 days	82,231	Lu <u>et al.</u> , 1977
<u>Lepomis macrochirus</u> (Bluegill)			
Fluorene	30 days	200 to 1,800	Finger <u>et al.</u> , 1985
Naphthalene	24 hours	310	McCarthy and Jimenez, 1985

Sources: USFWS (1987b).

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to mallards (Anas platyrhynchos). In one study by Patton and Dieter (1980), birds were fed 4,000 mg PAHs/kg (mostly as naphthalenes, and phenanthrene) for a period of 7 months. Results indicated no toxic effects except an increase in liver weight by 25%.

Many PAHs produce tumors in skin and in most epithelial tissues of practically all animal species tested. Certain carcinogenic PAHs are capable of passage across skin, lungs, and intestine. PAH carcinogens transform cells through genetic injury involving metabolism of the parent compound to a reactive diol epoxide. As a result the diol epoxide can form adducts with cellular molecules, such as DNA, RNA and proteins resulting in cell transformation (Dipple 1985; Ward et al. 1985.) In a study conducted by Lo and Sandi (1978) the following concentrations of individual PAHs were found to cause carcinogenicity following chronic oral exposure in rodents.

<u>Chemical</u>	<u>Concentration (mg/kg body weight)</u>
7,12-dimethylbenz(a)anthracene	0.00004 - 0.00025
Benzo(a)pyrene	0.002
Dibenz(a,h)anthracene	0.006
Benzo(a)anthracene	2.0
Benzo(b)fluoranthene	40.0
Benzo(k)fluoranthene	72.0
Indeno(1,2,3cd)pyrene	72
Chrysene	99
Anthracene	3,300.0

CRITERIA AND STANDARDS

No federal AWQC are available for any of the PAHs of interest.

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APPENDIX B

ASSUMPTIONS USED FOR EXPOSURE CALCULATIONS

DOCUMENTATION FOR AUTOMATED RISK EVALUATION SYSTEM (ARES) VERSION 2.1

1.0 What is ARES?

ARES Version 2.1 is a process for estimating the exposure of various receptors to environmental chemicals and the risks associated with those exposures. Designed within the Paradox Version 3.5 database structure, ARES calculates daily chemical exposures for each completed pathway for each potential receptor using the exposure formulas and factors presented in the Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual, Part A (EPA, 1989) and Supplemental Guidance (EPA, 1991a). After determining daily exposures, ARES calculates the potential carcinogenic and noncarcinogenic risks associated with those exposures using risk reference doses (RfDs) and cancer potency factors (CPFs) available in EPA's Integrated Risk Information System (IRIS, 1991) and Health Effects Assessment Summary Tables (HEAST) (EPA, 1991b). Where no RfD is available, a provisional value calculated from a Maximum Contaminant Level (MCL), if available, or chronic animal data is used. The following documentation provides a list of the exposure formulas and exposure factors used to calculate the chemical intakes at Camp Lejeune, Hadnot Point and the sources used to develop the exposure factors.

2.0 What Exposure Formulas Are Used In ARES for Camp Lejeune, Hadnot Point?

2.1 Groundwater, Oral and Inhalation Exposure (Vapors in Household Air)

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

Where:

- CGW = chemical concentration in groundwater (mg/L).
- IR_{gw} = intake rate for groundwater (L/day).
- EF_{gw} = exposure frequency for contaminated groundwater (days/year).
- ED = exposure duration (years).
- BW = body weight (kg).
- AT = period of time over which exposure is averaged (days).

2.2 Soil, Oral Exposure

$$\text{Intake (mg/kg/day)} = \frac{\text{CSo} \times \text{IR}_{\text{so}} \times \text{FCs} \times \text{FIs} \times \text{BFs} \times \text{EFs} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Where:

- CSo = chemical concentration in soil (mg/kg).
- IR_{so} = intake rate for soil (mg/day).
- FCs = conversion factor for soil (kg/mg).
- FIs = fraction of soil ingested from contaminated source (unitless).
- BFs = bioavailability factor for soil (unitless).
- EFs = exposure frequency for soil (days/year).
- ED = exposure duration (years).
- BW = body weight (kg).
- AT = averaging time (days).

2.3 Soil, Dermal Exposure

$$\text{Intake (mg/kg/day)} = \frac{\text{CS}_0 \times \text{FCs} \times \text{SAs} \times \text{AF} \times \text{ABS} \times \text{EFs} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Where:

- CS = chemical concentration in soil/sediment (mg/kg).
- FCs = conversion factor for soil/sediment (kg/mg).
- SAs = skin surface area available for soil/sediment contact (cm²/event).
- AF = soil/sediment to skin adherence factor (mg/cm²).
- ABS = chemical-specific absorption factor (unitless).
- EFs = exposure frequency for soil/sediment (events/year).
- ED = exposure duration (years).
- BW = body weight (kg).
- AT = period of time over which exposure is averaged (days).

3.0 What Exposure Factors Are Used In ARES for Camp Lejeune, Hadnot Point?

3.1 ABS

volatile organic compounds	0.25	Ryan <i>et al.</i> , 1987
semivolatile organic compounds	0.10	Ryan <i>et al.</i> , 1987
metals (other than chromium VI)	0.01	Ryan <i>et al.</i> , 1987

3.2 AF

2.77 mg/cm ²	kaolin clay on hands	EPA, 1988
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3.3 AT

carcinogenic effects	70 years x 365 days/year	EPA, 1989
noncarcinogenic effects	ED (years) x 365 days/year	EPA, 1989

3.4 BFs

The bioavailability factor is the ratio of the amount of a chemical that is absorbed through the gastrointestinal lining to the amount of that chemical that is ingested.

Lead	0.10	EPA, 1984
All other chemicals	1.00	default value

3.5 BW

Adult Worker

70 kg average (male and female) of 50th percentile values for age = 18 to 75 years EPA, 1985

3.6 CGW / CSo

Due to the paucity of data, the maximum detected chemical concentration was used to represent the reasonable maximum exposure (RME) concentration. Although groundwater at this facility is treated prior to consumption, current groundwater chemical concentrations will be used in this assessment to provide a conservative risk estimate.

3.7 ED

Adult Worker

25 years national 95th percentile time at one workplace EPA, 1991a

3.8 EFgw / EFs

Adult Worker

250 days/year assumed value for number of days spent at work EPA, 1991a

3.9 FCs

1 x 10⁻⁶ kg/mg

3.10 FIs

1.00 Assumes all ingested soil is from contaminated source.

3.11 IRgw

Adult Worker

1.0 L/day assumed value for commercial/ industrial consumption EPA, 1991a

3.12 IRso

Adult Worker

50 mg/day

EPA, 1991a

3.13 SAso

All surface area values are 50th percentile values from EPA, 1985. 50th percentile values are used because surface area is related to body weight, and average body weights over the ED were used in the exposure calculations.

Adult Worker

Values based on average adult (male and female) body part surface areas (m²) multiplied by a conversion factor of 10,000 cm²/m². For conservativeness it is assumed that base personnel wear a long-sleeved shirt and long pants while working. Also, it is assumed that workers will incidentally contact of the hands and half of the head.

hands	904
½ head	<u>602</u>
	1506 cm ²

4.0 What References Were Used in Developing ARES for Camp Lejeune, Hadnot Point?

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APPENDIX C
DRINKING WATER STANDARDS

TABLE C-1

DRINKING WATER STANDARDS AND HEALTH ADVISORIES

DRINKING WATER REGULATIONS AND HEALTH ADVISORIES

by

Office of Water
U.S. Environmental Protection Agency
Washington, D.C.
202-382-7571

SAFE DRINKING WATER HOTLINE
1-800-426-4791
Monday thru Friday, 8:30 AM to 5:00 PM EST

April 1991

LEGEND

Abbreviations column descriptions are:

- MCLG - Maximum Contaminant Level Goal. A non-enforceable concentration of a drinking water contaminant that is protective of adverse human health effects and allows an adequate margin of safety.
- MCL - Maximum Contaminant Level. Maximum permissible level of a contaminant in water which is delivered to any user of a public water system.
- RfD - Reference Dose. An estimate of a daily exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime.
- DWEL - Drinking Water Equivalent Level. A lifetime exposure concentration protective of adverse, non-cancer health effects, that assumes all of the exposure to a contaminant is from a drinking water source.

(*) The codes for the Status Reg and Status HA columns are as follows:

- F - final
D - draft
L - listed for regulation
P - proposed (Phase II and V proposals)

Other codes found in the table include the following:

- NA - not applicable
PS - performance standard 0.5 NTU - 1.0 NTU
II - treatment technique

** - No more than 5% of the samples per month may be positive. For systems collecting fewer than 40 samples/month, no more than 1 sample per month may be positive.

*** - guidance

- Large discrepancies between Lifetime and Longer-term HA values may occur because of the Agency's conservative policies, especially with regard to carcinogenicity, relative source contribution, and less than lifetime exposures in chronic toxicity testing. These factors can result in a cumulative UF (uncertainty factor) of 10 to 1000 when calculating a Lifetime HA.

DRINKING WATER STANDARDS AND HEALTH ADVISORIES

April 1991

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Chemicals	Standards			Health Advisories									Cancer Group
	Status Reg.*	MCLG (mg/l)	MCL (mg/l)	Status HA*	10-kg Child			70-kg Adult					
					One-day mg/l	Ten-day mg/l	Longer-term mg/l	Longer-term mg/l	RfD mg/kg/day	DWEL mg/l	Lifetime mg/l	mg/l at 10 ⁻⁴ Cancer Risk	
ORGANICS													
1-naphthylene	-	-	-	-	-	-	-	-	0.06	-	-	-	-
Acifluorfen	-	-	-	F	2	2	0.1	0.4	0.013	0.4	-	0.1	B2
Acrylamide	F	zero	TT	F	1.5	0.03	0.02	0.07	0.0002	0.007	-	0.001	B2
Acrylonitrile	L	-	-	D	0.02	0.02	0.001	0.004	0.0001	0.004	-	0.007	B1
Adipates (diethylhexyl)	P	0.5	0.5	-	-	-	-	-	0.7	20	0.5	-	C
Alachlor	F	zero	0.002	F	0.1	0.1	-	-	0.01	0.4	-	0.04	B2
Aldicarb	P	0.001	0.003	F	-	-	-	-	0.0002	0.004	0.001	-	D
Aldicarb sulfone	P	0.002	0.003	F	-	-	-	-	0.0003	0.1	0.002	-	D
Aldicarb sulfoxide	P	0.001	0.003	F	-	-	-	-	0.0002	0.004	0.001	-	D
Aldrin	-	-	-	D	0.0003	0.0003	0.0003	0.0003	0.00003	0.001	-	0.0002	B2
Ametryn	-	-	-	F	9	9	0.9	3	0.009	0.3	0.06	-	D
Ammonium Sulfamate	-	-	-	F	20	20	20	80	0.28	8	2	-	D
Anthracene (PAH)	-	-	-	-	-	-	-	-	0.3	-	-	-	D
Atrazine	F	0.003	0.003	F	0.1	0.1	0.05	0.2	0.005	0.2	0.003	-	C
Baygon	-	-	-	F	0.04	0.04	0.04	0.1	0.004	0.1	0.003	-	C
Bentazon	-	-	-	F	0.3	0.3	0.3	0.9	0.0025	0.09	0.02	-	D
Benz(a)anthracene (PAH)	P	zero	0.0001	-	-	-	-	-	-	-	-	-	B2
Benzene	F	zero	0.005	F	0.2	0.2	-	-	-	-	-	0.1	A
Benzo(a)pyrene (PAH)	P	zero	0.0002	-	-	-	-	-	-	-	-	-	B2*
Benzo(b)fluoranthene (PAH)	P	zero	0.0002	-	-	-	-	-	-	-	-	-	B2
Benzo(g,h,i)perylene (PAH)	-	-	-	-	-	-	-	-	-	-	-	-	D
Benzo(k)fluoranthene (PAH)	P	zero	0.0002	-	-	-	-	-	-	-	-	-	B2
bis-2-Chloroisopropyl ether	-	-	-	F	4	4	4	13	0.04	1	0.3	-	D
Bromacil	-	-	-	F	5	5	3	9	0.13	5	0.09	-	C
Bromobenzene	-	-	-	D	-	-	-	-	-	-	-	-	-

* Under review.

NOTE: Anthracene and Benzo(g,h,i)perylene -- not proposed in Phase V.

EPA-816-G-92-0012-12/01/91

Chemicals	Standards			Health Advisories									Cancer Group	
	Status Reg.*	MCLG (mg/l)	MCL (mg/l)	Status HA*	10-kg Child			70-kg Adult						
					One-day mg/l	Ten-day mg/l	Longer-term mg/l	Longer-term mg/l	RfD mg/kg/day	DWEL mg/l	Lifetime mg/l	mg/l at 10 ⁻⁴ Cancer Risk		
Bromochloroacetonitrile	L	-	-	D	-	-	-	-	-	-	-	-	-	-
Bromochloromethane	-	-	-	F	50	1	1	5	0.013	0.5	0.09	-	-	-
Bromodichloromethane (THM)	-	-	0.1	D	7	7	4	13	0.02	0.6	-	0.03	-	B2
Bromoform (THM)	L	-	0.1	D	5	2	2	6	0.02	0.6	-	0.4	-	B2
Bromomethane	-	-	-	F	0.1	0.1	0.1	0.5	0.001	0.05	0.01	-	-	D
Butyl benzyl phthalate (PAE)	P	0.1	0.01	-	-	-	-	-	0.2	6	-	-	-	C
Butylate	-	-	-	F	2	2	1	4	0.05	2	0.35	-	-	D
Butylbenzene n-	-	-	-	D	-	-	-	-	-	-	-	-	-	-
Butylbenzene sec-	-	-	-	D	-	-	-	-	-	-	-	-	-	-
Butylbenzene tert-	-	-	-	D	-	-	-	-	-	-	-	-	-	-
Carbaryl	-	-	-	F	1	1	1	1	0.1	4	0.7	-	-	D
Carbofuran	F	0.04	0.04	F	0.05	0.05	0.05	0.2	0.005	0.2	0.04	-	-	E
Carbon Tetrachloride	F	zero	0.005	F	4	0.2	0.07	0.3	0.0007	0.03	-	0.03	-	B2
Carboxin	-	-	-	F	1	1	1	4	0.1	4	0.7	-	-	D
Chloral Hydrate	L	-	-	D	-	-	-	-	-	-	-	-	-	-
Chloramben	-	-	-	F	3	3	0.2	0.5	0.015	0.5	0.1	-	-	D
Chlordane	F	zero	0.002	F	0.06	0.06	-	-	0.00006	0.002	-	0.003	-	B2
Chlorodibromomethane (THM)	L	-	0.1	D	7	7	2	8	0.02	0.7	0.02	-	-	C
Chloroethane	L	-	-	D	-	-	-	-	-	-	-	-	-	-
Chloroform (THM)	L	-	0.1	D	4	4	0.1	0.5	0.01	0.5	-	0.6	-	B2
Chloromethane	L	-	-	F	9	0.4	0.4	1	0.004	0.1	0.003	-	-	C
Chlorophenol (2-)	L	-	-	D	0.05	0.05	0.05	0.2	0.005	0.2	0.04	-	-	D
p-Chlorophenyl methyl sulfide/sulfone/sulfoxide	D	-	-	-	-	-	-	-	-	-	-	-	-	-
Chloropicrin	L	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlorothalonil	-	-	-	F	0.2	0.2	0.2	0.5	0.015	0.5	-	0.15	-	B2
Chlorotoluene o-	L	-	-	F	2	2	2	7	0.02	0.7	0.1	-	-	D
Chlorotoluene p-	L	-	-	F	2	2	2	7	0.02	0.7	0.1	-	-	D
Chlorpyrifos	-	-	-	D	0.03	0.03	0.03	0.1	0.003	0.1	0.02	-	-	D
Chrysene (PAH)	P	zero	0.0002	-	-	-	-	-	-	-	-	-	-	B2
Cyanazine	L	-	-	F	0.1	0.1	0.02	0.07	0.002*	0.07*	0.01*	-	-	D*

* Under review.

NOTE: Chrysene was proposed in second option.

Chemicals	Standards			Health Advisories									Cancer Group	
	Status Reg.*	MCLG (mg/l)	MCL (mg/l)	Status HA*	10-kg Child			70-kg Adult						
					One-day mg/l	Ten-day mg/l	Longer-term mg/l	Longer-term mg/l	RfD mg/kg/day	DWEL mg/l	Lifetime mg/l	mg/l at 10 ⁻⁴ Cancer Risk		
Cyanogen Chloride	L	-	-	-	-	-	-	-	-	-	-	-	-	-
Cymene p-	-	-	-	D	-	-	-	-	-	-	-	-	-	-
2,4-D	F	0.07	0.07	F	1	0.3	0.1	0.4	0.01	0.4	0.07	-	-	D
DCPA (Dacthal)	-	-	-	F	80	80	5	20	0.5	20	4	-	-	D
Dalapon	P	0.2	0.2	F	3	3	0.3	0.9	0.026	0.9	0.2	-	-	D
Diazinon	-	-	-	F	0.02	0.02	0.005	0.02	0.00009	0.003	0.0006	-	-	E
Dibenz(a,h)anthracene (PAH)	P	zero	0.0003	-	-	-	-	-	-	-	-	-	-	B2
Dibromoacetonitrile	L	-	-	D	2	2	2	8	0.02	0.8	0.02	-	-	C
Dibromochloropropane (DBCP)	F	zero	0.0002	F	0.2	0.05	-	-	-	-	-	0.003	-	B2
Dibromomethane	L	-	-	-	-	-	-	-	-	-	-	-	-	D
Dibutyl phthalate (PAE)	-	-	-	-	-	-	-	-	0.1	4	-	-	-	D
Dicamba	L	-	-	F	0.3	0.3	0.3	1	0.03	1	0.2	-	-	D
Dichloroacetaldehyde	L	-	-	D	-	-	-	-	-	-	-	-	-	-
Dichloroacetic acid	L	-	-	D	-	-	-	-	-	-	-	-	-	-
Dichloroacetonitrile	L	-	-	D	1	1	0.8	3	0.008	0.3	0.006	-	-	C
Dichlorobenzene p-	F	0.075	0.075	F	10	10	10	40	0.1	4	0.075	-	-	C
Dichlorobenzene o-,m-	F	0.6	0.6	F	9	9	9	30	0.089	3	0.6	-	-	D
Dichlorodifluoromethane	-	-	-	F	40	40	9	30	0.2	5	1	-	-	D
Dichloroethane (1,1-)	L	-	-	D	-	-	-	-	-	-	-	-	-	-
Dichloroethane (1,2-)	F	zero	0.005	F	0.7	0.7	0.7	2.6	-	-	-	0.04	-	B2
Dichloroethylene (1,1-)	F	0.007	0.007	F	2	.1	1	4	0.009	0.4	0.007	-	-	C
Dichloroethylene (cis-1,2-)	F	0.07	0.07	F	4	3	3	11	0.01	0.4	0.07	-	-	D
Dichloroethylene (trans-1,2-)	F	0.1	0.1	F	20	2	2	6	0.02	0.6	0.1	-	-	D
Dichloromethane	P	zero	0.005	F	10	2	-	-	0.06	2	-	0.5	-	B2
Dichlorophenol (2,4-)	L	-	-	D	0.03	0.03	0.03	0.1	0.003	0.1	0.02	-	-	D
Dichloropropane (1,1-)	-	-	-	D	-	-	-	-	-	-	-	-	-	-
Dichloropropane (1,2-)	F	zero	0.005	F	-	0.09	-	-	-	-	-	0.05	-	B2
Dichloropropane (1,3-)	L	-	-	D	-	-	-	-	-	-	-	-	-	-
Dichloropropane (2,2-)	L	-	-	D	-	-	-	-	-	-	-	-	-	-
Dichloropropene (1,1-)	L	-	-	D	-	-	-	-	-	-	-	-	-	-

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Chemicals	Standards			Health Advisories									Cancer Group
	Status Reg.*	MCLG (mg/l)	MCL (mg/l)	Status HA*	10-kg Child			70-kg Adult					
					One-day mg/l	Ten-day mg/l	Longer-term mg/l	Longer-term mg/l	RfD mg/kg/day	DWEL mg/l	Lifetime mg/l	mg/l at 10 ⁻⁴ Cancer Risk	
Dichloropropene (1,3-)	L	-	-	F	0.03	0.03	0.03	0.1	0.0003	0.01	-	0.02	B2
Dieldrin	L	-	-	F	0.0005	0.0005	0.0005	0.002	0.00005	0.002	-	0.0002	B2
Diethyl phthalate (PAE)	-	-	-	D	-	-	-	-	0.8	30	5	-	D
Diethylhexyl phthalate (PAE)	P	zero	0.004	D	-	-	-	-	0.02	0.7	-	0.3	B2*
Diisopropyl methylphosphonate	-	-	-	F	8	8	8	30	0.08	3	0.6	-	D
Dimethrin	-	-	-	F	10	10	10	40	0.3	10	2	-	D
Dimethyl methylphosphonate	-	-	-	D	-	-	-	-	-	-	-	-	-
Dimethyl phthalate (PAE)	-	-	-	-	-	-	-	-	-	-	-	-	D
1,3-Dinitrobenzene	-	-	-	F	0.04	0.04	0.04	0.14	0.0001	0.005	0.001	-	D
Dinitrotoluene (2,4-)	L	-	-	D	-	-	-	-	-	-	-	-	-
2,4-/2,6-Dinitrotoluene	L	-	-	D	-	-	-	-	-	-	-	-	B2
Dinoseb	P	0.007	0.007	F	0.3	0.3	0.01	0.04	0.001	0.04	0.007	-	D
Dioxane p-	-	-	-	F	4	0.4	-	-	-	-	-	0.7	B2
Diphenamid	-	-	-	F	0.3	0.3	0.3	1	0.03	1	0.2	-	D
Diquat	P	0.02	0.02	-	-	-	-	-	0.0022	0.08	0.02	-	D
Disulfoton	-	-	-	F	0.01	0.01	0.003	0.009	0.00004	0.001	0.0003	-	E
1,4-Dithiane	-	-	-	D	-	-	-	-	-	-	-	-	-
Diuron	-	-	-	F	1	1	0.3	0.9	0.002	0.07	0.01	-	D
Endothall	P	0.1	0.1	F	0.8	0.8	0.2	0.2	0.02	0.7	0.1	-	D
Endrin	P	0.002	0.002	F	0.02	0.02	0.003	0.01	0.0003	0.01	0.002	-	D
Epichlorohydrin	F	zero	TT	F	0.1	0.1	0.07	0.07	0.002	0.07	-	0.4	B2
Ethylbenzene	F	0.7	0.7	F	30	3	1	3	0.1	3	0.7	-	D
Ethylene dibromide (EDB)	F	zero	0.00005	F	0.008	0.008	-	-	-	-	-	0.00004	B2
Ethylene glycol	-	-	-	F	20	6	6	20	2	40	7	-	D
ETU	L	-	-	F	0.3	0.3	0.1	0.4	0.00008	0.003	-	0.006**	B2
Fenamiphos	-	-	-	F	0.009	0.009	0.005	0.02	0.00025	0.009	0.002	-	D
Fluometuron	-	-	-	F	2	2	2	5	0.013	0.4	0.09	-	D
Fluorene (PAH)	-	-	-	-	-	-	-	-	0.04	-	-	-	D
Fluorotrichloromethane	-	-	-	F	7	7	3	10	0.3	10	2	-	D
Fog Oil	-	-	-	D	-	-	-	-	-	-	-	-	-

* Under review.

** Not verified yet.

1-00719-03 12-12/01/91

Chemicals	Standards			Health Advisories								Cancer Group	
	Status Reg.*	MCLG (mg/l)	MCL (mg/l)	Status HA*	10-kg Child			70-kg Adult					
					One-day mg/l	Ten-day mg/l	Longer-term mg/l	Longer-term mg/l	RfD mg/kg/day	DWEL mg/l	Lifetime mg/l		mg/l at 10 ⁻⁴ Cancer Risk
Fonofos	-	-	-	F	0.02	0.02	0.02	0.07	0.002	0.07	0.01	-	D
Formaldehyde	-	-	-	D	10	5	5	20	0.15	5	1	-	B1
Gasoline, unleaded (benzene)	-	-	-	D	-	-	-	-	-	-	0.005	-	-
Glyphosate	P	0.7	0.7	F	20	20	1	1	0.1	4	0.7	-	D
Heptachlor	F	zero	0.0004	F	0.01	0.01	0.005	0.005	0.0005	0.02	-	0.0008	B2
Heptachlor epoxide	F	zero	0.0002	F	0.01	-	0.0001	0.0001	1.3E-05	0.0004	-	0.0004	B2
Hexachlorobenzene	P	zero	0.001	F	0.05	0.05	0.05	0.2	0.0008	0.03	-	0.002	B2
Hexachlorobutadiene	-	-	-	F	0.3	0.3	0.1	0.4	0.002	0.07	0.001	-	C
Hexachlorocyclopentadiene	P	0.05	0.05	-	-	-	-	-	0.007	0.2	-	-	D
Hexachloroethane	-	-	-	F	5	5	0.1	0.5	0.001	0.04	0.001	-	C
Hexane (n-)	-	-	-	F	10	4	4	10	-	-	-	-	D
Hexazinone	-	-	-	F	3	3	3	9	0.033	1	0.2	-	D
HMX	-	-	-	F	5	5	5	20	0.05	2	0.4	-	D
Hypochlorite	L	-	-	-	-	-	-	-	-	-	-	-	-
Hypochlorous acid	L	-	-	-	-	-	-	-	-	-	-	-	-
Indeno(1,2,3-c,d)pyrene (PAH)	P	zero	0.0004	D	-	-	-	-	-	-	-	-	B2
Isophorone	L	-	-	D	15	15	15	15	0.2	7	0.1	-	C
Isopropyl methylphosphonate	-	-	-	D	-	-	-	-	-	-	-	-	D
Isopropylbenzene	-	-	-	D	-	-	-	-	-	-	-	-	-
Lindane	F	2E-4	0.0002	F	1	1	0.03	0.1	0.0003	0.01	0.0002	-	C
Malathion	-	-	-	D	0.2	0.2	0.2	0.8	0.02	0.8	0.2	-	D
Maleic hydrazide	-	-	-	F	10	10	5	20	0.5	20	4	-	D
MCPA	-	-	-	F	0.1	0.1	0.1	0.4	0.0015	0.05	0.01	-	E
Methomyl	-	-	-	F	0.3	0.3	0.3	0.3	0.025	0.9	0.2	-	D
Methoxychlor	F	0.04	0.04	F	6	2	0.5	0.2	0.005	0.2	0.04	-	D
Methyl ethyl ketone	L	-	-	F	80	8	3	9	0.00005	0.9	0.2	-	D
Methyl parathion	-	-	-	F	0.3	0.3	0.03	0.1	0.00025	0.009	0.002	-	D
Methyl tert butyl ether	L	-	-	D	3	3	0.5	2	0.005	0.2	0.04	-	D
Metolachlor	L	-	-	F	2	2	2	5	0.15	5	0.1	-	C
Metribuzin	L	-	-	F	5	5	0.3	0.9	0.025	0.9	0.2	-	D

1-09716-01 (2-12/01/91)

Chemicals	Standards			Health Advisories								Cancer Group		
	Status Reg.*	MCLG (mg/l)	MCL (mg/l)	Status HA*	10-kg Child			70-kg Adult						
					One-day mg/l	Ten-day mg/l	Longer-term mg/l	Longer-term mg/l	RIID mg/kg/day	DWEL mg/l	Lifetime mg/l		mg/l at 10 ⁻⁴ Cancer Risk	
Monochloroacetic acid	L	-	-	D	-	-	-	-	-	-	-	-	-	-
Monochlorobenzene	F	0.1	0.1	F	2	2	2	7	0.02	0.7	0.1	-	-	D
Naphthalene	-	-	-	F	0.5	0.5	0.4	1	0.004	0.1	0.02	-	-	D
Nitrocellulose (non-toxic)	-	-	-	F	-	-	-	-	-	-	-	-	-	-
Nitroguanidine	-	-	-	F	10	10	10	40	0.1	4	0.7	-	-	D
Nitrophenols p-	-	-	-	D	0.8	0.8	0.8	3	0.008	0.3	0.06	-	-	D
Oxamyl (Vydate)	P	0.2	0.2	F	0.2	0.2	0.2	0.9	0.025	0.9	0.2	-	-	E
Ozone by-products	L	-	-	-	-	-	-	-	-	-	-	-	-	-
Paraquat	-	-	-	F	0.1	0.1	0.05	0.2	0.0045	0.2	0.03	-	-	E
Pentachloroethane	-	-	-	D	-	-	-	-	-	-	-	-	-	-
Pentachlorophenol	P	zero	0.001	F	1	0.3	0.3	1	0.03	1	-	0.03	-	B2
Phenanthrene (PAH)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Phenol	-	-	-	D	6	6	6	20	0.6	20	4	-	-	D
Picloram	P	0.5	0.5	F	20	20	0.7	2	0.07	2	0.5	-	-	D
Polychlorinated byphenyls (PCBs)	F	zero	0.0005	P	-	-	-	-	-	-	-	0.0005	-	B2
Prometon	-	-	-	F	0.2	0.2	0.2	0.5	0.015	0.5	0.1	-	-	D
Pronamide	-	-	-	F	0.8	0.8	0.8	3	0.075	3	0.05	-	-	C
Propachlor	-	-	-	F	0.5	0.5	0.1	0.5	0.013	0.5	0.09	-	-	D
Propazine	-	-	-	F	1	1	0.5	2	0.02	0.7	0.01	-	-	C
Propham	-	-	-	F	5	5	5	20	0.02	0.6	0.1	-	-	D
Propylbenzene n-	-	-	-	D	-	-	-	-	-	-	-	-	-	-
Pyrene (PAH)	-	-	-	-	-	-	-	-	0.03	-	-	-	-	D
RDX	-	-	-	F	0.1	0.1	0.1	0.4	0.003	0.1	0.002	0.03	-	C
Simazine	P	0.001	0.001	F	0.5	0.5	0.05	0.2	0.002	0.06	0.001	-	-	C
Styrene	F	0.1	0.1	F	20	2	2	7	0.2	7	0.1	-	-	C
2,4,5-T	L	-	-	F	0.8	0.8	0.8	1	0.01	0.35	0.07	-	-	D
2,3,7,8-TCDD (Dioxin)	P	zero	5E-08	F	1E-06	1E-07	1E-08	4E-08	1E-06	4E-08	-	2E-08	-	B2
Tebuthiuron	-	-	-	F	3	3	0.7	2	0.07	2	0.5	-	-	D
Terbacil	-	-	-	F	0.3	0.3	0.3	0.9	0.013	0.4	0.09	-	-	E
Terbufos	-	-	-	F	0.005	0.005	0.001	0.005	0.00013	0.005	0.0009	-	-	D

* Under review.

NOTE: Phenanthrene -- not proposed.

Chemicals	Standards			Health Advisories									Cancer Group
	Status Reg.*	MCLG (mg/l)	MCL (mg/l)	Status HA*	10-kg Child			70-kg Adult					
					One-day mg/l	Ten-day mg/l	Longer-term mg/l	Longer-term mg/l	RfD mg/kg/day	DWEL mg/l	Lifetime mg/l	mg/l at 10 ⁻⁴ Cancer Risk	
Tetrachloroethane (1,1,1,2-)	L	-	-	F	2	2	0.9	3	0.03	1	0.07	0.1	C
Tetrachloroethane (1,1,2,2-)	L	-	-	D	-	-	-	-	-	-	-	-	-
Tetrachloroethylene	F	zero	0.005	F	2	2	1	5	0.01	0.5	-	0.07	B2
Toluene	F	1	1	F	20	2	2	7	0.2	7	1	-	D
Toxaphene	F	zero	0.005*	F	0.5	0.04	-	-	0.1	0.0035	-	0.003	B2
2,4,5-TP	F	0.05	0.05	F	0.2	0.2	0.07	0.3	0.0075	0.3	0.05	-	D
1,1,2-Trichloro-1,2,2-trifluoroethane	-	-	-	-	-	-	-	-	-	-	-	-	-
Trichloroacetic acid	L	-	-	D	-	-	-	-	-	-	-	-	-
Trichloroactonitrile	L	-	-	D	0.05	0.05	-	-	-	-	-	-	-
Trichlorobenzene (1,2,4-)	P	0.009	0.009	F	0.1	0.1	0.1	0.5	0.001	0.05	0.009	-	D
Trichlorobenzene (1,3,5-)	-	-	-	F	0.6	0.6	0.6	2	0.006	0.2	0.04	-	D
Trichloroethane (1,1,1-)	F	0.2	0.2	F	100	40	40	100	0.035	1	0.2	-	D
Trichloroethane (1,1,2-)	P	0.003	0.005	F	0.6	0.4	0.4	1	0.004	0.1	0.003	-	C
Trichloroethanol (2,2,2-)	L	-	-	-	-	-	-	-	-	-	-	-	-
Trichloroethylene	F	zero	0.005	F	-	-	-	-	0.007	0.3	-	0.3	B2
Trichlorophenol (2,4,6-)	L	-	-	D	-	-	-	-	-	-	-	0.3	B2
Trichloropropane (1,1,1-)	-	-	-	D	-	-	-	-	-	-	-	-	-
Trichloropropane (1,2,3-)	-	-	-	F	0.6	0.6	0.6	2	0.006	0.2	0.04	-	-
Trifluralin	L	-	-	F	0.08	0.08	0.08	0.3	0.0075	0.3	0.005	-	C
Trimethylbenzene (1,2,4-)	-	-	-	D	-	-	-	-	-	-	-	-	-
Trimethylbenzene (1,3,5-)	-	-	-	D	-	-	-	-	-	-	-	-	-
Trinitroglycerol	-	-	-	F	0.005	0.005	0.005	0.005	-	-	0.005	-	-
Trinitrotoluene	-	-	-	F	0.02	0.02	0.02	0.02	0.0005	0.02	0.002	0.1	C
Vinyl chloride	F	zero	0.002	F	3	3	0.01	0.05	-	-	-	0.0015	A
White phosphorus	-	-	-	F	-	-	-	-	0.00002	0.0005	0.0001	-	D
Xylenes	F	10	10	F	40	40	40	100	2	60	10	-	D

* Under review.

Chemicals	Standards			Health Advisories							Cancer Group	
	Status Reg.*	MCLG (mg/l)	MCL (mg/l)	Status HA*	10-kg Child			70-kg Adult				
					One-day mg/l	Ten-day mg/l	Longer-term mg/l	Longer-term mg/l	RID mg/kg/day	DWEL mg/l		Lifetime mg/l
INORGANICS												
Aluminum	L	-	-	D	-	-	-	-	-	-	-	-
Ammonia	L	-	-	D	-	-	-	-	-	-	30	-
Antimony	P	0.003	0.01/0.005	D	0.015	0.015	0.015	0.015	0.0004	0.015	0.003	-
Arsenic	*	-	0.05	D	-	-	-	-	-	-	-	0.003
Asbestos (fibers/l > 10um)	F	7 MFL	7 MFL	-	-	-	-	-	-	-	-	700 MFL
Barium	P	2	2	F	-	-	-	-	0.07	2	2	-
Beryllium	P	zero	0.001	D	30	30	4	20	0.005	0.2	-	0.0008
Boron	L	-	-	D	4	0.9	0.9	3	0.09	3	0.6	-
Cadmium	F	0.005	0.005	F	0.04	0.04	0.005	0.02	0.0005	0.02	0.005	-
Chloramine	L	-	-	D	-	-	-	-	-	-	-	-
Chlorate	L	-	-	D	-	-	-	-	-	-	-	-
Chlorine	L	-	-	D	-	-	-	-	-	-	-	-
Chlorine dioxide	L	-	-	D	-	-	-	-	-	-	-	-
Chlorite	L	-	-	D	-	-	-	-	-	-	-	-
Chromium (total)	F	0.1	0.1	F	1	1	0.2	0.8	0.005	0.2	0.1	-
Copper	P	1.3	1.3	-	-	-	-	-	-	-	-	-
Cyanide	P	0.2	0.2	F	0.2	0.2	0.2	0.8	0.022	0.8	0.2	-
Fluoride*	F	4	4	-	-	-	-	-	0.06	-	-	-
Lead (at source)	P	zero	0.005*	-	-	-	-	-	-	-	-	-
Lead (at tap)	P	zero	TT*	-	-	-	-	-	-	-	-	-
Manganese	-	-	-	D	-	-	-	-	0.14	-	-	-
Mercury	F	0.002	0.002	F	-	-	-	0.002	0.0003	0.01	0.002	-
Molybdenum	L	-	-	D	0.08	0.08	0.01	0.05	0.001	0.05	0.05	-
Nickel	P	0.1	0.1	F	1	1	0.1	0.6	0.02	0.6	0.1	-
Nitrate (as N)	F	10	10	F	-	10*	-	-	1.6	-	-	-

* Under review.

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Chemicals	Standards			Health Advisories								Cancer Group		
	Status Reg.*	MCLG (mg/l)	MCL (mg/l)	Status HA*	10-kg Child			70-kg Adult						
					One-day mg/l	Ten-day mg/l	Longer-term mg/l	Longer-term mg/l	RfD mg/kg/day	DWEL mg/l	Lifetime mg/l		mg/l at 10 ⁻⁴ Cancer Risk	
Nitrite (as N)	F	1	1	F	-	1*	-	-	0.16*	-	-	-	-	D
Nitrate + Nitrite (both as N)	F	10	10	-	-	-	-	-	-	-	-	-	-	-
Selenium	F	0.05	0.05	-	-	-	-	-	0.005	-	-	-	-	-
Silver	L	-	-	D	0.2	0.2	0.2	0.2	0.005	0.2	0.1	-	-	D
Sodium	L	-	-	D	-	-	-	-	-	20***	-	-	-	-
Strontium	L	-	-	D	25	25	25	90	2.5	90	17	-	-	D
Sulfate	P	400/500	400/500	-	-	-	-	-	-	-	-	-	-	-
Thallium	P	0.0005	0.002/ 0.001	D	0.007	0.007	0.007	0.02	0.00007	0.002	0.0004	-	-	-
Vanadium	L	-	-	D	0.08	0.08	0.03	0.11	0.003	0.11	0.02	-	-	D
Zinc	L	-	-	D	4	4	2	9	0.2	9	2	-	-	D
Zinc chloride	-	-	-	D	-	-	-	-	-	-	-	-	-	-

RADIONUCLIDES

Beta particle and photon activity (formerly man-made radionuclides)	F	zero	4 mrem	-	-	-	-	-	-	-	-	4 mrem/y	A
Gross alpha particle activity	F	zero	15 pCi/L	-	-	-	-	-	-	-	-	-	A
Radium 226/228	F	zero	5 pCi/L	-	-	-	-	-	-	-	-	22/34 pCi/l	A
Radon	T	zero	-	-	-	-	-	-	-	-	-	150 pCi/l	A
Uranium	T	zero	-	-	-	-	-	-	-	-	-	170 pCi/l	A

* Under review.

*** Guidance.

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SECONDARY MAXIMUM CONTAMINANT LEVELS

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Chemicals	Status	SMCLs (mg/l)
Aluminum	F	0.05 to 0.2
Chloride	F	250
Color	F	15 color units
Copper	F	1
Corrosivity	F	non-corrosive
Fluoride*	F	2
Foaming Agents	F	0.5
Hexachlorocyclopentadiene		0.008
Iron	F	0.3
Manganese	F	0.05
Odor	F	3 threshold odor numbers
pH	F	6.5 - 8.5
Silver	F	0.10
Sulfate	F	250
Toluene	P	-
Total Dissolved Solids (TD)	F	500
Zinc	F	5

Status Codes: P - proposed, F - final

* Under review.

10007 17799 0001 102

MICR LOGY

	<u>Status</u>	<u>MCLG</u>	<u>MCL</u>
Cryptosporidium	L	-	-
<i>Giardia lamblia</i>	F	zero	TT
<i>Legionella</i>	F ^β	zero	TT
Standard Plate Count	F ^β	NA	TT
Total Coliforms (after 12/31/90)	F	zero	**
Turbidity (after 12/31/90)	F	NA	PS
Viruses	F ^β	zero	TT

Key: PS, TT, F, defined as previously stated.

^β: Final for systems using surface water; also being considered for regulation under groundwater disinfection rule.

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Table C-2. Detection Limits in Various Media for Pesticide, Volatile, and Semivolatile Chemicals Identified on HPIA.

Chemical	CAS Number	Quantitation Limits	
		Water µg/L	Low Soil/Sediment µg/Kg
<u>Pesticides</u>			
Aroclor 1254	11097-69-1	1.0	160.0
Aroclor 1260	11096-82-5	1.0	160.0
Dieldrin	60-57-1	0.10	16.0
4,4-DDE	72-55-9	0.10	16.0
4,4-DDT	50-29-3	0.10	16.0
<u>Volatile Organics</u>			
Methylene Chloride	75-09-2	5	5
Acetone	67-64-1	10	10
<u>Semivolatile Organics</u>			
Acenaphthene	83-32-9	10	330
Anthracene	120-12-7	10	330
Benzo(a)anthracene	56-55-3	10	330
Benzo(a)pyrene	50-32-8	10	330
Benzo(b)fluroanthene	205-99-2	10	330
Benzo(k)fluroanthene	207-08-9	10	330
Benzo(g,h,i)perylene	191-24-2	10	330
Bis(2-ethylhexyl)phthalate	117-81-7	10	330
Chrysene	218-01-9	10	330
Dibenzofuran	132-64-9	10	330
Di-n-butylphthalate	84-74-2	10	330
Fluoranthene	206-44-0	10	330
Fluorene	86-73-7	10	330
Indeno(1,2,3-cd)pyrene	193-39-5	10	330
Naphthalene	91-20-3	10	330
2-Methylnaphthalene	91-57-6	10	330
Phenanthrene	85-01-8	10	330
Pyrene	129-00-0	10	330

Source: EPA 1988

Table C-3. Detection Limits in Water for Inorganic Chemicals Identified on HPIA.

Chemical	Contract Required Detection Limit ($\mu\text{g/L}$)
<u>Inorganics</u>	
Aluminum	200
Antimony	60
Arsenic	10
Barium	200
Cadmium	5
Calcium	5000
Chromium	10
Cobalt	50
Copper	25
Iron	100
Lead	3
Magnesium	5000
Manganese	15
Nickel	40
Potassium	5000
Selenium	5
Silver	10
Sodium	5000
Vanadium	50
Zinc	20

Source: EPA 1988

APPENDIX D

CALCULATIONS USED TO DETERMINE EXPOSURE BY CHEMICAL INGESTION

The following calculations show the methodology for residential exposure: the ingestion of chemicals in drinking water. All analytical data are taken from "Baseline Risk Assessment for Hadnot Point Industrial Area Operable Unit: Shallow Soils and Castle Hayne Aquifer: Draft Final"

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

Where: CW = Chemical Concentration in Water (mg/liter): Benzene = 0.002mg/L, 1,2-Dichloroethene = 0.012, 0.001 and 0.011 mg/L (= data taken from Table 3-5)
IR = Ingestion Rate (liters/day) = 2 liters/day
EF = Exposure Frequency (days/year) = 365 days/year
ED = Exposure Duration (years) = 2 years (Short-term risks) AND 30 years (Long-term risks)
BW = Body Weight (kg) = 70 kg
AT = Averaging Time (period over which exposure is averaged--days) = 70 years x 365 days

SHORT-TERM RISKS (2 year exposure)

Bldg 902: Benzene Exposure

$$I = \frac{0.002 \text{ mg/L} \times 2 \text{ L/day} \times 365 \text{ days/year} \times 2 \text{ years}}{70 \text{ kg} \times 70 \text{ years} \times 365 \text{ days}} = 1.63\text{E-}06$$

Bldg 902: 1,2-Dichloroethene

$$I = \frac{0.012 \text{ mg/L} \times 2 \text{ L/day} \times 365 \text{ days/year} \times 2 \text{ years}}{70 \text{ kg} \times 2 \text{ years} \times 365 \text{ day/year}} = 3.4\text{E-}04$$

Bldg 1202: 1,2-Dichloroethene

$$I = \frac{0.001 \text{ mg/L} \times 2 \text{ L/day} \times 365 \text{ days/year} \times 2 \text{ years}}{70 \text{ kg} \times 2 \text{ years} \times 365 \text{ days/year}} = 2.8\text{E-}05$$

Bldg 1602: 1,2-Dichloroethene

$$I = \frac{0.011 \text{ mg/L} \times 2 \text{ L/day} \times 365 \text{ days/year} \times 2 \text{ years}}{70 \text{ kg} \times 2 \text{ years} \times 365 \text{ days/year}} = 3.1\text{E-}04$$

HEALTH RISKS

Carcinogenic Risk = Intake X Cancer Slope Factor

Benzene slope factor = 0.03 (Oral, from Table 2-5)

$$\text{Non-carcinogenic Risk} = \frac{\text{Exposure Level}}{\text{RfD}} = \text{Hazard Index Value}$$

1,2-Dichloroethene RfD = 1.0E-02 (Chronic, from Table 2-5)

BENZENE CANCER RISK = 1.63E-06 X 0.03 = 4.8E-08 (Bldg 902)

1,2-DICHLOROETHENE HAZARD INDEX VALUES = 3.4E-04 + 1.0E-02 = 3.4E-02 (Bldg 902)
 = 2.8E-05 + 1.0E-02 = 2.8E-03 (Bldg 1202)
 = 3.1E-04 + 1.0E-02 = 3.1E-02 (Bldg 1602)

All Buildings: Benzo(a)pyrene: No risk to receptors since BaP was not found in the groundwater
 All Buildings: Pyrene: No risk to receptors since pyrene was not found in the groundwater

LONG-TERM RISKS (30 year exposure)

Bldg 902: Benzene Exposure

$$I = \frac{0.002 \text{ mg/L} \times 2 \text{ L/day} \times 365 \text{ days/year} \times 30 \text{ years}}{70 \text{ kg} \times 70 \text{ years} \times 365 \text{ days}} = 2.45\text{E-}05$$

Bldg 902: 1,2-Dichloroethene

$$I = \frac{0.012 \text{ mg/L} \times 2 \text{ L/day} \times 365 \text{ days/year} \times 30 \text{ years}}{70 \text{ kg} \times 30 \text{ years} \times 365 \text{ days}} = 3.4\text{E-}04$$

Bldg 1202: 1,2-Dichloroethene

$$I = \frac{0.001 \text{ mg/L} \times 2 \text{ L/day} \times 365 \text{ days} \times 30 \text{ years}}{70 \text{ kg} \times 30 \text{ years} \times 365 \text{ days/year}} = 2.8\text{E-}05$$

Bldg 1602: 1,2-Dichloroethene

$$I = \frac{0.011 \text{ mg/L} \times 2 \text{ L/day} \times 365 \text{ days/year} \times 30 \text{ years}}{70 \text{ kg} \times 30 \text{ years} \times 365 \text{ days/year}} = 3.1\text{E-}04$$

HEALTH RISKS

Carcinogenic Risk = Intake X Cancer Slope Factor

Benzene slope factor = 0.03 (Oral, from Table 2-5)

$$\text{Non-carcinogenic Risk} = \frac{\text{Exposure Level}}{\text{RfD}} = \text{Hazard Index Value}$$

1,2-Dichloroethene RfD = 1.0E-02 (Chronic, from Table 2-5)

BENZENE RISKS = 0.03 x 2.45E-05 = 7.35E-07 (Bldg 902)

1,2-DICHLOROETHENE Hazard Index Value = $3.4E-04 + 1.0E-02 = 3.4E-02$ (Bldg 902)
= $2.8E-05 + 1.0E-02 = 2.8E-03$ (Bldg 1202)
= $3.1E-04 + 1.0E-02 = 3.1E-02$ (Bldg 1602)

All Buildings: Benzo(a)pyrene: No risk to receptors since no BaP was found in the groundwater

All Buildings: Pyrene: No risk to receptors since pyrene was not found in the groundwater

APPENDIX E
SUMMARY OF HEALTH INDICES AND RISKS

CAMP LEJEUNE ARES: NONCARCINOGENIC HAZARD INDICES

12:36 Friday, July 19, 1991

Zone 0900;CURR ADWRK;CHRONIC

Analyte	Pathway					Grand Total
	DERM		ORAL			
	Medium	Total	Medium		Total	
	SOIL		GW	SOIL		
12DCE			1.2E-02		1.2E-02	1.2E-02
2MNAP			2.9E-03		2.9E-03	2.9E-03
ANAPNE	2.9E-06	2.9E-06	1.6E-04	3.4E-07	1.6E-04	1.7E-04
ANTRC	2.4E-06	2.4E-06		2.9E-07	2.9E-07	2.7E-06
BAANTR	3.8E-05	3.8E-05		4.6E-06	4.6E-06	4.3E-05
BAPYR	3.3E-05	3.3E-05		3.9E-06	3.9E-06	3.7E-05
BBFANT	3.4E-05	3.4E-05		4.1E-06	4.1E-06	3.8E-05
BGHIPY	1.5E-05	1.5E-05		1.8E-06	1.8E-06	1.7E-05
BKFANT	2.9E-05	2.9E-05		3.4E-06	3.4E-06	3.2E-05
C6H6						
CHRY	3.5E-05	3.5E-05		4.2E-06	4.2E-06	4.0E-05
FANT	7.0E-05	7.0E-05		8.4E-06	8.4E-06	7.9E-05
FLRENE	4.9E-06	4.9E-06		5.9E-07	5.9E-07	5.5E-06
ICDPYR	1.8E-05	1.8E-05		2.1E-06	2.1E-06	2.0E-05
NAP			6.6E-01		6.6E-01	6.6E-01
PB	4.6E-02	4.6E-02	2.6E-01	5.6E-03	2.7E-01	3.2E-01
PHANTR	6.8E-05	6.8E-05		8.2E-06	8.2E-06	7.6E-05
PYR	7.2E-05	7.2E-05		8.6E-06	8.6E-06	8.1E-05
Total	4.7E-02	4.7E-02	9.4E-01	5.6E-03	9.5E-01	9.9E-01

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Zone 1200;CURR ADWRK;CHRONIC

Analyte	Pathway					Grand Total
	DERM		ORAL			
	Medium	Total	Medium		Total	
	SOIL		GW	SOIL		
12DCE			9.8E-04		9.8E-04	9.8E-04
2MNAP			6.5E-04		6.5E-04	6.5E-04
ANAPNE	4.9E-06	4.9E-06	8.2E-04	5.9E-07	8.2E-04	8.2E-04
ANTRC	2.0E-07	2.0E-07		2.4E-08	2.4E-08	2.3E-07
BAANTR	1.9E-05	1.9E-05		2.3E-06	2.3E-06	2.1E-05
BAPYR	1.9E-05	1.9E-05		2.3E-06	2.3E-06	2.1E-05
BBFANT	1.9E-05	1.9E-05		2.3E-06	2.3E-06	2.1E-05
BGHIPT	9.8E-06	9.8E-06		1.2E-06	1.2E-06	1.1E-05
BKFANT	2.0E-05	2.0E-05		2.4E-06	2.4E-06	2.3E-05
CHRY	3.7E-05	3.7E-05		4.4E-06	4.4E-06	4.1E-05
FANT	3.8E-05	3.8E-05		4.5E-06	4.5E-06	4.2E-05
FLRENE	6.4E-06	6.4E-06		7.7E-07	7.7E-07	7.2E-06
ICOPYR	1.1E-05	1.1E-05		1.3E-06	1.3E-06	1.2E-05
NAP			1.4E-01		1.4E-01	1.4E-01
PB	6.9E-02	6.9E-02	1.7E-01	8.3E-03	1.8E-01	2.5E-01
PHANTR	2.9E-05	2.9E-05		3.4E-06	3.4E-06	3.2E-05
PYR	3.9E-05	3.9E-05		4.7E-06	4.7E-06	4.4E-05
Total	6.9E-02	6.9E-02	3.1E-01	8.3E-03	3.2E-01	3.9E-01

Zone 1600;CURR ADWRK;CHRONIC

Analyte	Pathway					Grand Total
	DERM		ORAL			
	Medium	Total	Medium		Total	
	SOIL		GW	SOIL		
12DCE			1.1E-02		1.1E-02	1.1E-02
2MNAP	4.1E-05	4.1E-05		4.9E-06	4.9E-06	4.6E-05
NAP	2.2E-04	2.2E-04		2.7E-05	2.7E-05	2.5E-04
PB	3.0E-02	3.0E-02	5.3E-01	3.6E-03	5.3E-01	5.6E-01
Total	3.0E-02	3.0E-02	5.4E-01	3.6E-03	5.4E-01	5.7E-01

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Zone 0900;CURR ADWRK;CHRONIC

Analyte	Dermal WOE	Inhal. WOE	Oral WOE	Pathway					Grand Total
				DERM		ORAL			
				Medium		Medium			
				SOIL	Total	GW	SOIL	Total	
BAANTR	B2	B2	B2	4.7E-06	4.7E-06		5.6E-07	5.6E-07	5.3E-06
BAPYR	B2	B2	B2	4.0E-06	4.0E-06		4.8E-07	4.8E-07	4.5E-06
BBFANT	B2	B2	B2	4.2E-06	4.2E-06		5.0E-07	5.0E-07	4.7E-06
BKFANT	B2	B2	B2	3.5E-06	3.5E-06		4.2E-07	4.2E-07	3.9E-06
C6H6	A	A	A			2.0E-07		2.0E-07	2.0E-07
CHRY	B2	B2	B2	4.4E-06	4.4E-06		5.2E-07	5.2E-07	4.9E-06
ICDPYR	B2	B2	B2	2.2E-06	2.2E-06		2.6E-07	2.6E-07	2.4E-06
Total				2.3E-05	2.3E-05	2.0E-07	2.8E-06	3.0E-06	2.6E-05

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Zone 1200;CURR ADWRK;CHRONIC

Analyte	Dermal WOE	Inhal. WOE	Oral WOE	Pathway				Grand Total
				DERM		ORAL		
				Medium SOIL	Total	Medium SOIL	Total	
BAANTR	B2	B2	B2	2.3E-06	2.3E-06	2.8E-07	2.8E-07	2.6E-06
BAPYR	B2	B2	B2	2.3E-06	2.3E-06	2.8E-07	2.8E-07	2.6E-06
BBFANT	B2	B2	B2	2.3E-06	2.3E-06	2.8E-07	2.8E-07	2.6E-06
BKFANT	B2	B2	B2	2.5E-06	2.5E-06	3.0E-07	3.0E-07	2.8E-06
CHRY	B2	B2	B2	4.5E-06	4.5E-06	5.4E-07	5.4E-07	5.1E-06
ICDPYR	B2	B2	B2	1.4E-06	1.4E-06	1.6E-07	1.6E-07	1.5E-06
Total				1.5E-05	1.5E-05	1.9E-06	1.9E-06	1.7E-05

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