

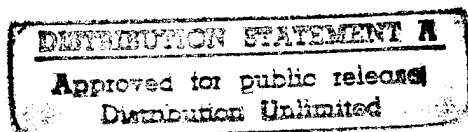
**United States Air Force
611th Air Support Group/
Civil Engineering Squadron**

Elmendorf AFB, Alaska

Final

Risk Assessment

**Point Lay Radar Installation,
Alaska**



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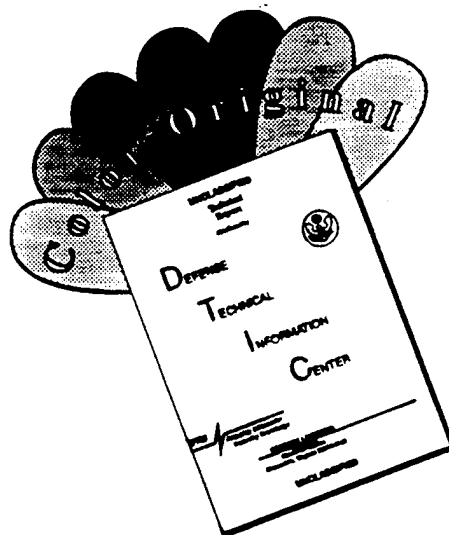
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PREFACE

This report presents the findings of Risk Assessments at sites located at the Point Lay radar installation in northern Alaska. The sites were characterized based on sampling and analyses conducted during Remedial Investigation activities performed during August and September 1993. This report was prepared by ICF Technology Incorporated.

This report was prepared between August 1995 and March 1996. Mr. Samer Karmi of the Air Force Center for Environmental Excellence was the Alaska Restoration Team Chief for this task. Dr. Jerome Madden and Mr. Richard Borsetti of the 611th CES/CEVR were the Remedial Project Managers for the project.

Approved:

Thomas McKinney
Program Director
ICF Technology Incorporated

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LIST OF ACRONYMS AND ABBREVIATIONS

ADD	Average Daily Dose
Air Force	United States Air Force
API	American Petroleum Institute
ARAR	Applicable or Relevant and Appropriate Requirement
AWQC	Ambient Water Quality Criteria
BCF	Bioconcentration Factors
CDI	Chronic Daily Intake
COC	Chemical of Concern
DEW	Distant Early Warning
DRPH	Diesel Range Petroleum Hydrocarbons
ECAO	Environmental Criterion Assessment Office of EPA
EPA	U.S. Environmental Protection Agency
ERA	Ecological Risk Assessment
GRPH	Gasoline Range Petroleum Hydrocarbons
ha	Hectare
HEAST	Health Effects Assessment Summary Tables
HI	Hazard Index
HQ	Hazard Quotient
HVOCs	Halogenated Volatile Organic Compounds
IRIS	Integrated Risk Information System
IRP	Installation Restoration Program
IS	Onsite Dietary Intake
LADD	Lifetime Average Daily Dose

LIST OF ACRONYMS AND ABBREVIATIONS (CONTINUED)

LOAEL	Lowest-Observed Adverse Effect Level
LOEL	Lowest Observed Effect Level
MCL	Maximum Contaminant Level
MDEP	Massachusetts Department of Environmental Protection
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
PAHs	Polynuclear Aromatic Hydrocarbons
PCBs	Polychlorinated Biphenyls
ppm	Parts Per Million
RBSLs	Risk-Based Screening Levels
RfD	Reference Dose
RIIs	Remedial Investigations
RI/FS	Remedial Investigation/Feasibility Study
RME	Reasonable Maximum Exposure
RRPH	Residual Range Petroleum Hydrocarbons
SF	Slope Factor
SVOCs	Semi-Volatile Organic Compounds
TPH	Total Petroleum Hydrocarbon
TRVs	Toxicity Reference Values
VOCs	Volatile Organic Compounds
UCL	Upper Confidence Limit
WOE	Weight-of-Evidence

1.0 INTRODUCTION

This document contains the baseline human health risk assessment and the ecological risk assessment (ERA) for the Point Lay Distant Early Warning (DEW) Line radar installation. Four sites at the Point Lay radar installation underwent remedial investigations (RIs) during the summer of 1993. The presence of chemical contamination in the soil, sediment, and surface water at the installation was evaluated and reported in the Point Lay Remedial Investigation/Feasibility Study (RI/FS) (U.S. Air Force 1996). The analytical data reported in the RI/FS form the basis for the human health and ecological risk assessments. The primary chemicals of concern (COCs) at the four sites are diesel and gasoline from past spills and/or leaks, chlorinated solvents, and manganese. The general location of the Point Lay radar installation is shown in Figure 1-1. The four sites investigated and the types of samples collected at each site are presented in Table 1-1.

The purpose of the risk assessment is to evaluate the human health and ecological risks that may be associated with chemicals released to the environment at the four sites investigated during the RIs. The risk assessment characterizes the probability that measured concentrations of hazardous chemical substances will cause adverse effects in humans or the environment in the absence of remediation. The risk assessment will be used to determine if remediation (site cleanup) is necessary and, if so, to rank sites for remedial action.

1.1 ORGANIZATION OF REPORT

Section 1.0 contains introductory information regarding the installation location and conditions, and a summary outline of the approach to the human health and ecological risk assessments. Section 2.0 is the Baseline Human Health Risk Assessment, and Section 3.0 is the Ecological Risk Assessment. References are presented in Section 4.0. Section 2.0, Baseline Human Health Risk Assessment, is composed of:

- **Selection of Site Contaminants.** Presents the COCs for human health and describes how they were selected for this risk assessment.
- **Exposure Assessment.** Identifies the pathways by which potential human exposures could occur, and estimates the magnitude, frequency, and duration of those exposures.
- **Toxicity Assessment.** Summarizes the toxicity of the selected COCs and the relationship between magnitude of exposure and the development of adverse health effects.
- **Risk Characterization.** Integrates the toxicity and exposure assessments to estimate the potential risks to human health from exposure to chemicals in environmental media.

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This is a detailed black and white map of Alaska, showing its coastline, major cities, and surrounding bodies of water. The map includes labels for the Arctic Ocean, Bering Sea, Gulf of Alaska, and Pacific Ocean. Key locations such as Barrow, Fairbanks, Anchorage, and Juneau are marked. The map also shows the Aleutian Islands and the Bering Strait.

2

LEGEND

▲ RADAR SITE

ALASKA REMOTE RADAR INSTALLATION

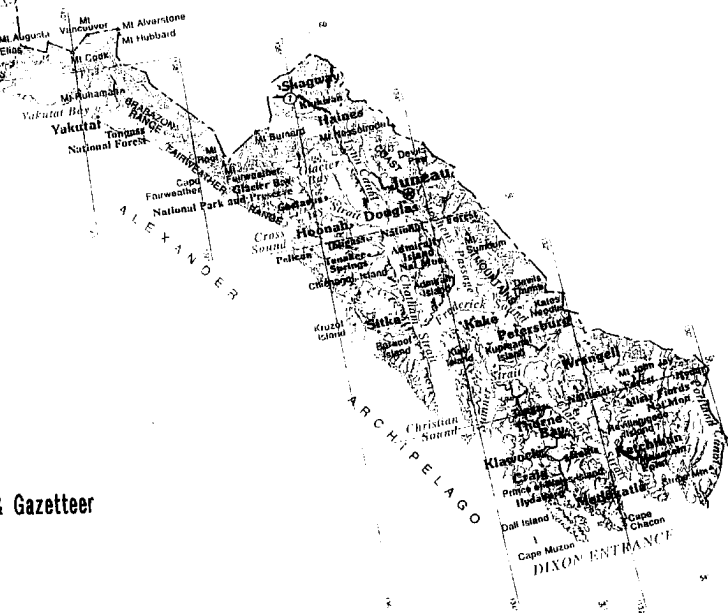
USAF 611th CES

FIGURE NO. 1-1

GENERAL
LOCATION
MAP



KILOMETERS 50 0 50 100 150 200 250 300
MILES 50 0 50 100 150 200



Source: Alaska Atlas & Gazetteer

TABLE 1-1. SITES EVALUATED AT POINT LAY DEW LINE INSTALLATION

SITE NAME	SITE ID NUMBER	SOIL	SEDIMENTS	SURFACE WATER
Deactivated Landfill	LF01	NA	X	X
Garage	SS06	X	X	X
Drainage Pathway from POL Tanks	SS07	NA	X	X
Crushed Drum Area	SS08	X	X	X

X Chemical analyses were performed on these media.
 NA No chemical analysis was performed.

- **Risk Characterization Uncertainty.** Describes the potential shortcomings in the data and the methods used to develop the risk assessment, and the uncertainties in the interpretation of the data and the risk characterization results.
- **Risk Assessment Summary and Conclusions.** Presents a summary of, and conclusions regarding, the potential human health risks associated with exposure to contaminated media at the four sites at the Point Lay DEW Line installation.

Section 3.0, the ERA, is composed of:

- **Selection of Site Contaminants.** Presents the COCs for ecological receptors and describes how they were selected for the ERA.
- **Ecological Exposure Assessment.** Identifies the potential receptors and representative species, habitat suitability, and exposure pathways.
- **Ecological Toxicity Assessment.** Describes the potential effects of site contaminants on the representative species.
- **Risk Characterization for Ecological Receptors.** Evaluates the likelihood of adverse effects on ecological receptors.
- **Ecological Uncertainty Analysis.** Describes the potential shortcomings in the data and methods used to develop the ERA, and the uncertainties in the interpretation of the data and the ecological risk characterization results.

- **Summary of Ecological Risk.** Presents a summary of ecological risks associated with contaminated media at the four sites at the installation.

Appendix A contains the human health risk assessment spreadsheets used to estimate chemical intake, noncancer hazard, and excess lifetime cancer risk. Appendix B consists of toxicology profiles. The exposure equations and calculations for ecological receptors are presented in Appendices C through F. Appendix G contains a summary of RI sampling and analyses and the RI analytical data for all sites from which the COCs were selected and upon which the human health and ecological risk assessments are based.

1.2 RISK ASSESSMENT GUIDANCE DOCUMENTS

The following guidance documents were used to develop the human health and ecological risk assessments:

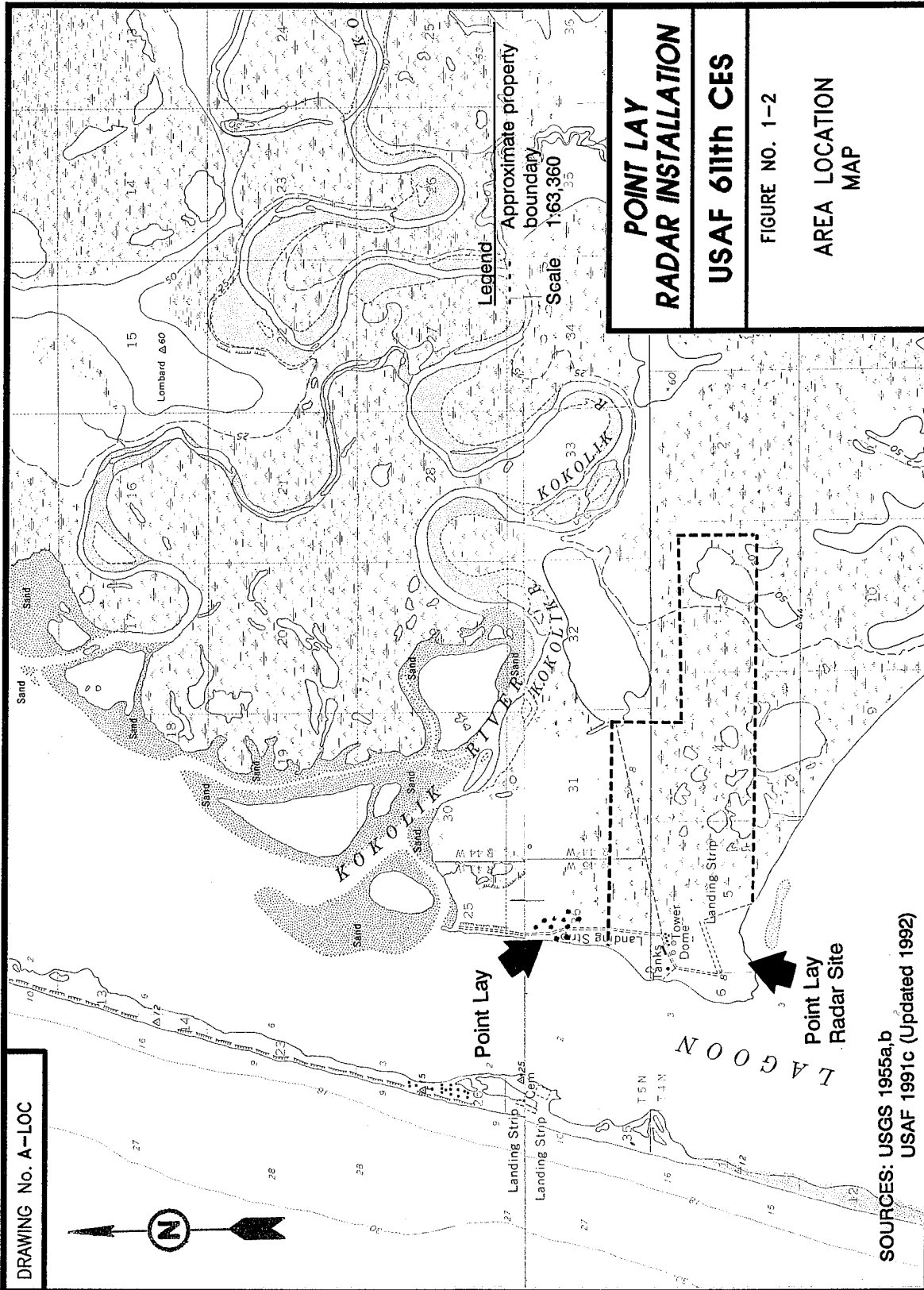
- *Risk Assessment Guidance for Superfund: Volume 1, Human Health Evaluation Manual (Part A)* (EPA 1989a);
- Region 10 Supplemental Risk Assessment Guidance for Superfund (EPA 1991a);
- *Risk Assessment Guidance for Superfund: Volume 2, Environmental Evaluation Manual* (EPA 1989b);
- General Guidance for Ecological Risk Assessment at Air Force Bases (MITRE 1990);
- Handbook to Support the Installation Restoration Program (IRP) Statements of Work (U.S. Air Force 1991); and
- Framework for Ecological Risk Assessment (EPA 1992a).

1.3 INSTALLATION DESCRIPTION AND ENVIRONMENTAL SETTING

The Point Lay DEW Line installation is located at 69°43'N, 163°00'W in the western portion of the Arctic Coastal Plain, on the west shoreline of Kasegaluk Lagoon. The installation was constructed in 1955, occupies 1,442 acres, and consists of a 24-module train, rotating radar, four communications dishes, garage, warehouse, hangar, and 3,519 foot lighted gravel runway. An area location map is presented in Figure 1-2, and a site plan of the installation is shown in Figure 1-3.

Geology and Geography. Geologically the area consists of surficial deposits of silty loam and an organic layer called a tundra mat that provides an insulating barrier between the air and the underlying perennially frozen ground. The area is underlain with shallow medium to fine-grained sands and silts. Permafrost underlies the entire installation.

DRAWING No. A-LOC



**POINT LAY
RADAR INSTALLATION**

USAF 611th CES

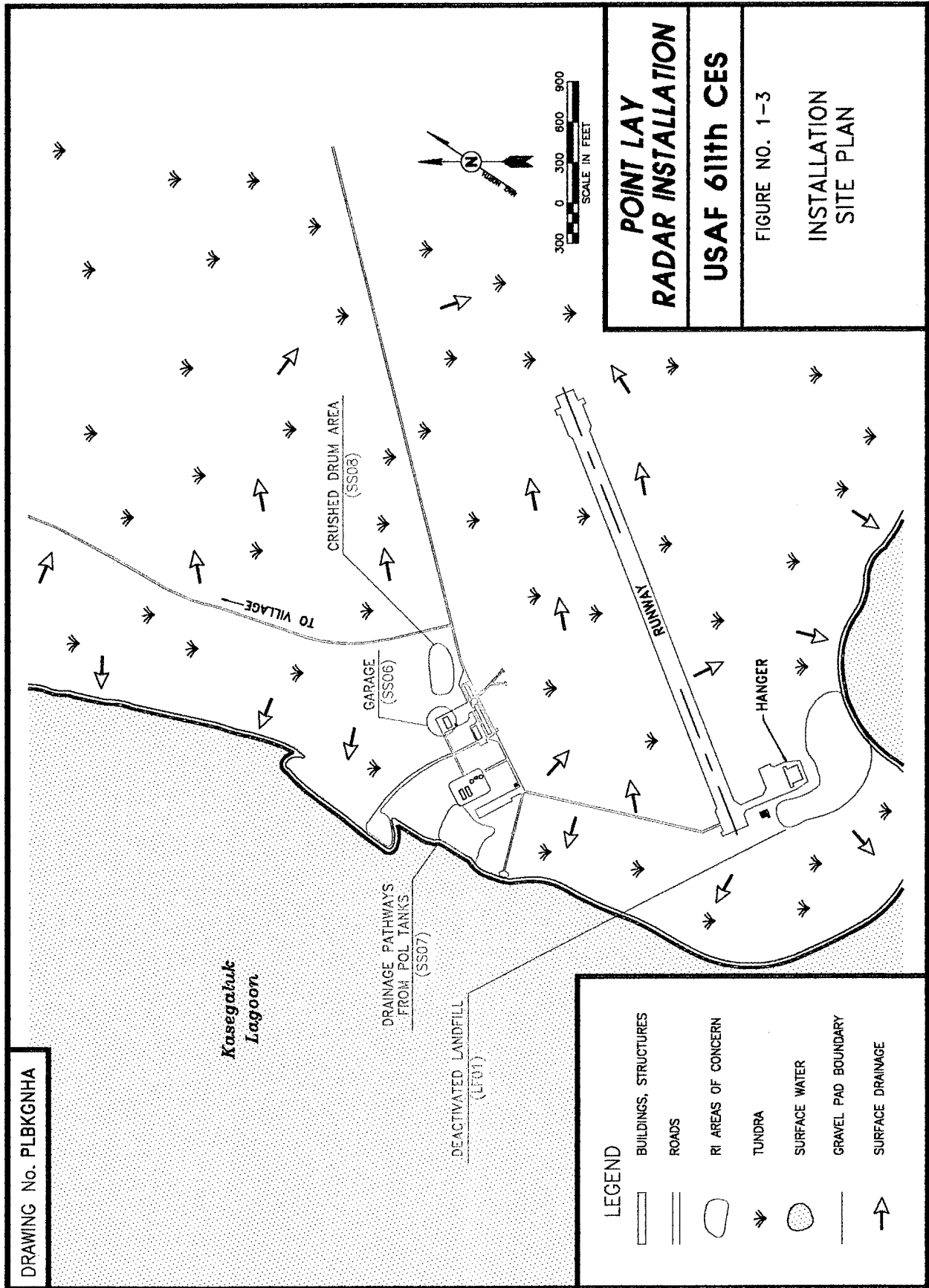
FIGURE NO. 1-2

AREA LOCATION
MAP

SOURCES: USGS 1955a,b
USAF 1991c (Updated 1992)

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Permafrost comprises geologic, hydrologic, and meteorologic characteristics that result in permanently frozen ground. Permafrost occurs in both unconsolidated sediments and bedrock. Its distribution is continuous on the Arctic Coastal Plain, and it has a significant impact on the flow of ground and surface water.

Permafrost acts as an impermeable barrier to the movement of groundwater because pore spaces are ice-filled in the zone of saturation. Recharge and discharge are, limited to unfrozen channels penetrating the permafrost zone. Permafrost restricts the downward percolation of water and increases runoff, enhancing the creation of lakes and swamps, and also restricts an aquifer's storage capacity and the number of locations from which groundwater may be withdrawn.

The interval between permafrost and ground surface is called the active zone because it freezes and thaws with seasonal weather changes. Water may be found in the active zone in the summer months. The thickness of the active zone at Point Lay varies from approximately one to six feet.

The local geography includes numerous small lakes and ponds. At the installation, the tundra mat and silts have been removed or destroyed, and gravel has been placed over the older sediments to provide a foundation for the facility structures.

Meteorology. Temperature and precipitation measurements have been recorded during the operation of the installation. The nearest National Weather Service monitoring station is located in Barrow, Alaska, approximately 185 miles northeast of Point Lay.

Temperatures at Point Lay generally are low throughout the year, with average summer temperatures ranging from 32°F to 53°F and average winter temperatures from -27°F to -5°F. The highest temperature recorded was 78°F in July 1955, and the lowest was -55°F in March 1956 (Hart Crowser 1987).

Annual precipitation at the installation averages 7 inches, including 21 inches of snow. Most precipitation occurs in July and August; precipitation between October and May is mostly snow.

Prevailing winds are northeasterly year-round with an average speed of 12 mph. There is little variation in wind speed throughout the year, although winds can reach 35 mph for extended periods and gusts will attain much greater speeds.

Demographics. The village of Point Lay is located about three quarters of a mile north of the installation and is connected by a gravel road. The village is one of the more recently established Inupiat villages along the Arctic coast. The population of Point Lay was 192 in 1993, 90 percent of whom were Inupiat. Of the employed individuals, 97 percent work for the federal, state, or North Slope Borough governments. Approximately 60 percent of the households in Point Lay derive half or more of their meals from subsistence activities based on an average of 21 meals per household per week (Harcharek 1994). The Point Lay installation is staffed by approximately four personnel who maintain the station facilities.

1.3.1 Sites Evaluated at the Point Lay DEW Line Installation

The Point Lay radar installation was investigated to evaluate possible contamination related to United States Air Force (Air Force) activities and historical waste disposal practices at the sites. Four sites at the Point Lay radar installation were determined to be of potential concern based on previous IRP sampling activities, literature search, pre-survey and reconnaissance trips, interviews with station personnel, and information on disposal practices at DEW Line stations. The sites were investigated during RI/FS activities to confirm the presence or absence of chemical contamination; define the extent and magnitude of confirmed chemical releases; gather adequate data to determine the magnitude of potential risks to human health and the environment; and gather adequate data to identify and select the appropriate remedial actions for sites where apparent risks exceed acceptable limits.

Four sites at the installation that were investigated during the RI were evaluated in the risk assessment. These sites are:

- **Deactivated Landfill (LF01)** - located southeast of the hangar, was used by the installation from approximately 1973 until closure in 1987. At the time of closure, the area was cleared of major surface debris, graded, covered with gravel, and seeded with grass. A small, seasonal stream appears to be eroding the landfill cover exposing rusty drums and landfill debris. Standing water along the landfill's western edge and surface runoff from the gravel pad area south of the hangar are possible source areas for the small stream. The water downstream from the landfill was noted as having a minor discoloration and biogenic sheen.
- **Garage (SS06)** - located approximately 250 feet north of the module train. It is an approximately 90 by 45 foot building that is elevated approximately five feet above the tundra and is surrounded by gravel pad on the north, east, and south sides. The building is used for vehicle maintenance and storage. Floor drains in this building discharged to the tundra beneath the structure and may have received vehicle maintenance wastes. The floor drains were sealed in 1993 to prevent future releases. Stained soils are visible below the floor drains under the Garage.
- **Drainage Pathway from POL Tanks (SS07)** - located along the bluff between the fuel storage area and the lagoon. The site consists of three small streams and a beach bluff located at the edge of the gravel pad. The site was historically a drum storage area. An ADEC representative inspecting this site during the reconnaissance visit felt this area appeared potentially contaminated. Styrofoam and fabric debris are partially buried in some areas along the bluff at this site.
- **Crushed Drum Area (SS08)** - located approximately 150 feet northeast of the module train. The gravel pad slopes gently to the tundra north and east of the site, and is adjacent to a drainage pathway that runs from below the east end of the module train to the south end of the gravel pad area at the site. It was

determined that an investigation was warranted at this site because of previous drum crushing activities associated with this site.

Table 1-1 contains a summary of the environmental media sampled at each of the sites. The analytical data obtained from these samples form the basis of the human health and ecological risk assessments in this document. Figures of each of the sites are presented in Section 2.0.

1.4 APPROACH TO HUMAN HEALTH RISK ASSESSMENT

The Point Lay DEW Line installation presents a unique challenge in the development of a human health risk assessment. Many of the conventional assumptions applied in risk assessments do not apply to the North Slope of Alaska. Point Lay is remote and sparsely populated. Native residents, largely Inupiat, follow a lifestyle that includes a significant subsistence component; much of their food consists of mammals (whales and caribou), aquatic life (arctic char), and birds (ptarmigan and ducks) abundant in this area of the arctic (Harcharek 1994). The climate is generally harsh, and the soil and surface water are frozen for approximately nine months of the year.

The general approach to the human health risk assessment is to quantify the excess lifetime cancer risk or the noncancer hazard for the site contaminants detected at each of the four sites at the installation. The maximum concentration of each chemical detected is used instead of an arithmetic mean or 95th percentile upper confidence limit (UCL) because contamination was detected infrequently and generally found to be of low concentration. Incorporating nondetects into the calculation of an average or UCL when the frequency of positive detects is low tends to yield low and unreliable estimates of contamination. Use of the maximum concentration yields a conservative estimate of risk or hazard.

To the extent possible, site-specific information is incorporated into the development of the exposure assumptions. The harsh climate naturally serves to limit exposure to contaminated soil, sediment, and surface water.

Residential exposure assumptions were used to reflect the upper-bound potential future risk. Several North Slope communities have requested use of inactive buildings at DEW Line installations; therefore, an evaluation using potential residential scenarios at the installations and sites was conducted.

Excess lifetime cancer risk and noncancer hazard are calculated for the soil/sediment ingestion and surface water ingestion pathways. Other pathways were eliminated from consideration as described in Section 2.2, Human Health Risk Exposure Assessment.

1.5 APPROACH TO ECOLOGICAL RISK ASSESSMENT

The objective of the ERA is to estimate potential impacts to aquatic and terrestrial plants and animals at the Point Lay radar installation. MITRE (1990) suggests that ERAs should "estimate

the potential for occurrence of adverse effects that are manifested as changes in the diversity, health and behavior" of ecosystems. MITRE proposes that this can be accomplished by:

- Estimating the health risk to individual species;
- Evaluating the health of the community of exposed species; and
- Determining the potential adverse effects of contamination over several life cycles of the species under study.

Because this is a screening level assessment, the scope of the ERA is limited to the first task: estimating the health risk to individual species. If a potential health risk to individual species was identified, further work may be recommended to evaluate the community and life cycle effects. It is important to note that the health risk to an individual species is different from the health risk to an individual within a species. The former refers to population level biology, where the individual is not considered a relevant endpoint. The latter assesses the risks to an individual. In this ecological assessment, the individual is considered only in the case of threatened or endangered species.

2.0 BASELINE HUMAN HEALTH RISK ASSESSMENT

The purpose of the baseline human health risk assessment for the Point Lay DEW Line installation is to provide a basis for developing a risk management plan, including remedial action alternatives based on data from the RI/FS. The risk assessment develops numerical estimates of cancer risk and noncancer hazard for each site where sufficient information is available. Where information is not adequate to quantify noncancer hazard or cancer risk for a given COC, a qualitative discussion of the toxicity of that COC is provided in the Toxicity Profiles (Appendix B).

This baseline human health risk assessment addresses issues unique to this location as described in the introduction. It is comprised of six sections:

- Identification of COCs - in which the chemicals detected in environmental samples are compared to risk-based screening levels (RBSLs) and concentrations considered to be applicable or relevant and appropriate requirements (ARAR);
- Exposure assessment - in which the frequency, duration, and magnitude of potential exposures to the COCs are estimated;
- Toxicity assessment - in which the toxicology of the COCs is assessed;
- Risk characterization - in which the potential for adverse health effects in humans as a result of exposure to the COCs is quantified (as appropriate) or qualitatively discussed;
- Uncertainty assessment - in which the general sources of uncertainty in the risk assessment process and the site-specific sources of uncertainty are discussed; and
- Risk Assessment Summary and Conclusions - in which the human health risks are summarized for each of the sites and conclusions based on these risks are presented.

2.1 IDENTIFICATION OF CHEMICALS OF CONCERN

Chemicals of potential concern to human health were selected for each site at the Point Lay facility based on comparison of chemical concentrations to RBSLs, naturally-occurring background concentrations, ARARs, and safe levels of essential human nutrients (e.g., calcium, magnesium, sodium, and potassium).

This section discusses the RI sampling strategy and an evaluation of data prior to screening (Section 2.1.1), describes and presents equations for calculating RBSLs (Section 2.1.2), identifies

chemicals that are essential human nutrients (Section 2.1.3), describes the collection of background samples (Section 2.1.4), and discusses the selection of COCs (Section 2.1.5).

2.1.1 Sampling Strategy and Evaluation of Analytical Data

The RI sampling strategy at the Point Lay sites was to characterize the nature and extent of potential contamination at each site. Suspected source areas were sampled to determine the concentrations of contaminants, if any, at the areas likely to have the highest concentrations. Migration pathways from the source areas were sampled to determine the extent, if any, that the contaminants had migrated from the sites. If no discernable pathways were evident, an attempt was made to sample around the source areas to determine the extent of site contaminants. Quick turn-around analyses were conducted on samples from the first sampling event, and a second round of sampling was conducted at those sites where further characterization of the nature and extent of contamination was needed.

Sample types included surface and subsurface soil/sediment samples and surface water samples. In almost all cases, samples were discrete grab samples from one sample location. Surface soil and sediment samples were collected in gravel and tundra areas at or near the ground surface (from ground surface to approximately six inches in depth). Subsurface soil samples were mainly collected in gravel pad areas where unsaturated conditions allowed vertical migration of contaminants. Sediment samples were collected below shallow ponds or streams, or in areas that visually appeared to have been previously covered with water. Surface water samples were collected from ponds, streams, springs, or leachate areas. Surface water samples underwent both total and dissolved metal analyses; however, the total metal analytical results were used in the risk assessment. A summary of the 1993 RI sampling and analyses conducted at the installation is presented in Appendix G.

Before screening for COCs, the results of the RI sampling program were sorted by medium (i.e., soil, sediment, and surface water) and reviewed for quality. The review included an evaluation of the analytical methods used, sample quantitation limits, and qualified data, and a comparison to background levels and laboratory and field blanks. Analytical data were reviewed for completeness, comparability, representativeness, precision, and accuracy. In addition, data validation qualifiers were considered in assessing the quality of the data. The review and validation of analytical data determined that a minimal amount of data was not usable. These data were qualified with an "R" and were not used in the risk assessment.

As outlined in the Risk Assessment Guidance for Superfund (EPA 1989a), site data were compared to available blank (laboratory, field, and trip) data. The data from blanks are presented in Appendix G. In accordance with EPA (1989a), if the detected concentration in a sample was less than 10 times the concentration from blanks for common laboratory contaminants (e.g., acetone, 2-butanone, methylene chloride, toluene, and the phthalate esters) the chemical was not selected for evaluation in the risk assessment. For those organic or inorganic chemicals that are not considered by the U.S. Environmental Protection Agency (EPA) to be common laboratory contaminants (all other compounds), if the detected concentration was less than five times the maximum concentration detected in the blanks, the chemical was not selected for evaluation in the risk assessment.

2.1.2 Risk-Based Screening Levels

An RBSL is a chemical concentration in a particular medium that yields a given cancer risk or hazard quotient (HQ) (e.g., 10^{-7} cancer risk; 0.1 HQ) under a given set of conditions. For Point Lay, the RBSLs were calculated for soil based on EPA default reasonable maximum exposure (RME) parameters (EPA 1991a). In developing the RBSLs, the most recent toxicity factors available from the Integrated Risk Information System (IRIS) and the Health Effects Assessment Summary Tables (HEAST) were used. IRIS and HEAST are databases of toxicity information for human health risk assessment maintained by the Environmental Criteria and Assessment Office (ECAO) of the EPA. The information presented on IRIS represents the consensus of EPA scientists regarding the toxicity of chemicals released to the environment. Toxicity factors that EPA has withdrawn from IRIS and HEAST or available from other sources, were not used in this risk assessment.

2.1.2.1 Formulae for Calculating RBSLs. The RBSL concentrations were derived using equations in EPA Region 10 guidance (EPA 1991a). The equations are also presented in a slightly different form in the Risk Assessment Guidance for Superfund Volume I, Part B (EPA 1991b). Exposure assessment and risk characterization algorithms for human health risk assessments use site-specific contaminant concentration data, factors describing exposure, and toxicity dose-response values [e.g., reference doses (RfDs) or carcinogen slope factors (SFs)]. These risk assessment algorithms were solved for the concentration term to derive the RBSL for soil and surface water. The algorithms are summarized as follows:

$$\text{Risk} = C \times \left(\frac{\text{CR} \times \text{EFD}}{\text{BW} \times \text{AT}} \right) \times \text{SF} \quad \text{or} \quad \text{HQ} = C \times \left(\frac{\text{CR} \times \text{EFD}}{\text{BW} \times \text{AT}} \right) / \text{RfD} \quad \text{EQUATION 1, 2}$$

Risk = Target Cancer Risk

C = Concentration

CR = Contact Rate

EFD = Exposure Frequency and Duration

BW = Body Weight

AT = Averaging Time

SF = Slope Factor

HQ = Target Hazard Quotient

RfD = Reference Dose

RBSLs are calculated based on a specific target cancer risk or HQ. EPA (1991a) recommends that a 1×10^{-7} target cancer risk and a target noncancer HQ of 0.1 be used for soil and a 1×10^{-6} risk and 0.1 HQ be used for surface water. The lower target cancer risk is used for screening soil because additional pathways, such as dermal contact and inhalation, are not accounted for by the calculations (EPA 1991a).

Equations (1) and (2) shown above are rearranged to solve for the concentration term (i.e., the RBSL):

$$C = \text{Risk} / \left(\left(\frac{\text{CR} \times \text{EFD}}{\text{BW} \times \text{AT}} \right) \times \text{SF} \right) \quad \text{or} \quad C = \text{HQ} / \left(\left(\frac{\text{CR} \times \text{EFD}}{\text{BW} \times \text{AT}} \right) / \text{RfD} \right) \quad \text{EQUATION 3, 4}$$

Surface Water Ingestion Equations. Using standard default exposure factors (EPA 1989b) for water ingestion, the equation for cancer risk from drinking water ingestion becomes:

$$\text{Risk} = C (\mu\text{g/L}) \times 0.001 \text{ mg}/\mu\text{g} \times \left(\frac{2 \text{ L/day} \times 350 \text{ day/year} \times 30 \text{ year}}{70 \text{ kg} \times 70 \text{ year} \times 365 \text{ day/year}} \right) \times \text{SF}_o \quad \text{EQUATION 5}$$

Equation 5 can be rearranged to solve for an RBSL with, for example, a target cancer risk of 10^{-6} :

$$C (\mu\text{g/L}) = 10^{-6} \times 1,000 \mu\text{g}/\text{mg} / \left[\left(\frac{2 \text{ L/day} \times 350 \text{ day/year} \times 30 \text{ year}}{70 \text{ kg} \times 70 \text{ year} \times 365 \text{ day/year}} \right) \times \text{SF}_o \right] \quad \text{EQUATION 6}$$

For non-carcinogens, the equation for the HQ for drinking water ingestion is:

$$\text{HQ} = C (\mu\text{g/L}) \times 0.001 \text{ mg}/\mu\text{g} \times \left(\frac{2 \text{ L/day} \times 350 \text{ day/year} \times 30 \text{ year}}{70 \text{ kg} \times 30 \text{ year} \times 365 \text{ day/year}} \right) / \text{RfD}_o \quad \text{EQUATION 7}$$

Equation 7 can be rearranged to provide an equation for the concentration that represents an HQ of one from ingestion:

$$C (\mu\text{g/L}) = 1 \times 1,000 \mu\text{g}/\text{mg} / \left[\left(\frac{2 \text{ L/day} \times 350 \text{ day/year} \times 30 \text{ year}}{70 \text{ kg} \times 30 \text{ year} \times 365 \text{ day/year}} \right) / \text{RfD}_o \right] \quad \text{EQUATION 8}$$

Soil or Sediment Ingestion Equations. The equation for calculating carcinogenic risk from soil or sediment ingestion, combining child (subscript c) and adult (subscript a) exposure, is as follows:

$$\text{Risk} = C (\text{mg/kg}) \times 0.000001 \text{ kg}/\text{mg} \times \quad \text{EQUATION 9}$$

$$\left[\left(\frac{200_c \text{ mg/day} \times 350_c \text{ day/year} \times 6 \text{ year}}{15_c \text{ kg} \times 365 \text{ day/year}} \right) + \left(\frac{100_a \text{ mg/day} \times 350_a \text{ day/year} \times 24 \text{ year}}{70_a \text{ kg} \times 365 \text{ day/year}} \right) \right] / 70 \text{ year} \times \text{SF}_o$$

Equation 9 can be rearranged to solve for the concentration that represents a target cancer risk of 10^{-7} :

$$C (\text{mg/kg}) = 10^{-7} \times 1,000,000 \text{ mg}/\text{kg} / \quad \text{EQUATION 10}$$

$$\left[\left(\frac{200_c \text{ mg/day} \times 350_c \text{ day/year} \times 6 \text{ year}}{15_c \text{ kg} \times 365 \text{ day/year}} \right) + \left(\frac{100_a \text{ mg/day} \times 350_a \text{ day/year} \times 24 \text{ year}}{70_a \text{ kg} \times 365 \text{ day/year}} \right) \right] / 70 \text{ year} \times \text{SF}_o$$

For non-carcinogens in soil, Equation 11 is used to calculate the HQ:

$$HQ = C \text{ (mg/kg)} \times 0.000001 \text{ kg/mg} \times$$

EQUATION 11

$$\left[\left(\frac{200_c \text{ mg/day} \times 350_c \text{ day/year} \times 6 \text{ year}}{15_c \text{ kg} \times 365 \text{ day/year}} \right) + \left(\frac{100_a \text{ mg/day} \times 350_a \text{ day/year} \times 24 \text{ year}}{70_a \text{ kg} \times 365 \text{ day/year}} \right) / 30 \text{ year} \right] / RfD_o$$

Equation 11 can be rearranged to solve for the concentration that represents an HQ of 0.1:

$$C \text{ (mg/kg)} = 0.1 \times 1,000,000 \text{ mg/kg} /$$

EQUATION 12

$$\left[\left(\left(\left(\frac{200_c \text{ mg/day} \times 350_c \text{ day/year} \times 6 \text{ year}}{15_c \text{ kg} \times 365 \text{ day/year}} \right) + \left(\frac{100_a \text{ mg/day} \times 350_a \text{ day/year} \times 24 \text{ year}}{70_a \text{ kg} \times 365 \text{ day/year}} \right) \right) / 30 \text{ year} \right) / RfD_o \right]$$

2.1.3 Screening of Chemicals by Comparing Maximum Detected Concentrations of Essential Human Nutrients

Based on EPA's guidance (1991a), calcium, magnesium, potassium, iron, and sodium are considered to be essential human nutrients and were eliminated from the human health risk assessment at the screening stage. These chemicals are often detected but are not toxic in humans except at extremely high doses. No quantitative toxicity information is available for these elements from EPA sources; therefore, these metals are not selected as COCs for this risk assessment.

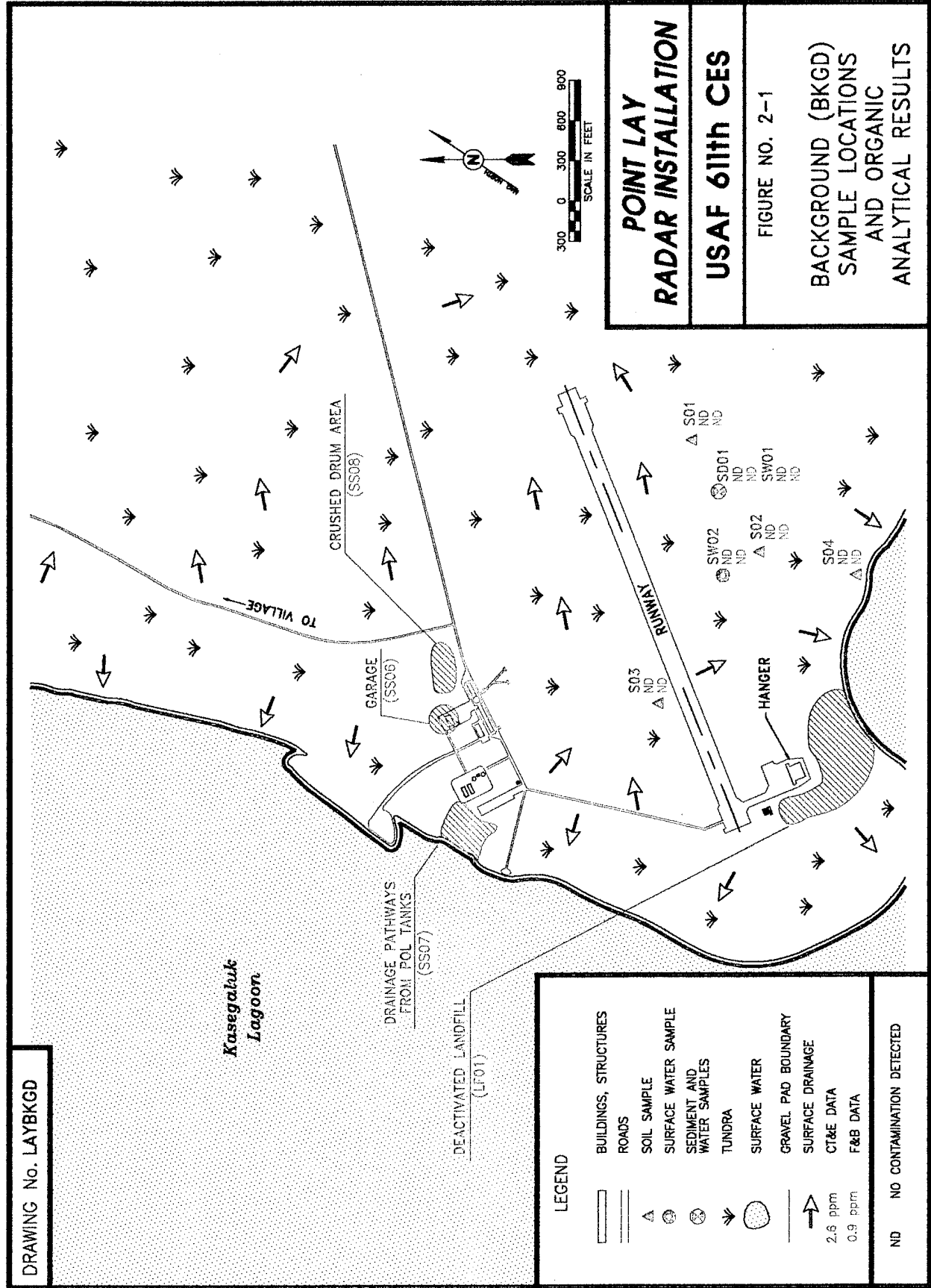
2.1.4 Concentrations of Organic and Inorganic Constituents in Background Samples

Four soil, one sediment, and two surface water samples were collected in an area of the radar installation assumed to be unaffected by installation operations. These samples served to determine the background concentrations of naturally occurring organic constituents in soil, sediment, and surface water (Figure 2-1). Soil and sediment background samples were collected from a depth of zero to six inches.

In order to obtain a representative range of background inorganic (metal) concentrations in soil, sediments, and surface waters of the North Slope, 44 samples (29 soil or sediment, and 15 water) from seven North Slope radar installations were collected. In addition to Point Lay, the installations included Barter Island, Bullen Point, Oliktok Point, Point Lonely, Point Barrow, and Wainwright. Approximately four soil or sediment and two surface water background samples were collected and analyzed for metals at each of the seven radar installations. In determining potential risks in surface water, total metal concentrations were used in the risk assessment. Analytical results for background samples collected at Point Lay are presented in Appendix G.

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2.1.5 Selection of Chemicals of Concern

Soil and Sediment. The maximum concentrations of the chemicals detected in soil or sediment samples at the Point Lay installation and not considered to be essential human nutrients were compared, on a site-by-site basis, to the corresponding background concentrations, RBSLs, and where available, federal or state ARARs. Chemicals detected without an RBSL or ARAR were retained as COCs if concentrations exceeded background levels. A chemical with an RBSL or ARAR was selected as a COC for soil and sediment if the maximum concentration at which the chemical was detected exceeded the corresponding background concentration and the RBSL (based either on cancer risk or noncancer hazard) or ARAR (Table 2-1). Thus, for example, the maximum concentration of diesel range petroleum hydrocarbons (DRPH) at the Deactivated Landfill (LF01), 624 mg/kg, exceeds the background concentration range and the state ARAR of 500 mg/kg. Therefore, DRPH were selected as a COC for the soils at the Deactivated Landfill site.

The COCs for soil/sediment at each site were compared to background concentrations, RBSLs, and ARARs in Table 2-1. The chemicals retained as COCs exceed background concentrations, and the RBSL or an ARAR. A chemical was not retained if the level detected was less than the corresponding RBSL and ARAR, even though background levels were exceeded. The COCs selected that exceed background levels, but do not have an RBSL or an ARAR are discussed below. The COCs selected at each site that exceed an RBSL, ARAR, or both, are discussed in Sections 2.1.5.1 to 2.1.5.5.

Surface Water. The maximum concentrations of the chemicals detected in surface water samples at Point Lay were compared, on a site-by-site basis, to the corresponding background concentrations, RBSLs, and where available, federal or state ARARs. Chemicals detected without an RBSL or ARAR were retained as COCs if concentrations exceeded background levels. A chemical with an RBSL or ARAR was selected as a COC for surface water if the maximum concentration at which the chemical was detected exceeded the corresponding background concentration, and the RBSL (based either on cancer risk or noncancer hazard) or ARAR (Table 2-1). Thus, for example, the maximum concentration of gasoline range petroleum hydrocarbons (GRPH) at the Deactivated Landfill (LF01), 223 µg/L, exceeds the background concentration of <50 µg/L (not detected) and the RBSL based on cancer risk of 50 µg/L. Therefore, GRPH were selected as a COC for the surface water at the Deactivated Landfill (LF01) site.

The COCs for surface water at each site were compared to background concentrations, RBSLs, and ARARs in Table 2-1. The chemicals retained as COCs exceed background concentrations and the RBSL or an ARAR. A chemical was not retained if the level detected was less than the corresponding RBSL and ARAR, even though background levels were exceeded. The COCs selected that exceed background levels, but do not have an RBSL or ARAR are discussed below. The COCs at each site that exceed an RBSL, ARAR, or both, are discussed in Sections 2.1.5.1 to 2.1.5.5.

Chemicals Without RBSLs and ARARs. Several chemicals detected above background levels could not be thoroughly screened because an RBSL could not be calculated and no ARAR was available (Table 2-1). A list of these chemicals is presented in Table 2-2. The cancer risk and

TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT POINT LAY

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL ^a		ARAR ^b	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Deactivated Landfill (LF01)	Sediment	GRPH	624	mg/kg	<50-<100	--	--	500 ^c	Yes
		GRPH	11.9	mg/kg	<3J-<4J	--	--	100 ^c	No
		Toluene	0.042	mg/kg	<0.030-<0.150	--	5,400	--	No
		Ethylbenzene	0.054	mg/kg	<0.030-<0.150	--	2,700	--	No
		Xylenes (total)	0.724	mg/kg	<0.030-<0.150	--	54,000	--	No
		Benzyl alcohol	1.38	mg/kg	<6.9-<15.0	--	8,100	--	No
		p-Isopropyltoluene	0.067	mg/kg	<0.030-<0.150	--	--	--	Yes*
		Naphthalene	0.409	mg/kg	<0.030-<0.150	--	1,100	--	No
		Tetrachloroethene	0.727	mg/kg	<0.030-<0.150	1.23	270	--	No
		1,2,4-Trimethylbenzene	0.552	mg/kg	<0.030-<0.150	--	--	--	Yes*
		1,3,5-Trimethylbenzene	0.342	mg/kg	<0.030-<0.150	--	--	--	Yes*
		Aluminum	2,400	mg/kg	1,500-25,000	--	--	--	No
		Barium	260	mg/kg	27-390	--	1,890	--	No
		Calcium	6,300	mg/kg	360-59,000	--	--	--	No
		Chromium	13	mg/kg	<4.3-47	--	135	--	No
		Copper	55	mg/kg	<2.7-45	--	999	--	No
		Iron	70,000	mg/kg	5,400-35,000	--	--	--	No
		Lead	18	mg/kg	<5.1-22	--	--	500 ^d	No
		Magnesium	1,900	mg/kg	360-7,400	--	--	--	No
		Manganese	220J	mg/kg	25-290	--	3,780	--	No
		Nickel	13	mg/kg	4.2-46	--	540	--	No
		Potassium	280	mg/kg	<300-2,200	--	--	--	No
		Sodium	120	mg/kg	<160-680	--	--	--	No

TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT POINT LAY (CONTINUED)

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL ^a		ARAR ^b	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Deactivated Landfill (LF01) (Continued)	Sediment (Continued)	Vanadium	11	mg/kg	6.3-59	--	189	--	No
		Zinc	380	mg/kg	9.2-95	--	8,100	--	No
	Surface Water ^f	DRPH	240	µg/L	<1,000	--	292	--	No
		GRPH	223	µg/L	<50J	50	730	--	Yes
		Benzene	23	µg/L	<1	0.617	--	5 ^c	Yes
		Toluene	9	µg/L	<1	--	96.5	1,000 ^f	No
		Ethylbenzene	13J	µg/L	<1	--	158	700 ^f	No
		Xylenes (total)	21.7	µg/L	<2	--	7,300	10,000 ^f	No
		cis-1,2-Dichloroethene	6.9	µg/L	<1.0	--	36.5	70 ^f	No
		Dichlorodifluoromethane	58	µg/L	<1	--	51.7	--	Yes
		p-Isopropyltoluene	1.7	µg/L	<1	--	--	--	Yes*
		Naphthalene	3.4J	µg/L	<1	--	150	--	No
		Tetrachloroethene	109	µg/L	<1	0.143	36.5	5 ^f	Yes
		Trichloroethene	3.3	µg/L	<1	0.25	--	5 ^e	Yes
		Trichlorofluoromethane	3.8	µg/L	<1	--	165	--	No
		1,2,4-Trimethylbenzene	10	µg/L	<1	--	--	--	Yes*
		1,3,5-Trimethylbenzene	6.7	µg/L	<1	--	--	--	Yes*
		Aluminum	180	µg/L	<100-350	--	--	--	No
		Barium	210	µg/L	<50-93	--	256	2,000 ^h	No
		Calcium	83,000	µg/L	4,500-85,000	--	--	--	No
		Iron	33,000	µg/L	180-2,800	--	--	--	No
		Magnesium	25,000	µg/L	<5,000-53,000	--	--	--	No

TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT POINT LAY (CONTINUED)

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL ^a		ARAR ^b	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Deactivated Landfill (LF01) (Continued)	Surface Water ¹ (Continued)	Manganese	960	µg/L	<50-510	-	18.3	-	Yes
		Potassium	8,100	µg/L	<5,000	-	-	-	No
		Sodium	47,000	µg/L	8,400-410,000	-	-	-	No
Garage (SS06)	Soil/Sediment	DRPH	33,400J	mg/kg	<50-<100	-	-	500 ^c	Yes
		GRPH	937	mg/kg	<3J-<4J	-	-	100 ^c	Yes
		RRPH	40,000	mg/kg	<100	-	-	2,000 ^c	Yes
		Benzene	0.3J	mg/kg	<0.02-<0.04	2.2	-	0.5 ^c	No
		Toluene	4	mg/kg	<0.02-<0.04	-	5,400	-	No
		Ethylbenzene	14	mg/kg	<0.03-<0.04	-	2,700	-	No
		Xylenes (Total)	42J	mg/kg	<0.04-<0.08	-	54,000	-	No
		cis-1,2-Dichloroethene	0.022J	mg/kg	<0.030-<0.150	-	270	-	No
		Isopropylbenzene	0.022J	mg/kg	<0.030-<0.150	-	-	-	Yes*
		p-Isopropyltoluene	0.042J	mg/kg	<0.030-<0.150	-	-	-	Yes*
		Methylene Chloride	0.23BJ	mg/kg	<0.030-<0.150	8.53	1,620	90 ^g	No
		Naphthalene	0.158J	mg/kg	<0.030-<0.150	-	1,100	-	No
		n-Propylbenzene	0.068J	mg/kg	<0.030-<0.150	-	-	-	Yes*
		Tetrachloroethene	43J	mg/kg	<0.03-<0.04	1.23	270	-	Yes
		1,1,2-Trichloroethane	1.8J	mg/kg	<0.03-<0.04	1.1	110	5 ^g	Yes
		Trichloroethene	2J	mg/kg	<0.03-<0.04	5.8	-	-	No
		1,2,4-Trimethylbenzene	0.315J	mg/kg	<0.030-<0.150	-	-	-	Yes*
		1,3,5-Trimethylbenzene	0.497J	mg/kg	<0.030-<0.150	-	-	-	Yes*
		Aluminum	2,300	mg/kg	1,500-25,000	-	-	-	No
		Barium	290	mg/kg	27-390	-	1,890	-	No
		Calcium	2,000	mg/kg	360-59,000	-	-	-	No

TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT POINT LAY (CONTINUED)

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL ^a		ARAR ^b	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Garage (SS06) (Continued)	Soil/Sediment (Continued)	Chromium	54	mg/kg	<4.3-47	--	135	--	No
		Iron	20,000	mg/kg	5,400-35,000	--	--	--	No
		Lead	195	mg/kg	<5.1-22	--	--	500 ^d	No
		Magnesium	1,300	mg/kg	360-7,400	--	--	--	No
		Manganese	270	mg/kg	25-290	--	3,780	--	No
		Nickel	16	mg/kg	4.2-46	--	540	--	No
		Potassium	430	mg/kg	<300-2,200	--	--	--	No
		Sodium	250	mg/kg	<160-680	--	--	--	No
		Vanadium	13	mg/kg	6.3-59	--	189	--	No
		Zinc	85	mg/kg	9.2-95	--	8,100	--	No
		Naphthalene	2.1	µg/L	<1	--	150	--	No
		1,3,5-Trimethylbenzene	1.2	µg/L	<1	--	--	--	Yes*
		Barium	360	µg/L	<50-93	--	256	2,000 ^h	Yes
Drainage Pathway from POL Tanks (SS07)	Surface Water ⁱ	Calcium	52,000	µg/L	4,100-88,000	--	--	--	No
		Iron	5,600	µg/L	<100-28,000	--	--	--	No
		Magnesium	26,000	µg/L	<5,000-54,000	--	--	--	No
		Manganese	1,700	µg/L	<50-510	--	18.3	--	Yes
		Sodium	45,000	µg/L	8,200-450,000	--	--	--	No
		DRPH	27.7J	mg/kg	<50-<100	--	--	500 ^c	No
		GRPH	3J	mg/kg	<3J-<4J	--	--	100 ^c	No
		Benzene	0.07J	mg/kg	<0.02-<0.04	2.2	--	0.5 ^c	No
		Toluene	0.04	mg/kg	<0.02-<0.04	--	5,400	--	No
		Ethylbenzene	0.2	mg/kg	<0.03-<0.04	--	2,700	--	No

TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT POINT LAY (CONTINUED)

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL ^a		ARAR ^b	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Drainage Pathway from POL Tanks (SS07) (Continued)	Sediment (Continued)	Xylenes (Total)	0.4J	mg/kg	<0.04-<0.08	--	54,000	--	No
		Naphthalene	0.030J	mg/kg	<0.04-<0.150	--	1,100	--	No
	Surface Water	DRPH	961	µg/L	<1,000	--	292	--	Yes
		GRPH	189	µg/L	<50	50	730	--	Yes
		Benzene	1.7	µg/L	<1	0.617	--	5 ^e	Yes
		Ethylbenzene	5J	µg/L	<1	--	158	700 ^f	No
		Xylenes (Total)	12J	µg/L	<2	--	7,300	10,000 ^f	No
		1,1-Dichloroethene	2.3	µg/L	<1	0.142	32.9	7 ^e	Yes
		cis-1,2-Dichloroethene	178	µg/L	<1	--	36.5	70 ^f	Yes
		trans-1,2-Dichloroethene	3.6	µg/L	<1	--	73	100 ^f	No
		Tetrachloroethene	2	µg/L	<1	0.143	36.5	5 ^f	Yes
		Trichloroethene	133	µg/L	<1	0.25	--	5 ^e	Yes
		DRPH	17,500	mg/kg	<50-<100	--	--	500 ^c	Yes
		GRPH	2,430	mg/kg	<3J-<4J	--	--	100 ^c	Yes
		RRPH	120	mg/kg	<100	--	--	2,000 ^c	No
Crushed Drum Area (SS08)	Soil/Sediment	Benzene	1.4	mg/kg	<0.02-<0.04	2.2	--	0.5 ^c	Yes
		Toluene	12.4	mg/kg	<0.02-<0.04	--	5,400	--	No
		Ethylbenzene	18.2	mg/kg	<0.03-<0.04	--	2,700	--	No
		Xylenes (Total)	91.6	mg/kg	<0.04-<0.08	--	54,000	--	No
		Acenaphthalene	1.85J	mg/kg	<6.9-<15.0	--	--	--	Yes*
		Benzyl Alcohol	0.680	mg/kg	<6.9-<15.0	--	8,100	--	No
		n-Butylbenzene	11.6	mg/kg	<0.03-<0.150	--	--	--	Yes*
		sec-Butylbenzene	5.03	mg/kg	<0.03-<0.150	--	--	--	Yes*
		di-n-Butylphthalate	8.48B	mg/kg	<6.9-<15.0	--	2,700	8,000 ^k	No

TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT POINT LAY (CONTINUED)

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL ^a		ARAR ^b	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Crushed Drum Area (SS08) (Continued)	Soil/Sediment (Continued)	Dibenzofuran	1.83J	mg/kg	<6.9-<15.0	-	-	-	Yes*
		Fluorene	1.95J	mg/kg	<6.9-<15.0	-	1,080	-	No
		Isopropylbenzene	3.23	mg/kg	<0.03-<0.150	-	-	-	Yes*
		p-Isopropyltoluene	6.04	mg/kg	<0.03-<0.150	-	-	-	Yes*
		2-Methylnaphthalene	11.7	mg/kg	<6.9-<15.0	-	-	-	Yes*
		Naphthalene	26.2	mg/kg	<0.03-<0.150	-	1,100	-	No
		Phenanthrene	2.44	mg/kg	<6.9-<15.0	-	-	-	Yes*
		n-Propylbenzene	7.88	mg/kg	<0.03-<0.150	-	-	-	Yes*
		1,2,4-Trimethylbenzene	48.0	mg/kg	<0.03-<0.150	-	-	-	Yes*
		1,3,5-Trimethylbenzene	19.6	mg/kg	<0.03-<0.150	-	-	-	Yes*
		Aluminum	3,400	mg/kg	1,500-25,000	-	-	-	No
		Barium	360	mg/kg	27-390	-	1,890	-	No
		Calcium	1,200	mg/kg	360-59,000	-	-	-	No
		Chromium	6.7	mg/kg	<4.3-47	-	135	-	No
		Copper	8.6	mg/kg	<2.7-45	-	999	-	No
		Iron	23,000	mg/kg	5,400-35,000	-	-	-	No
		Magnesium	1,600	mg/kg	360-7,400	-	-	-	No
		Manganese	220J	mg/kg	25-290	-	3,780	-	No
		Nickel	15	mg/kg	4.2-46	-	540	-	No
		Potassium	590	mg/kg	<300-2,200	-	-	-	No
		Selenium	17J	mg/kg	<7.8-<170	-	135	-	No
		Sodium	94	mg/kg	<160-680	-	-	-	No

TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT POINT LAY (CONTINUED)

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL ^a		ARAR ^b	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Crushed Drum Area (SS08) (Continued)	Soil/Sediment (Continued)	Vanadium	19	mg/kg	6.03-59	--	189	--	No
		Zinc	44	mg/kg	9.2-95	--	8,100	--	No
	Surface Water ^d	Benzene	15	µg/L	<1	0.617	--	5 ^e	Yes
		Toluene	43	µg/L	<1	--	96.5	1,000 ^f	No
		Ethylbenzene	12J	µg/L	<1	--	158	700 ^f	No
		Xylenes (Total)	82J	µg/L	<2	--	7,300	10,000 ^f	No
		p-Isopropyltoluene	1.2	µg/L	<1	--	--	--	Yes*
		Naphthalene	15	µg/L	<1	--	150	--	No
		n-Propylbenzene	1.3	µg/L	<1	--	--	--	Yes*
		1,2,4-Trimethylbenzene	14	µg/L	<1	--	--	--	Yes*
		1,3,5-Trimethylbenzene	11	µg/L	<1	--	--	--	Yes*
		Aluminum	120	µg/L	<100-350	--	--	--	No
		Barium	330	µg/L	<50-93	--	256	2,000 ^h	Yes
		Calcium	57,000	µg/L	4,500-88,000	--	--	--	No
		Iron	6,300	µg/L	180-2,800	--	--	--	No
		Magnesium	29,000	µg/L	2,900-53,000	--	--	--	No
		Manganese	610	µg/L	<50-510	--	18.3	--	Yes
		Potassium	5,400	µg/L	<5,000-5,000	--	--	--	No
		Sodium	28,000	µg/L	8,400-410,000	--	--	--	No

Chemicals without an RBSL or ARAR are considered chemicals of potential concern and are discussed in Section 2.1.5.

Risk-Based Screening Level.

Applicable or Relevant and Appropriate Requirement.

ADEC 1991.

OSWER 1991.

* a b c d

TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT POINT LAY (CONTINUED)

e	MCL, 52 FR 25690.
f	MCL, 56 FR 3526 (30 January 1991).
g	57 FR 31776 (17 July 1992).
h	MCL, 56 FR 30266 (01 January 1991).
i	ADEC 1991.
j	55 FR 30798 - Proposed Rule RCRA Corrective Action for SWMUs 40 CFR [Section 264.521 (a)(2)(i-iv)] Health-Based Criteria for Carcinogens.
k	55 FR 30798, Proposed Rule RCRA Corrective Action for SWMUs 40 CFR [Section 264.521 (a)(2)(i-iv)], Health-Based Criteria for Systematic Toxicant.
l	The concentrations reported for metals in surface water are total metals.
B	The analyte was detected in the associated blank.
J	Result is an estimate.
U	Non Detect.

TABLE 2-2. CHEMICALS WITHOUT RBSLS AND ARARS OBSERVED IN THE SOIL, SEDIMENT, OR SURFACE WATER AT THE POINT LAY INSTALLATION

SUBSTITUTED BENZENES	
1,2,4-Trimethylbenzene	
1,3,5-Trimethylbenzene	
p-Isopropyltoluene	
Isopropylbenzene	
n-Butylbenzene	
n-Propylbenzene	
sec-Butylbenzene	
POLYNUCLEAR AROMATIC HYDROCARBONS	
Acenaphthalene	
Dibenzofuran	
2-Methylnaphthalene	
Phenanthrene	
ESSENTIAL HUMAN NUTRIENTS	
Calcium	
Iron	
Magnesium	
Potassium	
Sodium	

noncancer hazard for these chemicals cannot, therefore, be quantified. This section is a qualitative discussion of the potential for these chemicals to cause toxicity among the receptor groups identified at the Point Lay installation. The essential human nutrients were discussed in Section 2.1.3 and will not be discussed further here. Essential nutrients are not considered COCs in this risk assessment.

The American Petroleum Institute (API) recently published an evaluation of the environmental fate, transport, and toxicity of twelve organic chemicals found frequently in petroleum products. The twelve were selected from a large list of "candidates" based on:

- Abundance in crude and refined petroleum products, including residual and used oils;
- Chemical/physical properties that represent a range of mobilities in soil and solubilities in aqueous environments; and
- Toxicity in mammals and aquatic organisms (API 1994).

Two of the chemicals detected at the Point Lay installation, 1,2,4-trimethylbenzene and 2-methylnaphthalene, were selected from the list of twelve chemicals (API 1994) and are used in this risk assessment as surrogates for the chemicals without RBSLs or ARARs. These chemicals have similar chemical structures and, therefore, will represent the substituted benzenes and the polynuclear aromatic hydrocarbons that do not have toxicity criteria (Table 2-2).

1,2,4-Trimethylbenzene has a low order of toxicity in mammals (API 1994). No effect was observed on the kidneys of rats that received 0.5 or 2.0 g/kg orally five days per week for four weeks. Inhalation of high concentrations of 1,2,4-trimethylbenzene produces central nervous system depression in humans and rats. Lung toxicity, including bronchitis, pneumonitis, and edema was also observed in humans. 1,2,4-Trimethylbenzene has not been observed to be carcinogenic or mutagenic in laboratory studies of rats and cultured mammalian cells. Potential exposure of receptors to 1,2,4-trimethylbenzene at the Point Lay installation would probably be limited to oral ingestion of soil and at the maximum concentration measured (48 mg/kg soil) would be expected to be nontoxic.¹ For the purposes of this risk assessment, 1,2,4-trimethylbenzene is considered to be a reasonable surrogate for the substituted benzenes observed at the Point Lay installation.

Because of the lack of toxicology information available for 2-methylnaphthalene, naphthalene will be used as a surrogate in this discussion of polynuclear aromatic hydrocarbons without RBSLs and ARARs. Naphthalene has a low order of toxicity in mammals (API 1994). The toxicology of this chemical has been well characterized in several species, including humans, rats, rabbits, and mice. The toxicity in humans is known from cases of accidental or intentional (suicide) ingestion

¹Based on the following calculation: assume average daily soil ingestion rate of 200 mg of soil per day and 48 mg of 1,2,4-trimethylbenzene per kg of soil (maximum concentration measured at the Point Lay installation). This yields a dose of 0.0001 mg of 1,2,4-trimethylbenzene per kg body weight per day. The oral dose of 1,2,4-trimethylbenzene received by rats that showed no kidney effects was equivalent to 2,000 mg of 1,2,4-trimethylbenzene for kg body weight, which is approximately 20,000,000 times greater than the estimated dose for potential receptors at the Point Lay installation.

of contaminated food or mothballs, and the most common effect is liver damage (jaundice) and destruction of red blood cells resulting in anemia. These effects occur at exposure levels that far exceed the levels to which the receptor groups at the Point Lay installation could be exposed. Dose-response information is available from studies in rats, mice, and rabbits. High doses of naphthalene administered over several days to one month resulted in cataract formation and other less serious ocular effects. High doses administered over several days to three months produced mild toxic effects on the liver, lung, kidney, and immunological system. The no effect level of oral exposure in these species occurs in the range of 100-300 mg naphthalene per kg body weight per day (100-300 mg/kg/day). The oral exposure levels to 2-methylnaphthalene that may occur through soil ingestion at the Point Lay installation are approximately 0.000033 mg/kg/day. Furthermore, the RBSL for naphthalene (1,100 mg/kg) exceeds the maximum concentration of 2-methylnaphthalene (11.7 mg/kg). Therefore, any exposure to 2-methylnaphthalene in the soil at the Point Lay installation is expected to be nontoxic.²

Dibenzofuran is a heterocyclic compound that is not usually considered a polynuclear aromatic hydrocarbon. Structurally this chemical is composed of two benzene rings attached to a furan ring which, except for the oxygen atom in the furan ring, is identical to fluorene. Toxicologically, little has been published regarding the toxicity of dibenzofuran. No toxicity values appear on IRIS and it has been classified as "data inadequate for quantitative risk assessment" on HEAST. The toxicology of another structurally similar compound, 2,3-benzofuran, has been described in ATSDR (1991a). Exposure of rats and mice to this compound for up to two years was associated with pathological changes in the pulmonary artery, stomach, liver, and kidney at doses of 30-60 mg/kg/day. Based on the maximum concentration of dibenzofuran observed in the soil at Point Lay, 1.83 mg/kg, the dose to the potential receptors is conservatively estimated to be 0.000005 mg/kg/day.³ Therefore, the levels of dibenzofuran in the soil at Point Lay is assumed to be nontoxic.

In conclusion, the chemicals without RBSLs and ARARs observed in the soil and surface water at the Point Lay installation do not pose a health risk for the receptor groups that will be evaluated in this risk assessment.

Chemicals with RBSLs and/or ARARs. Following are discussions of the COCs at each site that exceeded background levels and an RBSL, ARAR, or both. Table 2-3 is a summary of the COCs selected for the sites at the Point Lay installation.

2.1.5.1 Deactivated Landfill (LF01). DRPH were identified as a COC for the soil matrix at the Deactivated Landfill (Figure 2-2). The maximum concentration of DRPH exceeded the background concentration and the ARAR concentration for petroleum hydrocarbon contamination of soil (ADEC 1991) (Table 2-1).

²Based on the following assumptions: soil ingestion rate, 200 mg/kg; 70 kg body weight for typical receptor; maximum soil concentration of 2-methylnaphthalene, 11.7 mg/kg.

³Based on the assumption that receptors with an average body weight of 70 kg will consume 200 mg of soil per day containing 1.83 mg of dibenzofuran per kg of soil.

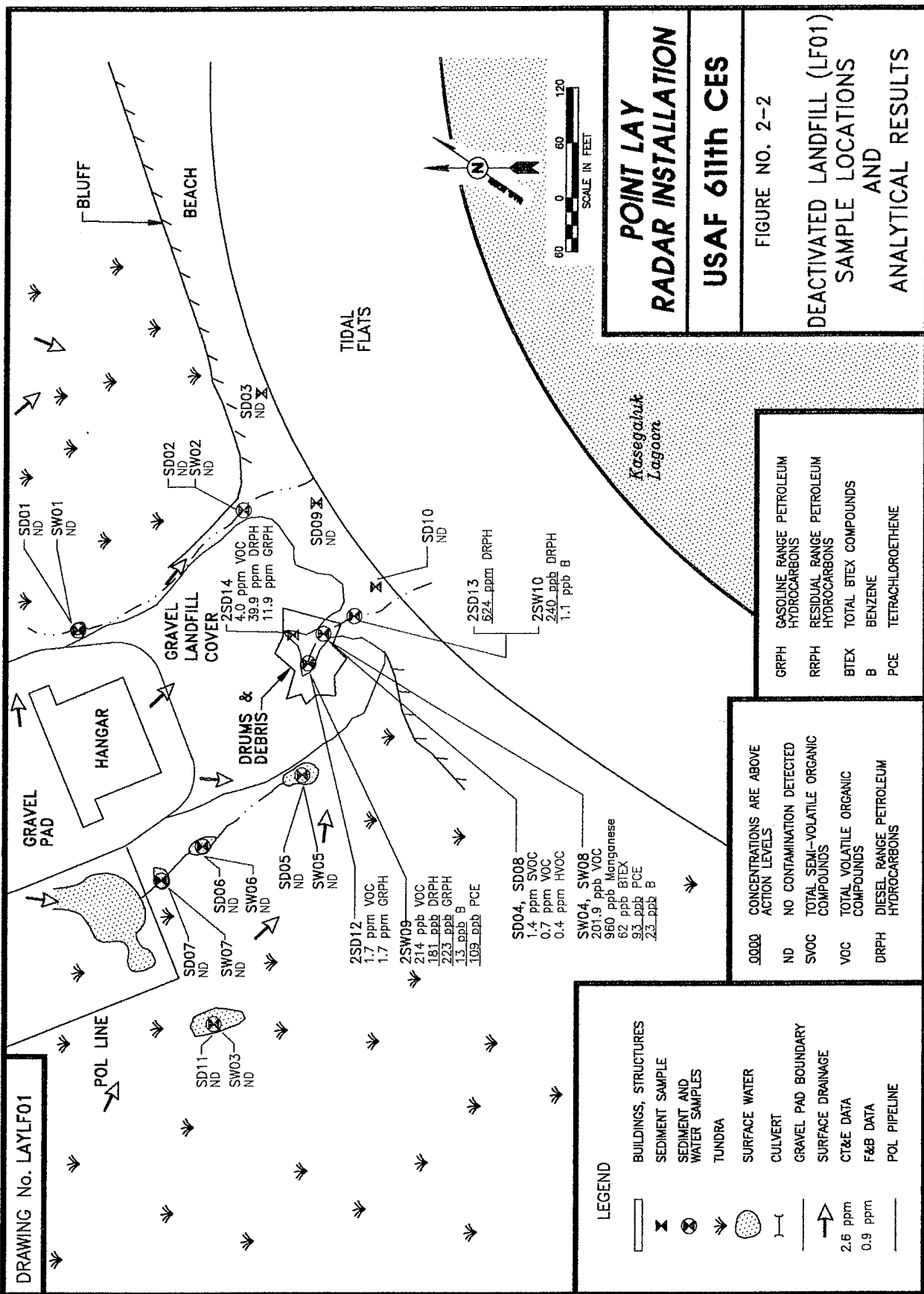
TABLE 2-3. SUMMARY OF THE CHEMICALS OF CONCERN AT POINT LAY

SITE	CHEMICALS OF CONCERN	
	SOIL/SEDIMENT	SURFACE WATER
Deactivated Landfill (LF01)	DRPH	GRPH Benzene Tetrachloroethene Trichloroethene Dichlorodifluoromethane Manganese
Garage (SS06)	DRPH GRPH RRPH Tetrachloroethene 1,1,2-Trichloroethane	Barium Manganese
Drainage Pathways from POL Tanks (SS07)	NONE	DRPH GRPH Benzene Tetrachloroethene Trichloroethene 1,1-Dichloroethene cis-1,2-Dichloroethene
Crushed Drum Area (SS08)	DRPH GRPH Benzene	Benzene Barium Manganese

* The summary of COCs on this table includes only those chemicals detected that exceed background levels and an RBSL, ARAR, or both. COCs that exceed background levels but do not have an RBSL or ARAR are discussed in Section 2.1.5. (Page 2-9).

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GRPH, benzene, tetrachloroethene, dichlorodifluoromethane, trichloroethene, and manganese were identified as COCs for surface water at the site (Figure 2-2). GRPH exceeded the RBSL based on cancer risk. Benzene exceeded the RBSL based on cancer risk and the ARAR concentration for benzene contamination of soil (ADEC 1991). Dichlorodifluoromethane and manganese exceeded their RBSLs based on noncancer hazard. Trichloroethene exceeded the RBSL based on cancer risk. Tetrachloroethene exceeded the RBSL based on noncancer hazard and cancer risk and the surface water ARAR, which is a maximum contaminant level (MCL) promulgated under the federal Safe Drinking Water Act (Table 2-1).

2.1.5.2 Garage (SS06). DRPH, GRPH, residual range petroleum hydrocarbons (RRPH), tetrachloroethene, and 1,1,2-trichloroethane were identified as COCs for the soil matrix at the Garage (Figure 2-3). The maximum concentrations of DRPH, GRPH, and RRPH exceeded the background concentrations and the ARAR concentrations for petroleum hydrocarbon contamination of soil (ADEC 1991) (Table 2-1). 1,1,2-Trichloroethane exceeded background, the RBSL based on cancer risk, and the ARAR which is an MCL promulgated under the Federal Safe Drinking Water Act. Tetrachloroethene exceeded the background concentration and the RBSL based on cancer risk.

Barium and manganese were identified as COCs for the surface water at the Garage (Figure 2-3). Both chemicals exceeded their background concentrations and RBSLs based on noncancer hazard (Table 2-1).

2.1.5.3 Drainage Pathway from POL Tanks (SS07). No COC was identified for the soil matrix at the Drainage Pathway from POL Tanks site (Figure 2-4, Table 2-1).

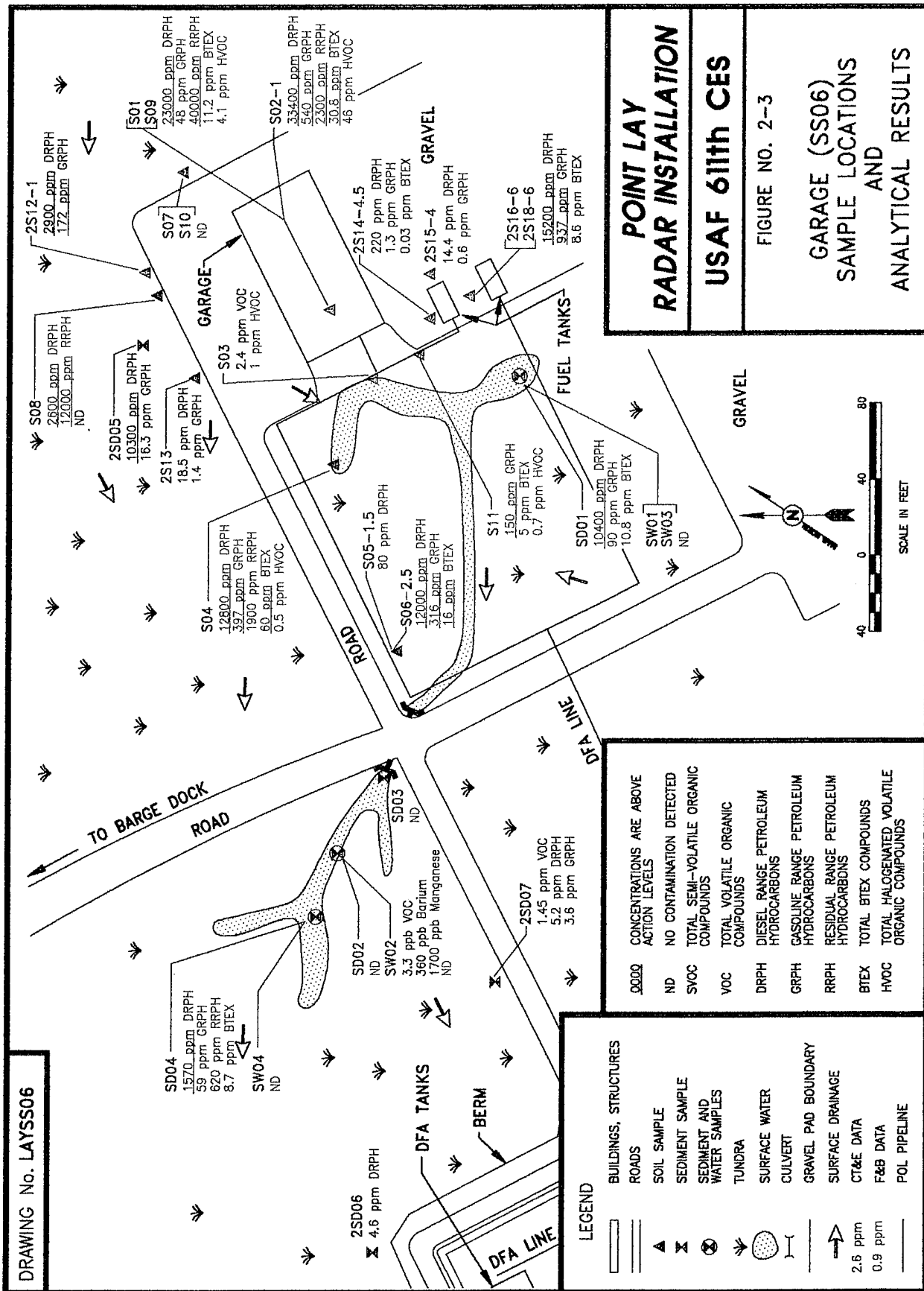
DRPH, GRPH, benzene, tetrachloroethene, trichloroethene, 1,1-dichloroethene, and cis-1,2-dichloroethene were identified as COCs for the surface water at the Drainage Pathway from POL Tanks site (Figure 2-4). DRPH and cis-1,2-dichloroethene exceeded their RBSLs based on noncancer hazard; cis-1,2-dichloroethene also exceeded an ARAR promulgated under the federal Safe Drinking Water Act. GRPH, benzene, tetrachloroethene, trichloroethene, and 1,1-dichloroethene exceeded their RBSLs based on cancer risk. In addition, trichloroethene exceeded an ARAR, which is an MCL promulgated under the federal Safe Drinking Water Act (Table 2-1).

2.1.5.4 Crushed Drum Area (SS08). DRPH, GRPH, and benzene were identified as COCs for the soil matrix at the Crushed Drum Area (Figure 2-5). DRPH and GRPH exceeded their background concentrations and the ARAR concentrations for petroleum hydrocarbon contamination of soil (ADEC 1991) (Table 2-1). Benzene exceeded the background concentration, the RBSL based on cancer risk, and the ARAR concentration for benzene in soil (ADEC 1991).

Benzene, barium, and manganese were identified as COCs for the surface water at the Crushed Drum Area (Figure 2-5). Benzene exceeded the background concentration, the RBSL based on

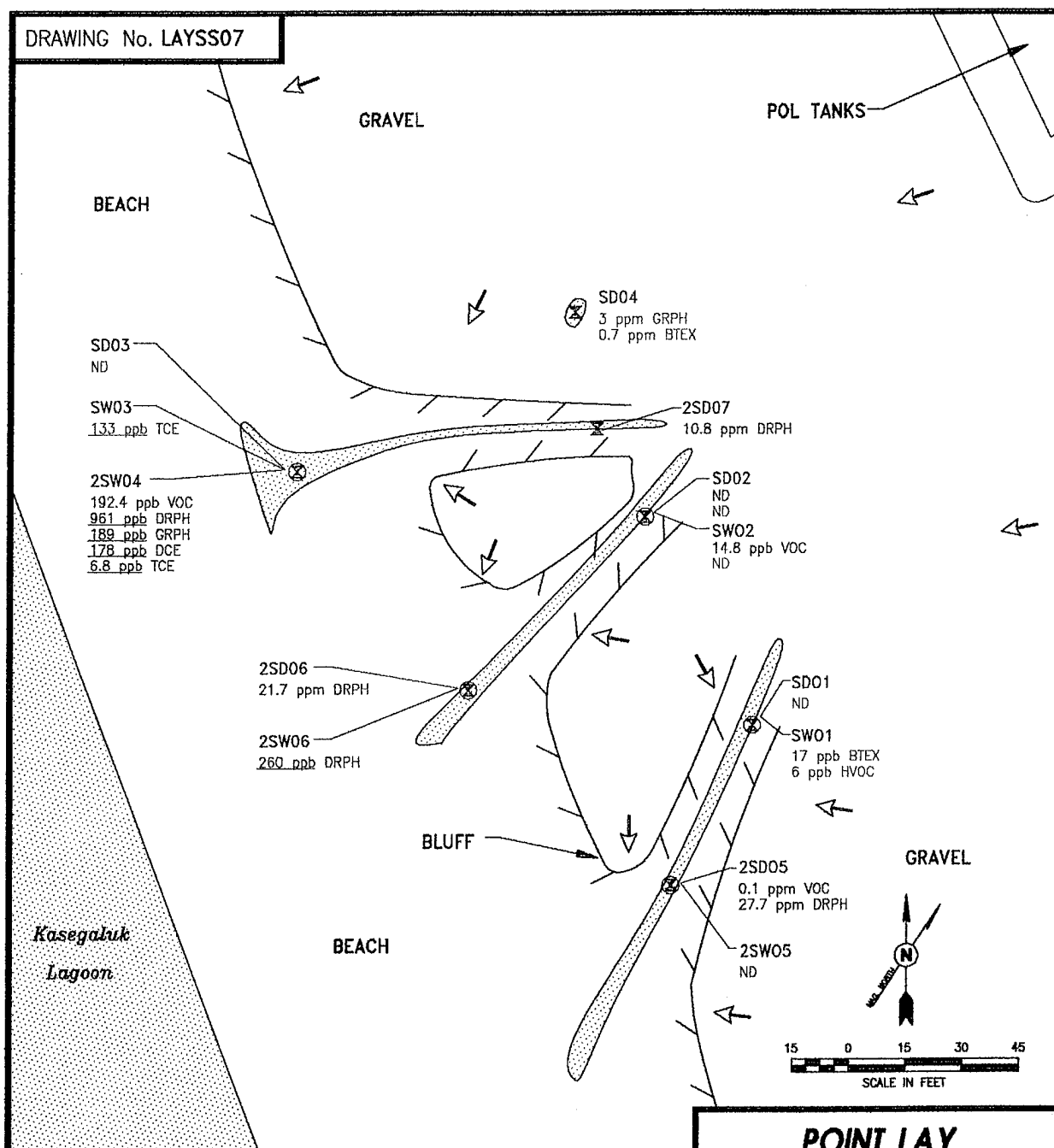
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DRAWING No. LAYSS06



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DRAWING No. LAYSS07



LEGEND

- BLUFF
- SEDIMENT SAMPLE
- SEDIMENT AND WATER SAMPLES
- SURFACE WATER
- GRAVEL PAD BOUNDARY
- SURFACE DRAINAGE
- 2.6 ppm CT&E DATA
- 0.9 ppm F&B DATA

0000 CONCENTRATIONS ARE ABOVE ACTION LEVELS

- ND NO CONTAMINATION DETECTED
- VOC TOTAL VOLATILE ORGANIC COMPOUNDS
- DRPH DIESEL RANGE PETROLEUM HYDROCARBONS
- GRPH GASOLINE RANGE PETROLEUM HYDROCARBONS
- BTEX TOTAL BTEX COMPOUNDS
- HVOC TOTAL HALOGENATED VOLATILE ORGANIC COMPOUNDS
- DCE cis-1,2-DICHLOROETHENE
- TCE TRICHLOROETHENE

POINT LAY RADAR INSTALLATION

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FIGURE NO. 2-4
DRAINAGE PATHWAY FROM
POL TANKS (SS07)
SAMPLE LOCATIONS
AND
ANALYTICAL RESULTS

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DRAWING No. LAYSS08

CONCENTRATIONS ARE ABOVE ACTION LEVELS

ND NO CONTAMINATION DETECTED
SVOC TOTAL SEMI-VOLATILE ORGANIC COMPOUNDS
VOC TOTAL VOLATILE ORGANIC COMPOUNDS
DRPH DIESEL RANGE PETROLEUM HYDROCARBONS
GRPH GASOLINE RANGE PETROLEUM HYDROCARBONS
RRPH RESIDUAL RANGE PETROLEUM HYDROCARBONS
BTX TOTAL BTX COMPOUNDS
B BENZENE

LEGEND

BUILDINGS, STRUCTURES
ROADS
SOIL SAMPLE
SEDIMENT SAMPLE
SEDIMENT AND WATER SAMPLES
TUNDRA
SURFACE WATER
CULVERT
GRAVEL PAD BOUNDARY
SURFACE DRAINAGE
CT&E DATA
F&B DATA

**POINT LAY
RADAR INSTALLATION**

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FIGURE NO. 2-5

**CRUSHED DRUM AREA (SS08)
SAMPLE LOCATIONS
AND
ANALYTICAL RESULTS**

SCALE IN FEET

MOBILE TRAIN

GRAVEL PAD

DITCH

CIRCULAR DEPRESSION

ROAD

CRUSHED DRUMS

SD01
6.1 ppm SVOC
57.4 ppm VOC
1100 ppm DRPH
46 ppm GRPH
120 ppm RRPH
10.8 ppm BTX

SW01
152 ppb VOC
330 ppb Barium
610 ppb Manganese
152 ppb BTX
15 ppb B

S01
7200 ppm DRPH
550 ppm GRPH

S07
550 ppm GRPH

2S08
1.2 ppm VOC
3050 ppm DRPH
22.6 ppm GRPH

2S12
10.5 ppm DRPH

2S10-1
17500 ppm DRPH
2330 ppm GRPH
122.2 ppm BTX

2S09
27.4 ppm SVOC
176.8 ppm VOC
6290 ppm DRPH
2430 ppm GRPH
49.2 ppm BTX

S03-2
ND

S04
ND

S05-2
ND

SD2-2
ND

SD02
ND

SD03
ND

SW02
ND

2S11
1.1 ppm VOC
4.2 ppm DRPH
2.0 ppm GRPH

2S15
1.1 ppm VOC
4.2 ppm DRPH
2.0 ppm GRPH

<u>0000</u>	CONCENTRATIONS ARE ABOVE ACTION LEVELS
ND	NO CONTAMINATION DETECTED
SVOC	TOTAL SEMI-VOLATILE ORGANIC COMPOUNDS
VOC	TOTAL VOLATILE ORGANIC COMPOUNDS
DRPH	DIESEL RANGE PETROLEUM HYDROCARBONS
GRPH	GASOLINE RANGE PETROLEUM HYDROCARBONS
RPHH	RESIDUAL RANGE PETROLEUM HYDROCARBONS
BTEX	TOTAL BTEX COMPOUNDS
B	BENZENE

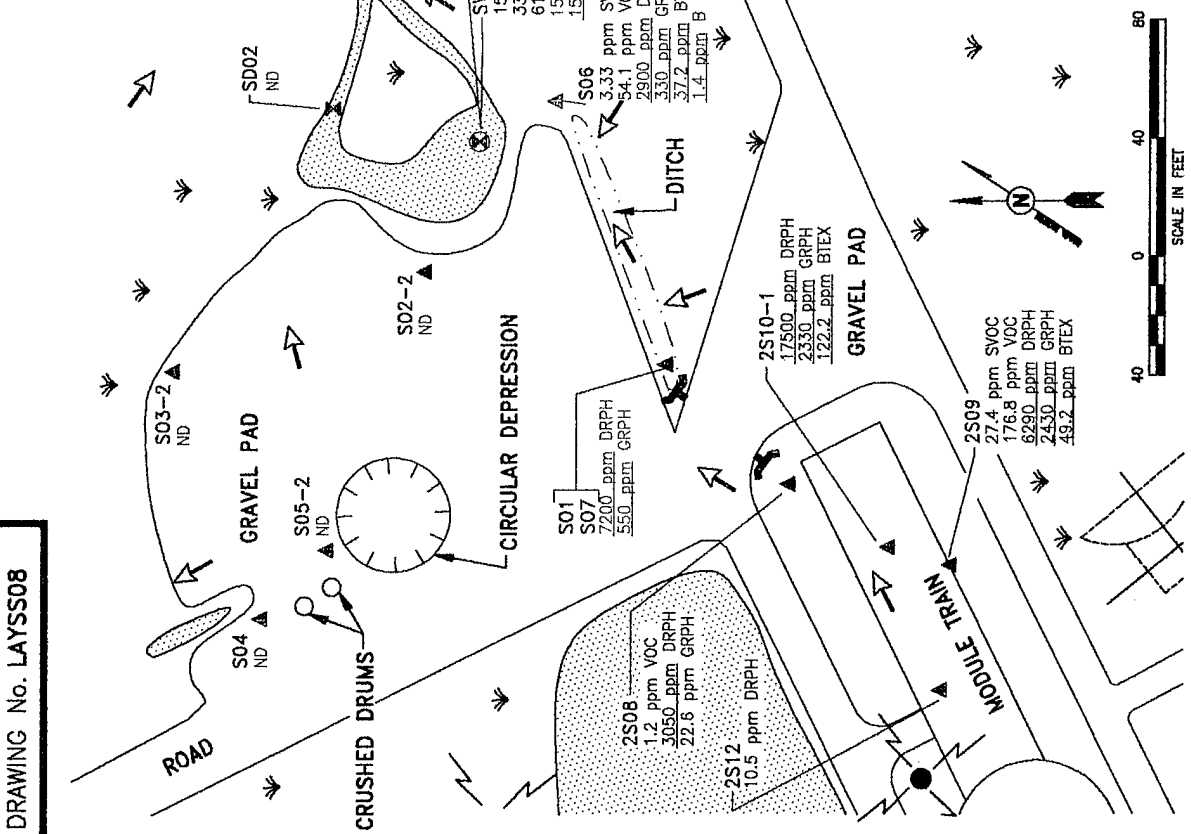
**POINT LAY
RADAR INSTALLATION**

USAF 611th CES

FIGURE NO. 2--5

CRUSHED DRUM AREA (SS08)
SAMPLE LOCATIONS
AND
ANALYTICAL RESULTS

	BUILDINGS, STRUCTURES
	ROADS
	SOIL SAMPLE
	SEDIMENT SAMPLE
	SEDIMENT AND WATER SAMPLES
	TUNDRA
	SURFACE WATER
	CULVERT
	GRAVEL PAD BOUNDARY
	SURFACE DRAINAGE
2.6 ppm	CT&E DATA
0.9 ppm	F&B DATA



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cancer risk, and the ARAR, which is an MCL promulgated under the federal Safe Drinking Water Act. Barium exceeded the background concentration and the RBSL based on noncancer risk. Manganese exceeded the background concentration and the RBSL based on noncancer hazard (Table 2-1).

2.1.5.5 Summary of Chemicals of Concern. The assessment of human health risk at the Point Lay radar installation is based on the COCs identified in this section. A summary of the COCs is presented in Table 2-3.

2.2 EXPOSURE ASSESSMENT

The exposure assessment section of a baseline human health risk assessment identifies and describes potential receptors and the exposure pathways by which exposure may occur, and estimates the magnitude of those exposures. This section includes an analysis of which pathways are complete (Section 2.2.1), migration and fate of COCs (Section 2.2.2), an estimation of the total intake of the chemicals (Section 2.2.3), and a summary of how the average daily dose (ADD) was calculated (Section 2.2.4).

2.2.1 Pathway Analysis

Pathway analysis involves the evaluation of the components of potential exposure pathways and a determination of whether each pathway is complete. An exposure pathway describes the course a chemical will take from a source to an exposure point where a receptor can come into contact with the chemical. A complete exposure pathway has five components:

- source of contamination;
- release mechanism;
- transport mechanism;
- exposure point; and
- receptor.

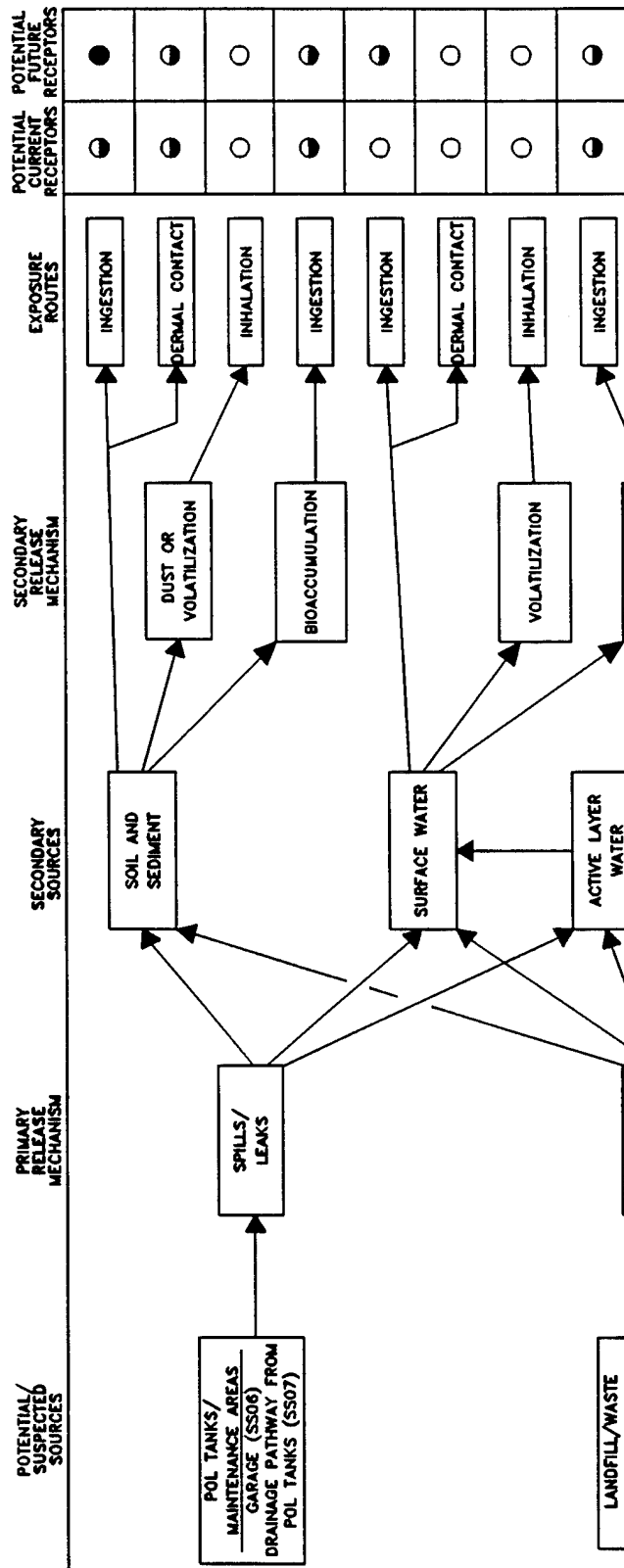
If one component of an exposure pathway does not exist, then exposure will not occur and there is no health risk. For example, if a shallow aquifer was contaminated with tetrachloroethene, but that aquifer was not used as a water supply, no exposure point would exist, and a ground water ingestion pathway would not be complete.

The potential exposure pathways evaluated for the Point Lay human health risk assessment are presented in Figure 2-6 and Table 2-4, and are discussed in Sections 2.2.1.1 through 2.2.1.4.

2.2.1.1 Soil and Sediment Ingestion. Point Lay installation workers and Point Lay residents may be exposed to soil and sediment contaminated by previous operations at the installation. The most likely exposure routes are incidental ingestion of soil and dermal absorption of contaminants in the soil. Site-specific characteristics will limit the magnitude, frequency, and duration of exposures to soil and sediment. The ground is covered with snow and ice, which eliminate soil or sediment exposure, for approximately nine months of the year.

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DRAWING No. LAY-FLOW



- COMPLETE EXPOSURE PATHWAY FOR HUMANS (DEWLINE WORKERS AND NATIVE NORTHERNERS)
- POTENTIALLY COMPLETE PATHWAY
- INCOMPLETE PATHWAY

**BULLEN POINT
RADAR INSTALLATION**

USAF 611th CES

FIGURE NO. 2-6

HUMAN HEALTH
RISK ASSESSMENT
POTENTIAL
EXPOSURE PATHWAYS

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TABLE 2-4. EXPOSURE PATHWAY ANALYSIS FOR POINT LAY HUMAN HEALTH RISK ASSESSMENT

POTENTIALLY CONTAMINATED MEDIUM	POTENTIAL ROUTES OF EXPOSURE	POTENTIAL RECEPTORS	PATHWAY COMPLETE?	EXPOSED POPULATION ESTIMATE
Soil	Ingestion, dermal absorption	DEW Line workers, native community residents	Ingestion, Yes Dermal Contact, No	192 ^a
Sediments	Ingestion, dermal absorption	DEW Line workers, native community residents	Ingestion, Yes Dermal Contact, No	192 ^a
Air	Inhalation of volatiles from soil or surface water or inhalation of fugitive dust	DEW Line workers, native community residents	No, volatile concentrations in soil and surface water are very low; dust generation is not likely because of marshy vegetated landscape and high humidity; and snow and ice cover most of the year.	0
Surface Water	Incidental ingestion, dermal absorption	DEW Line workers, native community residents	Maybe, drinking water supplies are either upgradient from installation or in unaffected areas. Site surface water could be used under a future use scenario. Fishing occurs in unaffected areas. Swimming does not occur on-site; however, incidental exposure may occur during installation operations or trespassing by native villagers.	192 ^a
Ground Water	Ingestion, dermal absorption	DEW Line workers, native community residents	No, permafrost limits presence of ground water to shallow active layer that is not used for any purpose.	0

^a Exposed population estimate is based on the population of the nearby village of Point Lay (Harcharek 1994).

In the summer months when snow cover is generally absent, cool temperatures (30°F to 46°F) (University of Alaska 1978) keep both workers and villagers in heavy, long-sleeved clothing and gloves that eliminate dermal contact with, and hand-to-mouth transfer of, soil. Therefore, although both the incidental soil ingestion and dermal contact pathways are unlikely to be complete, the incidental ingestion of soil or sediment will be evaluated further in this risk assessment in order to provide conservative estimates of risk.

The exposure assumptions used to evaluate the soil and sediment ingestion pathway are upper bound residential scenario assumptions and, therefore, probably overestimate the true hazard or risk associated with this pathway. The purpose of using residential assumptions is to evaluate the hazard or risk associated with future residential use of the Point Lay installation. Although the Air Force does not plan to convert the Point Lay radar installation to unmanned operation, it is possible that the installation may be retired and released for civilian use, in which case residential use of the installation may occur.

2.2.1.2 Inhalation. Point Lay installation workers and native northerners may be exposed to site contamination by inhalation of organic compounds volatilized from the soil or surface water, or by inhalation of windborne dust to which contamination has adsorbed. These exposure pathways are not considered complete for the Point Lay Risk Assessment because snow and ice cover the site for approximately nine months of the year, and during the summer months the high humidity, vegetative cover, and thawing of surface and active layer water significantly limit the entrainment of dust particles in ambient air. The generally low temperatures and high moisture content of the soil also tend to inhibit volatilization. The inhalation pathway will not be considered further in this risk assessment.

2.2.1.3 Water Ingestion. Surface water features, particularly those potentially contaminated by operations at the installation, are not likely to be used for drinking or other domestic purposes even on an incidental basis. This is because these surface water features are not reliable, being frozen for most of the year. Ingestion of surface water will, however, be considered a potentially complete exposure pathway to reflect the upper-bound potential future risk under a future use scenario. Under current conditions, surface water at the installation is not used for domestic or other purposes. Water for the area is obtained from a freshwater lake near the village of Point Lay. Water from the lake is pumped to a treatment plant and from there to two holding tanks with a 2.5 million gallon total capacity. From the holding tanks, water is delivered by truck to the houses in the village (Upicksoun 1995). This lake is upgradient and unaffected by contamination at the installation.

2.2.1.4 Ground Water. Permafrost limits the presence of ground water to the active layer, which thaws during the summer months. The water present in the active layer is not known to be used for any purpose; therefore, a ground water pathway will be eliminated from consideration in this risk assessment.

2.2.2 Migration and Fate of Chemicals of Concern

The COCs selected for Point Lay generally fall into three classes:

- Refined and residual petroleum hydrocarbons (DRPH, GRPH, and RRPH),
- Volatile and semi-volatile organic compounds (VOCs and SVOCs): benzene, dichlorodifluoromethane, 1,1-dichloroethene, cis-1,2-dichloroethene, tetrachloroethene, trichloroethene, and 1,1,2-trichloroethane), and
- metals (barium and manganese), and

This section presents a summary of the migration and fate of each of these classes given the environmental conditions at Point Lay.

Once released to the environment, the COCs are immediately subject to several processes, including evaporation and volatilization, bulk flow, soil adsorption, dissolution in surface or active layer water, biodegradation, and photooxidation. The extent to which the COCs undergo each of these processes depends on their chemical and physical properties (e.g., K_{OC} , K_{OW} , water solubility, vapor pressure, Henry's law constant), the volume released, soil flora, meteorological conditions, soil moisture, and organic carbon content.

The migration of petroleum hydrocarbons released to the gravel pads and tundra is expected to follow the rank order: GRPH > DRPH > RRPH. GRPH is generally considered to include hydrocarbons with carbon chain ranges from C5 to C12 that tend to be relatively mobile and less persistent than longer chain hydrocarbons. Depending on the length of time since a spill or leak occurred, the petroleum hydrocarbons observed in soil samples would be expected to be enriched in components that have carbon chain ranges greater than C10 or C11, have high K_{OC} and K_{OW} values, low vapor pressure and water solubility, that are not rapidly biodegradable. Petroleum components that fit this profile are higher molecular weight n-alkanes, mono- and poly-aromatics, and cycloalkanes. These components would tend to appear in laboratory analyses as diesel range or heavy oil range organics (DRPH and RRPH).

The migration of VOCs is expected to be rapid compared to the petroleum hydrocarbons. These compounds tend to have high vapor pressures which favor volatilization, high water solubility, and low K_{OC} and K_{OW} values. Therefore, the VOCs would tend to be highly mobile in the environment and dissipate rapidly after a spill or leak. In the results of field sampling, VOC concentrations would be expected to be fairly low depending on the time since the spill or leak occurred. The frigid conditions on the North Slope, however, would tend to reduce the mobility due to volatilization or evaporation.

In conclusion, the petroleum hydrocarbons and metals observed at the Point Lay installation are generally expected to be fairly persistent and of low mobility. The VOCs and SVOCs are expected to be less persistent and of greater mobility in the environment and, therefore, of less significance regarding human health risk. Exposure by contact with soils, primarily through accidental ingestion, is expected to predominate compared to exposure by inhalation.

The metals observed at Point Lay are probably of natural origin and not due to the operation or activities at the radar installation. The presence of manganese in water samples is often associated with leachate from landfills or contaminated areas since the anaerobic and acidic conditions tend to release naturally occurring manganese from the soil. Metals will tend to be persistent and of low mobility in the environment.

2.2.3 Estimation of Chemical Intake

The exposure assessment for the Point Lay DEW Line installation required the development of site-specific assumptions because of the unique location on the North Slope of Alaska. This section of the report focuses on the exposure variables for which site-specific assumptions were made. These variables include:

- exposure frequency;
- exposure duration;
- ingestion of locally produced meat (e.g., caribou, fish, and birds);
- ingestion of locally produced vegetation (e.g., berries);
- soil ingestion rate; and
- rate of dermal contact with soil.

The exposure assumptions used in the human health risk assessment are presented in Table 2-5.

Three potential receptor groups will be evaluated for the Point Lay Risk Assessment: an adult assigned to maintenance work at the Point Lay installation (DEW Line worker), an adult native of the North Slope of Alaska (native), and a native child (child). The native adult and child are considered to represent the reasonable maximum exposure that might occur at the installation under a future use scenario that includes residential receptors. Although there are no plans to do so, the Point Lay installation may be released for civilian residential use in the future.

The estimation of chemical intake requires the evaluation of several exposure variables: exposure point concentration; exposure frequency; exposure duration; averaging time; ingestion of locally produced meat, fish, and vegetation; soil ingestion; drinking water ingestion; dermal contact with soil; inhalation; and body weight. These exposure variables are discussed in the following sections.

2.2.3.1 Exposure Point Concentration. Based on the amount of analytical data available for the risk assessment of the Point Lay installation, and the requirement that the risk characterization be conducted individually for each of the four sites, only maximum concentrations of the COCs were used for exposure point concentrations. This approach yields a conservative upper-bound estimate of the average daily dose (ADD) to which potential receptors may be exposed.

2.2.3.2 Exposure Frequency. The exposure frequency variable is an estimate of the amount of time a potential receptor may contact contaminated media. For the DEW Line worker, the exposure frequency estimate is based on a duty rotation of eleven months on-site, one month off-site. These estimates are based on knowledge of the site worker's daily activities and

TABLE 2-5. EXPOSURE ASSUMPTIONS FOR ESTIMATING CHEMICAL INTAKE

PARAMETER	DEW LINE WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN CHILD
Exposure Frequency - Soil Ingestion (days/year)	30	30	30
Exposure Frequency - Water Ingestion (days/year)	180	180	N/A
Exposure Duration (years)	10	55 ^a	6 ^a
Soil Ingestion Rate (mg/day)	50	100	200
Drinking Water Ingestion Rate (L/day)	2	2	N/A
Average Body Weight (kg)	70	70	15
Averaging Time (days)	25,550 ^b (cancer) 3,650 ^{c,d} (noncancer)	25,550 ^b (cancer) 20,075 ^c (noncancer) 17,885 ^d (noncancer)	2,190 ^d (noncancer)

N/A Not applicable; drinking water pathway evaluated for adult only.

^a Exposure duration for water ingestion pathway is 55 years (future use scenario). For soil ingestion, exposure duration is 6 years as a child and 49 years as an adult.

^b Averaging time for the evaluation of cancer risk by the soil and water ingestion pathways.

^c Averaging time for the evaluation of noncancer hazard by the water ingestion pathway.

^d Averaging time for the evaluation of noncancer hazard by the soil ingestion pathway.

vacation allowance. During the eleven months of on-site duty, it is estimated that the worker is outside for four hours per day. The remaining 20 hours is spent inside the module trains or enclosed vehicles, where exposure to contaminated media is not expected to occur. An estimated exposure frequency for the DEW Line worker is, therefore, 11 months/year x 30 days/month x 4 hours/day x 1 day/24 hours = 55 days/year. The primary environmental medium of concern is, however, contaminated soil, and this estimate of exposure frequency does not account for the number of days per year that snow covers the ground and eliminates the potential for contact with contaminated soil. Six months is a conservative average estimate of the number of months per year of snow cover at the arctic DEW Line installations. To be even more conservative, it is assumed that a worker's tour of duty includes all six of the months without snow cover, thus soil ingestion exposure frequency of 30 days/year for the DEW Line worker is recommended.

The soil ingestion exposure frequency estimate for a native adult or child of the North Slope is based on an estimate of the frequency with which the individual would be at a DEW Line installation involved in activities that include exposure to soil. Such visits are likely to occur at installations sited near a village like Point Lay. In this case, a conservative estimate of exposure would be 4 hrs/day x 30 days per month x 1 day/24 hrs x 6 months of exposed soil = 30 days per year.

The exposure frequency for water ingestion was conservatively estimated at 180 days/year that surface water would be available (i.e., not frozen) and is based on a potential future scenario where site surface water is used as a drinking water source.

2.2.3.3 Exposure Duration. The exposure duration variable is an estimate of the amount of time a potential receptor will remain at or near a DEW Line installation over a lifetime. For the DEW Line worker the exposure duration is an estimate of the maximum tour of duty at an installation. A conservative estimate of the duration of a tour at a particular installation is 10 years based on conversations with DEW Line workers. For the potential native receptor, a conservative estimate of exposure duration is 55 years. EPA's default reasonable maximum exposure duration is 30 years; however, this is based on the overall U.S. population. Because the Alaskan natives are more likely to remain in their village for a longer period, 55 years was determined to be a more appropriate estimate based on best professional judgement.

2.2.3.4 Averaging Time. The averaging time represents the period of time over which exposure is averaged and is based on the assumption that intermittent exposure at a given contaminant concentration is equivalent to a continuous exposure at a lower concentration. For the DEW Line worker, the averaging time is based on the EPA default lifetime of 70 years for evaluation of carcinogens, and 10 years (equivalent to the exposure duration) for the evaluation of noncarcinogens. For the native northern adult an averaging time of 70 years for carcinogens was also chosen. To evaluate exposure to noncarcinogens in soil and sediment for the native northern adult and child, an averaging time of 49 years as an adult and 6 years as a child was used (to account for 55 year total exposure). To evaluate the exposure of native northern receptors to noncarcinogens in water an averaging time of 55 years was used.

2.2.3.5 Ingestion of Locally Produced Meat, Fish and Vegetation. The food supplies of DEW Line installation workers are largely imported from outside the area. Occasionally, a worker would be expected to ingest a locally caught fish or game animal, but the frequency and magnitude of this ingestion is expected to have a negligible effect on exposure to the COCs. Food supplies for the residents of Point Lay are partly imported from outside the area, and some reports indicate that the reliance on hunting and fishing for subsistence is decreasing as the economy moves from subsistence to wage labor (Chance 1990). However, although Inupiat in general have less time to hunt and fish than in the past, up to 50 percent of their food may derive from subsistence activities (Harcharek 1994). Most of the hunting and fishing occurs outside the village and away from the Point Lay DEW Line installation in areas unaffected by the installation. It is not likely that contamination observed at the installation has affected the mammals, birds, and vegetation that may be collected for consumption. Therefore, the consumption of locally produced food is unlikely to pose a significant risk of adverse health effects and will not be considered a complete exposure pathway. The ERA, Section 3.0, presents a detailed assessment of risks to ecological receptors.

2.2.3.6 Soil Ingestion Rate. A conservative approach to estimating soil ingestion rate is to assume the EPA default soil ingestion rates of 50 mg/day for workers (EPA 1991a) and 100 mg/day for adults in a residential setting is applicable to the Point Lay installation. The EPA default soil ingestion rate for children is 200 mg/day; this is the recommended value for the risk assessment.

2.2.3.7 Drinking Water Ingestion Rate. There are no circumstances at the Point Lay installation that would invalidate the EPA default adult drinking water ingestion rate of 2 L/day. Therefore, this is the recommended value for both workers and natives. However in most, if not all, cases drinking water is imported from offsite, so this may not be a complete route of exposure.

By convention (EPA 1989a), noncancer hazard and cancer risk associated with the drinking water pathway are evaluated for an adult receptor, not a child (Table 2-3). The basis for this approach is the ratio of drinking water ingestion rate to body weight that is assumed to remain relatively constant from childhood to adulthood.

2.2.3.8 Dermal Contact with Soil Rate. Because of the harsh North Slope weather, potential receptors (both workers and natives) are expected to be heavily clothed and gloved. Observations made by RI field personnel indicate that potential human receptors were heavily clothed during the months of the field investigations (August and September 1993). Therefore, dermal exposure to contaminated soils is considered negligible. In addition, the duties of installation workers that involve soil work (excavating, grading, etc.) are conducted in equipment with enclosed cabs. Thus, a dermal contact rate does not appear to be necessary for the exposure assessment.

2.2.3.9 Inhalation Rate. The inhalation pathway is not complete (Section 2.2.1.2), so no estimate for this variable is necessary.

2.2.3.10 Body Weight. There are no circumstances at the Point Lay installation that would invalidate the EPA default adult body weight of 70 kg. Therefore, this is the recommended value for both workers and natives. The recommended body weight for children is the EPA default value of 15 kg.

2.2.4 Quantifying Exposure

For each complete, or potentially complete, exposure pathway at the Point Lay installation (soils ingestion, drinking water ingestion), the ADD for estimating noncancer hazard and the lifetime average daily dose (LADD) for estimating excess lifetime cancer risk were calculated. The equations used for the calculation of ADD and LADD are presented in Table 2-6.

The exposure assumptions assigned to each variable in these equations are presented in Table 2-5. The estimates of ADD and LADD for the COCs at each site are presented in the risk characterization spreadsheets in Appendix A.

2.3 TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to weigh available evidence regarding the potential for particular contaminants to cause adverse effects in exposed individuals and to provide, where possible, an estimate of the relationship between the extent of exposure to a contaminant and the increased likelihood or severity of adverse effects or both. This is done separately for

TABLE 2-6. EQUATIONS USED FOR ESTIMATING POTENTIAL DOSE

EXPOSURE ROUTE	EQUATION	PARAMETER DEFINITIONS
Ingestion of Soil	Native Northern Adults/Children $ADD \text{ or } LADD \text{ (mg/kg/day)} = \frac{C_s * CF * EF}{AT} \sum_{i=1}^n \frac{IR_i * ED_i}{BW_i}$ DEW Line workers: $ADD \text{ or } LADD \text{ (mg/kg/day)} = \frac{C_s * CF * IR * EF * ED}{BW * AT}$	C_s = concentration in soil (mg/kg) CF = conversion factor (10^{-6} kg/mg) IR = ingestion rate (mg/day) EF = exposure frequency (days/year) ED = exposure duration (years) BW = body weight (kg) AT = averaging time (days/year x years)
Ingestion of Surface Water	$ADD \text{ or } LADD \text{ (mg/kg/day)} = \frac{C_w * CF * IR * EF * ED}{BW * AT}$	C_w = concentration in surface water ($\mu\text{g/L}$) CF = conversion facton (10^{-3} mg/μg) IR = ingestion rate (L/day) EF = exposure frequency (days/year) ED = exposure duration (years) BW = body weight (kg) AT = averaging time (days/year x years)

noncarcinogenic effects (Section 2.3.1) and carcinogenic effects (Section 2.3.2). Toxicity summaries are presented in Section 2.3.3.

Toxicity assessment for environmental contaminants generally is accomplished in two steps: hazard identification and dose-response assessment. Hazard identification is the process of determining whether exposure to an agent can cause an increase in the incidence of a particular adverse health effect (e.g., cancer, birth defects) and whether the adverse health effect is likely to occur in humans. Hazard identification involves characterizing the nature and strength of the evidence of causation. Dose-response evaluation is the process of quantitatively evaluating the toxicity information and characterizing the relationship between the dose of the contaminant administered or received and the incidence of adverse health effects in the exposed population. From this quantitative dose-response relationship, toxicity values (e.g., RfDs and SFs) are derived that can be used to estimate the incidence or potential for adverse effects as a function of human exposure to the agent. These toxicity values are used in the risk characterization step to estimate the likelihood of adverse effects occurring in humans at particular exposure levels.

2.3.1 Toxicity Assessment for Noncarcinogenic Effects

An RfD is the toxicity value used most often in evaluating noncarcinogenic effects resulting from exposures at contaminated sites. Various types of RfDs are available depending on the exposure route (oral or inhalation), the critical effect (developmental or other), and the length of exposure being evaluated (chronic, subchronic, or single event). The oral RfDs used to estimate the noncancer hazard associated with exposure to soils, sediments, and surface water at the Point Lay installation are presented in Table 2-7.

A chronic RfD is defined as an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chronic RfDs are developed specifically to be protective for long-term exposure to a compound. Chronic RfDs generally should be used to evaluate the potential noncancerous effects associated with exposure periods between 7 years (approximately 10 percent of a human lifetime) and a lifetime. Many chronic RfDs have been reviewed and verified by the intra-agency RfD Workgroup and entered into the EPA's IRIS database.

2.3.1.1 Concept of Threshold. For many noncancerous effects, protective mechanisms are believed to exist that must be overcome before the adverse effect is manifested. For example, where a large number of cells perform the same or similar function, the cell population may have to be significantly depleted before the adverse effect is seen. As a result, a range of exposures from zero to some finite level exists that can be tolerated by the organism with essentially no chance of expression of adverse effects. In developing a toxicity value for evaluating noncancerous effects (i.e., an RfD), the approach is to identify the upper bound of this tolerance range (i.e., the maximum subthreshold level). Because variability exists among humans, attempts are made to identify a subthreshold level that protects sensitive individuals in the population. For most chemicals, this level can only be estimated; the RfD incorporates uncertainty factors indicating the degree of extrapolation used to derive the estimated value. RfD summaries in IRIS also contain a statement expressing the overall confidence that the evaluators

TABLE 2-7. TOXICITY CRITERIA FOR NONCANCER EFFECTS OF THE CHEMICALS OF CONCERN FOR POINT LAY

CHEMICAL	ORAL REFERENCE DOSE (RfD) (mg/kg-day)	TARGET ORGAN OR CRITICAL EFFECT (species) ^a	UNCERTAINTY FACTOR ^b	ORAL RfD SOURCE ^c
Barium	0.07	NOAEL (humans)	3	IRIS
Benzene	NA	NA	NA	IRIS
DRPH	0.08 ^d	liver effects (mice)	10,000	ECAO
Dichlorodifluoromethane	0.2	reduced body weight (rats)	100	IRIS
1,1-Dichloroethene	0.009	hepatic lesions (rat)	1,000	IRIS
cis-1,2-Dichloroethene	0.01	decreased hematocrit and hemoglobin (rat)	3,000	HEAST
GRPH	0.2 ^d	decreased body weight (rats)	1000	ECAO
Manganese (water)	0.005	CNS effects (humans)	1	IRIS
RRPH	0.08 ^d	liver effects (mice)	10,000	ECAO
Tetrachloroethene	0.01	liver effects (mice)	1,000	IRIS
1,1,2-Trichloroethane	0.004	blood effects (mice)	1,000	IRIS
Trichloroethene	NA	NA	NA	IRIS

^a A target organ is the organ apparently most sensitive to the toxicity of a chemical. A critical effect is reported when EPA has not identified a target organ for the toxicity of a given chemical.

^b The uncertainty factors used to develop oral reference doses are generally applied in multiples of 10 to account for shortcomings in the toxicological database. The greater the uncertainty factor, the lower the confidence level that can be placed on that RfD. Factors of 10 are applied to account for human variability in toxic response, extrapolation from animal studies to humans, extrapolation of short-term exposures to long-term exposures, and the extrapolation of a lowest-observed adverse effect level (LOAEL) to a no observed adverse effect level (NOAEL).

^c Sources of oral RfD values are IRIS (Integrated Risk Information System), HEAST (Health Effects Assessment Summary Tables), or ECAO (The Environmental Criteria and Assessment Office of EPA).

^d Oral RfD values for DRPH, GRPH, and RRPH are based on (EPA 1992b) and are considered provisional.

CNS Central nervous system.

NA Not available.

NOAEL The oral RfD for barium is based on a No Observed Adverse Effects Level in humans who received barium in drinking water.

have in the RfD (high, medium, or low). The RfD is generally considered to have uncertainty spanning an order of magnitude or more, so the RfD should not be viewed as a strict scientific demarcation between levels that are toxic and nontoxic.

2.3.2 Toxicity Assessment For Carcinogenic Effects

An SF and the accompanying weight-of-evidence (WOE) determination are the toxicity data most commonly used to evaluate potential human carcinogenic risks. The methods EPA uses to derive these values are outlined below. Additional information can be obtained by consulting EPA's *Guidelines for Carcinogen Risk Assessment* (EPA 1986a) and IRIS Background Document #2 (IRIS 1995). The SFs for the COCs at Point Lay are presented in Table 2-8.

2.3.2.1 Concept of Nonthreshold Effects. Risk evaluation based on the presumption of a threshold is generally thought to be inappropriate for carcinogens. In the evaluation of carcinogenicity, EPA assumes that a small number of molecular events can evoke changes in a single cell and lead to uncontrolled cellular proliferation and eventually to a clinical state of disease (cancer). This hypothesized mechanism for carcinogenesis is referred to as "nonthreshold" because all levels of exposure pose a finite probability of causing the development of cancer. That is no dose is thought to be risk-free, and an effect threshold cannot be estimated. For carcinogens, EPA uses a two-part evaluation in which the substance first is assigned a WOE classification based on the evidence of its carcinogenicity, and then an SF is calculated.

2.3.2.2 Assigning a Weight-of-Evidence. In the first step of the evaluation, the carcinogenicity data are evaluated to determine the likelihood that the agent is a human carcinogen. The evidence is characterized separately for human studies and animal studies as sufficient, limited, inadequate, no data, or evidence of no effect. The characterizations of these two types of data are combined, and based on the extent to which the agent has been shown to be a carcinogen in experimental animals, humans, or both, the agent is given a provisional WOE classification. EPA scientists then adjust the provisional classification upward or downward, based on other supporting evidence of carcinogenicity.

The EPA classification system for WOE is shown in Table 2-9.

2.3.2.3 Generating a Slope Factor. For chemicals classified as known or probable human carcinogens, a toxicity value that defines quantitatively the relationship between dose and response (i.e., the slope factor) is calculated. Slope factors (SFs) typically are calculated for potential carcinogens in classes A, B1, and B2. Quantitative estimation of SFs for the chemicals in class C is done on a case-by-case basis.

Generally, the SF is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime. The SF is used in risk assessments to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen. SFs should always be accompanied by the WOE classification to indicate the strength of the evidence that the agent is a human carcinogen.

TABLE 2-8. TOXICITY VALUES FOR THE CARCINOGENICITY OF THE CHEMICALS OF CONCERN AT POINT LAY

CHEMICAL	WEIGHT-OF-EVIDENCE	TUMOR TYPE (species)	ORAL SLOPE FACTOR (kg-day/mg)	ORAL SLOPE FACTOR SOURCE ^a
Barium	NA	NA	NA	IRIS
Benzene	A	leukemia (humans)	0.029	IRIS
Dichlorodifluoromethane	NA	NA	NA	IRIS
cis-1,2-Dichloroethene	NA	NA	NA	IRIS
1,1-Dichloroethene	C	adrenal pheochromocytoma (rats)	0.6	IRIS
DRPH	NA	NA	NA	NA
GRPH	C	liver adenoma/carcinoma (mouse)	0.0017	ECAO
Manganese (water)	NA	NA	NA	IRIS
RRPH	NA	NA	NA	NA
Tetrachloroethene	C-B2	not specified	0.052	ECAO
1,1,2-Trichloroethane	C	heptacellular carcinoma	0.057	IRIS
Trichloroethene	C-B2	not specified	0.011	ECAO

^a IRIS, Integrated Risk Information System; ECAO, Environmental Criteria and Assessment Office of EPA.
NA Not available.

TABLE 2-9. EPA WEIGHT-OF-EVIDENCE CLASSIFICATION SYSTEM FOR CARCINOGENICITY

GROUP	DESCRIPTION
A	Human carcinogen.
B1 or B2	Probable human carcinogen.
	B1 indicates that limited human data are available.
	B2 indicates sufficient evidence in animals and inadequate or no evidence in humans.
C	Possible human carcinogen.
D	Not classifiable as to human carcinogenicity.
E	Evidence of noncarcinogenicity for humans.

2.3.2.4 Identifying the Appropriate Data Set. In deriving SFs, the available information about a chemical is evaluated, and an appropriate data set is selected. Human data of high quality are preferable to animal data. If animal data are used, the species that responds most similarly to humans (with respect to factors such as metabolism, physiology, and pharmacokinetics) is preferred. When no clear choice is possible, the most sensitive species is given the greatest emphasis. Occasionally, in situations where no single study is judged most appropriate yet several studies collectively support the estimate, the geometric mean of estimates from all studies may be adopted as the SF. This practice ensures the inclusion of all relevant data.

2.3.2.5 Extrapolating to Lower Doses. Because risk at low exposure levels is difficult to measure directly either by animal experiments or by epidemiologic studies, the development of an SF generally entails applying a model to the available data set and using the model to extrapolate from the relatively high doses administered to experimental animals (or the exposures noted in epidemiologic studies) to the lower exposure levels expected for human contact in the environment.

A number of mathematical models and procedures have been developed to extrapolate from carcinogenic responses observed at high doses to responses expected at low doses. Different extrapolation methods may provide a reasonable fit to the observed data but may lead to large differences in the projected risk at low dose.

In general, after the data are fit to the appropriate model, the upper 95th percent confidence limit of the slope of the resulting dose-response curve is calculated. This value is known as the SF and represents an upper 95th percent confidence limit on the probability of a response per unit intake of a chemical over a lifetime (i.e., there is only a 5 percent chance that the probability of a response could be greater than the estimated value on the basis of the experimental data and

model used). In some cases, SFs based on human dose-response data are based on the "best" estimate instead of the upper 95th percent confidence limits. Because the dose-response curve generally is linear only in the low-dose region, the SF estimate only holds true for low doses. Information concerning the limitations on use of SFs can be found in IRIS.

2.3.2.6 Summary of Dose-Response Parameters. Toxicity values for carcinogenic effects can be expressed in several ways. The SF generally is considered to be the upper 95th percent confidence limit of the slope of the dose-response curve and is expressed as (mg/kg-day)⁻¹. Thus:

$$\begin{aligned}\text{Slope factor} &= \text{risk per unit dose} \\ &= \text{risk per mg/kg-day}\end{aligned}$$

Where data permit, SFs listed in IRIS are based on absorbed doses, although many of them have been based on administered doses.

2.3.3 Summaries of the Toxicity of the Chemicals of Concern

Tables 2-7 and 2-8 present chronic cancer and noncancer health effects criteria (oral SFs and RfDs, respectively) for the COCs. The toxicological properties of the COCs and the toxicological basis of the health effects criteria listed in Tables 2-7 and 2-8 are discussed in Appendix B.

2.4 RISK CHARACTERIZATION

In the risk characterization, the toxicity and exposure assessments are summarized and integrated into quantitative and qualitative expressions of risk. To characterize potential noncancerous effects, comparisons are made between projected intakes (ADD) of substances and toxicity values (e.g., the RfD). To characterize potential carcinogenic effects, probabilities that an individual will develop cancer over a lifetime of exposure are estimated from projected intakes (LADD) and chemical-specific dose-response information (e.g., the SF). Major assumptions, scientific judgements and to the extent possible, estimates of the uncertainties embodied in the assessment are also presented. In this section methods of quantifying risks are discussed and applied to individual sites at the Point Lay installation.

2.4.1 Quantifying Risks

This section describes the steps for quantifying risk or hazard indices for both carcinogenic and noncancerous effects to be applied to each exposure pathway analyzed. The first two subsections cover procedures for individual substances and are followed by a subsection on procedures for quantifying risks associated with simultaneous exposures to several substances.

2.4.1.1 Risks from Individual Substances - Carcinogenic Effects. For carcinogens, risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen (i.e., incremental or excess individual lifetime cancer risk). The guidelines provided in this section are consistent with EPA (1986a).

For some carcinogens, there may be sufficient information on mechanism of action that a modification of the approach outlined below is warranted. Alternative approaches may be considered in consultation with ECAO on a case-by-case basis.

The SF converts estimated daily intakes averaged over a lifetime of exposure directly to incremental risk of an individual developing cancer. Because relatively low intakes (compared to those experienced by test animals) are most likely from environmental exposures, it generally can be assumed that the dose-response relationship will be linear in the low-dose portion of the multistage model dose-response curve. Under this assumption the SF is a constant, and risk will be directly related to intake. Thus, the linear form of the carcinogenic risk equation is usually applicable for estimating cancer risks. This linear low-dose equation is described below.

LINEAR LOW-DOSE CANCER RISK EQUATION

$$\text{Risk} = \text{LADD} \times \text{SF}$$

where:

Risk = a unitless probability (e.g., 2×10^{-5}) of an individual developing cancer;
LADD = LADD averaged over 70 years (mg/kg-day); and
SF = slope factor, expressed in (mg/kg-day)⁻¹

Because the SF is often an upper 95th percentile confidence limit of the probability of response based on experimental animal data used in the multistage model, the carcinogenic risk estimate generally will be an upper-bound estimate. This means that the "true risk" will probably not exceed the risk estimate derived through use of this model and is likely to be lower than predicted.

2.4.1.2 Noncancer Hazards from Individual Substances - Noncancerous Effects. The measure used to describe the potential for noncancerous toxicity in an individual is not expressed as the probability of an individual suffering an adverse effect. EPA does not at the present time use a probabilistic approach to estimate the potential for noncancerous health effects. Instead, the potential for noncancerous effects is evaluated by comparing an exposure level over a specified time period (e.g., some fraction of a lifetime) with a RfD derived for a similar exposure period. This ratio of exposure to toxicity is called a hazard quotient.

The noncancer HQ assumes there is a level of exposure (i.e., the RfD) below which it is unlikely even for sensitive populations to experience adverse health effects. If the exposure level (ADD) exceeds this threshold (i.e., if ADD/RfD exceeds unity), there may be concern for potential noncancer effects. As a rule, the greater the value of ADD/RfD above unity, the greater the level of concern. Ratios of ADD/RfD should not be interpreted as statistical probabilities; a ratio of 0.001 does not mean that there is a one in one thousand chance of the effect occurring. Further, it is important to emphasize that the level of concern does not increase linearly as the RfD is approached or exceeded because RfDs do not have equal accuracy or precision and are not based on the same severity of toxic effects. Thus, the slopes of the dose-response curve in excess of the RfD can range widely depending on the substance.

NONCANCER HAZARD QUOTIENT

Noncancer Hazard Quotient = ADD/RfD

where:

ADD = average daily dose (or intake);

RfD = reference dose

ADD and RfD are expressed in the same units and represent the same exposure period (e.g., chronic, subchronic, or short-term).

2.4.1.3 Aggregate Risks for Multiple Substances. Estimating risk or hazard potential by considering one chemical at a time might significantly underestimate the risks associated with simultaneous exposures to several substances. To assess the overall potential for cancer and noncancer effects posed by multiple chemicals, EPA has developed *Guidelines for the Health Risk Assessment of Chemical Mixtures* (EPA 1986b). These guidelines can be applied to the case of simultaneous exposures to several chemicals from a variety of sources by more than one exposure pathway. Information on specific mixtures is rarely available. Even if such data exist, they are often difficult to use. Monitoring for "mixtures" or modeling the movement of mixtures across space and time present significant technical problems given the likelihood that individual components will behave differently in the environment (i.e., fate and transport).

Although the calculation procedures differ for carcinogenic and noncarcinogenic effects, both sets of procedures assume dose additivity in the absence of information on specific mixtures.

Carcinogenic Effects. The cancer risk equation described below is used to estimate the incremental individual lifetime cancer risk for simultaneous exposure to several carcinogens based on EPA's risk assessment guidelines. This equation represents an approximation of the precise equation for combining risks, that accounts for the joint probabilities of the same individual developing cancer as a consequence of exposure to two or more carcinogens. The difference between the precise equation and the approximation described in the equation below is negligible for total cancer risks less than 0.1. Thus, the simple additive equation is appropriate for most risk assessments.

CANCER RISK EQUATION FOR MULTIPLE SUBSTANCES

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

where:

Risk_T = the total cancer risk, expressed as a unitless probability; and

Risk_i = the risk estimate for the i^{th} substance.

The risk summation techniques described in the cancer risk equation above assume that intakes of individual substances are small. They also assume independence of action by the compounds involved (i.e., there are no synergistic or antagonistic chemical interactions and all chemicals

produce the same effect, i.e., cancer). If these assumptions are incorrect, over- or under-estimation of the actual multiple-substance risk could result.

A separate total cancer risk for each exposure pathway is calculated by summing the substance-specific cancer risks. Resulting cancer risk estimates should be expressed using one significant figure only.

There are several limitations to this approach. First, because each SF is an upper 95th percentile estimate of potency and upper 95th percentiles of probability distributions are not strictly additive, the total cancer risk estimate might become artificially more conservative as risks from a number of different carcinogens are summed. If one or two carcinogens drive the risk, however, this problem is not of concern. Second, it often will be the case that substances with different weights of evidence for human carcinogenicity are included. The cancer risk equation for multiple substances sums all carcinogens equally, giving as much weight to class B or C as to class A carcinogens. In addition, SFs derived from animal data will be given the same weight as SFs derived from human data. Finally, the action of two different carcinogens might not be independent.

Noncancerous Effects. To assess the overall potential for noncancerous effects posed by more than one chemical, a hazard index approach has been developed based on EPA's *Guidelines for Health Risk Assessment of Chemical Mixtures* (EPA 1986b). This approach assumes that simultaneous subthreshold exposures to several chemicals could result in an adverse health effect. It also assumes that the magnitude of the adverse effect will be proportional to the sum of the ratios of the subthreshold exposures. The hazard index (HI) is equal to the sum of the HQs. When the hazard index exceeds unity, there may be concern for potential health effects. Any single chemical with an exposure level greater than the toxicity value will cause the hazard index to exceed unity, and for multiple chemical exposures, the hazard index can exceed unity even if no single chemical exposure exceeds its RfD. The equation used to determine noncancer hazard index is as follows:

NONCANCER HAZARD INDEX

$$\text{Hazard Index} = \text{ADD}_1/\text{RfD}_1 + \text{ADD}_2/\text{RfD}_2 + \dots + \text{ADD}_i/\text{RfD}_i$$

where:

ADD_i = average daily dose (or intake) for the i^{th} toxicant;

RfD_i = reference dose for the i^{th} toxicant; and

ADD and RfD are expressed in the same units and represent the same exposure period (i.e., chronic, subchronic, or shorter-term).

Where appropriate, a separate chronic hazard index can be calculated from the ratios of the chronic daily intake (CDI) to the chronic RfD for individual chemicals as described below.

CHRONIC NONCANCER HAZARD INDEX

$$\text{Chronic Hazard Index} = \text{LADD}_1/\text{RfD}_1 + \text{LADD}_2/\text{RfD}_2 + \dots + \text{LADD}_i/\text{RfD}_i$$

where:

LADD_i = lifetime average daily dose for the i^{th} toxicant in mg/kg-day, and

RfD_i = chronic reference dose for the i^{th} toxicant in mg/kg-day.

There are several limitations to this approach. As mentioned earlier, the level of concern does not increase linearly as the RfD is approached or exceeded because the RfDs do not have equal accuracy or precision and are not based on the same severity of effect. Moreover, HQs are combined for substances with RfDs based on critical effects of varying toxicological significance. It will often be the case that RfDs of varying levels of confidence, including different uncertainty adjustments and modifying factors, will be combined (e.g., extrapolation from animals to humans, from lowest-observed adverse effect levels (LOAELs) to no observed adverse effect levels (NOAELs), and from one exposure duration to another).

Another limitation with the hazard index approach is that the assumption of dose additivity is most properly applied to compounds that induce the same effect by the same mechanism of action. Consequently, application of the hazard index equation to a number of compounds that are not expected to induce the same type of effects or that do not act by the same mechanism could overestimate the potential for effects. Such an approach is, however, appropriate at a screening level. This possibility is generally not of concern if only one or two substances are responsible for driving the hazard index above unity. If the hazard index is greater than unity as a consequence of summing several HQs of similar value, it is appropriate to segregate the compounds by effect and by mechanism of action and derive separate hazard indices for each group.

2.4.2 Site-Specific Risk Characterization

Soil and Sediment Exposures. The quantification of noncancer hazard and excess lifetime cancer risk associated with the soil ingestion pathway at Point Lay was based on analytical data from soil and sediment samples collected within the interval from ground surface to permafrost. No attempt was made to segregate surface soil samples from subsurface samples in the human health risk characterization.

The noncancer hazard and the excess lifetime cancer risk associated with the ingestion of soil or sediment containing COCs has been estimated for an hypothetical native northerner based on six years of exposure as a child and 49 years of exposure as an adult. For the DEW Line worker, cancer risk has been estimated based on ten years of exposure averaged over a default lifetime of 70 years. Noncancer hazard for the DEW Line worker was based on a 10 year exposure.

Surface Water Exposures. The noncancer hazard and the excess lifetime cancer risk associated with the ingestion of surface water containing COCs has been estimated based on a native northern adult and a DEW Line worker. A native northern child receptor was not

considered because, unlike exposure to soil, which is expected to be greater in a child than in an adult, because the ratio of drinking water ingestion rate to body weight is assumed to be relatively constant from childhood to adulthood, a greater drinking water ingestion rate is assumed for adults. Since a greater number of years is spent as an adult, estimating hazard or risk for water ingestion based on an adult is a more conservative approach. The exposure duration estimate for the DEW Line worker was 10 years and for the native northern adult, 55 years. Exposures were averaged over a 10 year period for DEW Line worker exposure to noncarcinogens, and 55 years for native northern adult exposure to noncarcinogens. Exposures were averaged over a 70 year period for both receptor groups to characterize the risk associated with exposure to carcinogens in surface water.

Ingestion of surface water at the Point Lay installation is not considered to be a complete pathway under a current use scenario. The residents of Point Lay currently receive their domestic water from a freshwater lake near the village. Water from the lake is transported by a pipeline to a treatment facility in the city and then distributed throughout the village by truck. Under a future use scenario, however, it is possible that the buildings at the Point Lay installation could be used for residences or additional residential structures could be erected at the installation. Therefore, because sources of water may change in the future, potential ingestion of surface water at the installation will be evaluated for the DEW Line worker and native northern adult under a future use exposure scenario only.

Table 2-10 contains a site-by-site summary of the COCs in each medium, and the noncancer hazard and excess lifetime cancer risk associated with exposure to the COCs in the soils, sediments, and surface water. COCs without toxicity data (RBSLs or ARARs) are not included on Table 2-10, but are discussed in Section 2.1.5. Appendix A contains the spreadsheets used to calculate the noncancer hazard and excess lifetime cancer risk estimates presented in Table 2-10.

Risk Characterization of Petroleum Hydrocarbons. Petroleum hydrocarbons represent a primary source of contamination at the Point Lay installation. The laboratory analysis of soil, sediment, and surface water samples revealed the presence of DRPH, GRPH, and RRPH. To characterize the risk associated with exposure to these compounds, we applied the provisional RfDs and the SF developed by EPA for petroleum hydrocarbons (EPA 1992b). These provisional RfDs provide the best available tool for characterizing the risk associated with exposure to the petroleum hydrocarbons. The RfD for JP-4 presented in EPA (1992b) was used to represent DRPH and RRPH, and the RfD and SF for unleaded gasoline were used to represent GRPH.

The noncancer hazard associated with exposure to DRPH, GRPH, and RRPH was estimated by dividing the compound- and site-specific ADD by the appropriate provisional RfD (EPA 1992b). The excess lifetime cancer risk associated with exposure to GRPH was estimated by multiplying the compound- and site-specific LADD by the SF for unleaded gasoline (EPA 1992b).

Although the provisional RfDs and SF represent the best available numerical estimate of toxicity, there is a significant amount of uncertainty associated with their use at the Point Lay installation. The RfDs and SF are based on studies in mice and rats by the inhalation route of exposure;

TABLE 2-10. SUMMARY OF NONCANCER HAZARD AND EXCESS LIFETIME CANCER RISK FOR POINT LAY

SITE	MEDIUM	NONCANCER CHEMICALS OF CONCERN ^a	NONCANCER HAZARD ^c			CARCINOGENIC CHEMICALS OF CONCERN ^a	EXCESS LIFETIME CANCER RISK ^d		
			DEW LINE WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN ADULT/CHILD		DEW LINE WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN ADULT/CHILD
Deactivated Landfill (LF01)	Soil/Sediment	DRPH	<0.001	ND ^e	0.009	None	---	---	---
	Surface Water	GRPH Manganese Tetrachloroethene Dichlorodifluoromethane	3	3	NA ^f	GRPH Benzene Tetrachloroethene Trichloroethene	1 x 10 ⁻⁵	7 x 10 ⁻⁵	NA ^f
Garage (SS06)	Soil/Sediment	DRPH GRPH RRPH Tetrachloroethene 1,1,2-Trichloroethane	0.05	NA ^e	1	GRPH Tetrachloroethene 1,1,2-Trichloroethane	3 x 10 ⁻⁸	NA ^e	7 x 10 ⁻⁷
	Surface Water	Barium Manganese	5	5	NA	None ^b	---	---	---
Drainage Pathway from POL Tanks (SS07)	Soil/Sediment	None ^b	---	---	---	None ^b	---	---	---
	Surface Water	DRPH GRPH Tetrachloroethene 1,1-Dichloroethene cis-1,2-Dichloroethene	0.4	0.4	NA	GRPH Benzene 1,1-Dichloroethene Tetrachloroethene Trichloroethene	7 x 10 ⁻⁶	4 x 10 ⁻⁵	NA ^f
Crushed Drum Area (SS08)	Soil	DRPH GRPH	0.01	NA ^e	0.3	GRPH Benzene	3 x 10 ⁻⁸	NA ^e	7 x 10 ⁻⁷
	Surface Water	Barium Manganese	2	2	NA	Benzene	9 x 10 ⁻⁷	5 x 10 ⁻⁶	NA ^f

BOLD Hazard or risk exceeds regulatory benchmarks: HQ > 1, Cancer risk > 1 x 10⁻⁶.

^a All COCs are listed together regardless of whether they contribute to the hazard index, cancer risk, or both.

^b None, no COCs selected.

^c Hazard index, noncancer hazard index. The hazard index is the sum of the HQs for all of the COCs associated with a given medium, pathway, and receptor group.

^d Cancer risk, excess lifetime cancer risk. The cancer risk is the sum of the excess lifetime cancer risks for all of the carcinogenic COCs associated with a given medium, pathway, and receptor group.

^e Children are assumed to have a soil ingestion rate greater than that for adults. Therefore, under a residential scenario, the estimates of noncancer hazard and cancer risk associated with soil ingestion are estimated for a combined adult and child receptor only. This estimate is considered a conservative upper bound on the true hazard or risk.

^f Drinking water ingestion, unlike soil ingestion, is evaluated for an adult receptor but not a child receptor because adults are assumed to have a longer exposure duration at a greater water ingestion rate. Therefore, the hazard or risk estimated will represent an upper bound, conservative estimate. For soil ingestion, the child soil ingestion rate is assumed to exceed that for adults. Therefore, a combination of the adult and child receptor groups is used to evaluate soil ingestion risk and hazard.

however, for this risk assessment, exposure of humans by the ingestion route is being evaluated. Furthermore, in the absence of a more thorough study to compare the DRPH, GRPH, and RRPH to known petroleum refinery streams, it is not clear how well the provisional values represent the toxicity of diesel and gasoline in humans.

Risk Characterization of Chemicals Detected. Chemicals detected above background levels without RBSLs or ARARs are evaluated in Section 2.1.5 (page 2-9). Based on the information in that section, and the relatively low levels detected at the sites, these chemicals are not expected to pose a health risk. Risk characterizations of chemicals detected that exceed RBSL, ARARs, or both are discussed on a site-by-site basis below.

2.4.2.1 Deactivated Landfill (LF01).

Soils and Sediments. The noncancer hazard associated with the ingestion of soil at the Deactivated Landfill by an hypothetical native northern adult/child is 0.009, and by a DEW Line worker is less than 0.001, based on the maximum concentration of the COC (Tables 2-10 and A-1). The presence of DPRH accounts entirely for the quantifiable noncancer hazard for these receptor/pathway combinations.

No carcinogenic COC was identified for the soil/sediment at this site; therefore, the excess lifetime cancer risk associated with ingestion of soil/sediment cannot be quantified.

Surface Water. The noncancer hazard associated with the ingestion of surface water at the Deactivated Landfill by a hypothetical native northern adult or by a DEW Line worker is 3, based on the maximum concentrations of the COCs (Tables 2-10 and A-2). Manganese, GRPH, tetrachloroethene, and dichlorodifluoromethane account for the quantifiable noncancer hazard for these receptor/pathway combinations. Manganese accounts for more than 90 percent of this noncancer hazard.

The excess lifetime cancer risk associated with the ingestion of surface water at this site by a native northern adult is 7×10^{-5} , and by a DEW Line worker is 1×10^{-5} , based on the maximum concentrations of the COCs (Table A-3). The presence of GRPH, benzene, tetrachloroethene, and trichloroethene accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations. Tetrachloroethene accounts for 90 percent or more of the cancer risk.

2.4.2.2 Garage (SS06).

Soils and Sediments. The noncancer hazard associated with the ingestion of soil at the Garage by an hypothetical native northern adult/child is 1, and by a DEW Line worker is 0.05, based on the maximum concentrations of the COCs (Tables 2-10 and A-4). The presence of DRPH, GRPH, RRPH, 1,1,2-trichloroethane, and tetrachloroethene accounts entirely for the quantifiable noncancer hazard for these receptor/pathway combinations. DRPH and RRPH together account for more than 90 percent of the noncancer hazard.

The excess lifetime cancer risk associated with the ingestion of soil or sediment at this site by an hypothetical native northern adult/child is 7×10^{-7} , and by a DEW Line worker is 3×10^{-8} , based on the maximum concentrations of the COCs (Table A-5). The presence of GRPH, 1,1,2-trichloroethane, and tetrachloroethene accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

Surface Water. The noncancer hazard associated with the ingestion of surface water at the Garage by a hypothetical native northern adult or by a DEW Line worker is 5, based on the maximum concentrations of the COCs (Tables 2-10 and A-6). Manganese and barium account for the quantifiable noncancer hazard for these receptor/pathway combinations. Manganese accounts for 99 percent of the noncancer hazard.

No carcinogenic COC was identified for the surface water this site; therefore, the excess lifetime cancer risk associated with ingestion of soil or sediment cannot be quantified.

2.4.2.3 Drainage Pathway from POL Tanks (SS07).

Soils and Sediments. No COC was selected for the soil at The Drainage Pathway from POL Tanks site (Table 2-3). This does not indicate that exposure to chemicals in the soil at this site is without health risk; however, the concentrations measured were below the concentrations considered acceptable under Region X guidance (EPA 1991a) or federal ARARs.

Surface Water. The noncancer hazard associated with the ingestion of surface water at the site by a hypothetical native northern adult or by a DEW Line worker is 0.4, based on the maximum concentrations of the COCs (Tables 2-10 and A-7). DRPH, GRPH, tetrachloroethene, 1,1-dichloroethene, and cis-1,2-dichloroethene account for the quantifiable noncancer hazard for these receptor/pathway combinations. DRPH and cis-1,2-dichloroethene together account for more than 90 percent of the noncancer hazard.

The excess lifetime cancer risk associated with the ingestion of surface water at this site by native northern adults is 4×10^{-5} , and by a DEW Line worker is 7×10^{-6} , based on the maximum concentrations of the COCs (Table A-8). The presence of GRPH, benzene, tetrachloroethene, trichloroethene, and 1,1-dichloroethene accounts for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

2.4.2.4 Crushed Drum Area (SS08).

Soils and Sediments. The noncancer hazard associated with the ingestion of soil at the Crushed Drum Area by an hypothetical native northern adult/child is 0.3, and by a DEW Line worker is 0.01, based on the maximum concentrations of the COCs (Tables 2-10 and A-9). The presence of DPRH and GRPH accounts entirely for the quantifiable noncancer hazard for these receptor/pathway combinations.

The excess lifetime cancer risk associated with the ingestion of soil or sediment at this site by an hypothetical native northern adult/child is 7×10^{-7} , and by a DEW Line worker is 3×10^{-8} , based on the maximum concentrations of the COCs (Table A-10). The presence of GRPH and

benzene accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

Surface Water. The noncancer hazard associated with the ingestion of surface water at the Crushed Drum Area by a hypothetical native northern adult or by a DEW Line worker is 2, based on the maximum concentrations of the COCs (Tables 2-10 and A-11). Manganese and barium account for the quantifiable noncancer hazard for these receptor/pathway combinations. Manganese accounts for 99 percent of the noncancer hazard.

The excess lifetime cancer risk associated with the ingestion of surface water at this site by native northern adults is 5×10^{-6} , and by DEW Line workers is 9×10^{-7} , based on the maximum concentration of the COC (Table A-12). The presence of benzene accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

2.5 RISK CHARACTERIZATION UNCERTAINTY

Several sources of uncertainty affect the estimates of excess lifetime cancer risk and noncancer hazard as presented in this risk assessment. The sources are generally associated with:

- Sampling and analysis of soil, sediment, and surface water;
- Assigning the source of contamination;
- Exposure assumptions, including estimates of exposure point concentrations;
- Evaluation of the toxicity of the COCs; and
- Methods and assumptions used to characterize the cancer risk and noncancer hazard.

Uncertainties associated with sampling and analyses include the inherent variability (standard error) in the analyses, representativeness of the samples, sampling errors, and heterogeneity of the sample matrix. The quality assurance/quality control program used in conducting the sampling and analyses serves to reduce errors, but it can not eliminate all errors associated with sampling and analyses. There is some uncertainty in the selection of COCs with respect to sample quantitation limits for a given chemical. In some cases a chemical may have had detected values below the COC screening criteria as well as samples with quantitation limits greater than the screening criteria. In these cases it should be understood that only the samples with adequate quantitation limits are applicable to the screening process. Thus, the number of samples used to screen a chemical would be less than the total number of analyses for that chemical.

Simplifying assumptions were made about the environmental fate and transport of the site contamination, specifically, that no contaminant loss or transformation occurs. Thus, the data

chosen to represent exposure point concentrations in the sample-by-sample risk calculations are an additional source of potential error.

The depth at which a soil sample was collected was not considered in the risk characterization, so exposure to subsurface contamination was considered to be equally likely as exposure to surface contamination. This approach would tend to overestimate the true risk.

The estimation of exposure requires many assumptions to describe potential exposure situations. There are uncertainties regarding the likelihood of exposure, frequency of contact with contaminated media, the concentration of contaminants at exposure points, and the time period of exposure. These tend to simplify and approximate actual site conditions. In general, these assumptions are intended to be conservative and yield an overestimate of the true risk or hazard.

The toxicological database is also a source of uncertainty. The EPA has outlined some of the sources of uncertainty in the database (EPA 1986a,b, 1989a). These sources include extrapolation between exposure routes, from high to low doses, and from animals to humans; species, gender, age, and strain differences in uptake, metabolism, organ distribution, and target site susceptibility; and human population variability with respect to diet, environment, activity patterns, and cultural factors. The toxicity factors from IRIS and HEAST, which are used to estimate the toxicity of the COCs, are developed using a highly conservative methodology and probably tend to overestimate the potential hazards to humans.

Use of the provisional RfDs and SFs for DRPH, GRPH, and RRPH are an additional source of uncertainty in the toxicity assessment and risk characterization. Although the provisional RfDs represent the best available numerical estimate of toxicity, there is a significant amount of uncertainty associated with their use at the Point Lay installation. The RfDs and SFs are based on studies in mice and rats by the inhalation route of exposure; whereas, in this risk assessment, exposure of humans by the ingestion route only is being evaluated. Furthermore, in the absence of more thorough studies to compare the toxicity of DRPH, GRPH, and RRPH to the toxicity of known refinery streams, it is not clear how well the provisional values represent the toxicity of diesel, gasoline, and residual oils in humans.

In the risk characterization, the assumption was made that the total risk of developing cancer from exposure to site contaminants is the sum of the risk attributed to each individual contaminant. Likewise, the potential for the development of noncancer adverse effects is the sum of the HQs estimated for exposure to each individual contaminant. This approach does not account for the possibility that chemicals act synergistically or antagonistically but probably results in an overestimate of the true risk.

In addition to the more general sources of uncertainty associated with risk assessment methodology, there are site-specific sources of uncertainty. Primarily, these sources are associated with the lifestyle of the native northerners, the time spent on the sites that were investigated during the RI, and specific exposure assumptions (soil ingestion rate, exposure frequency, and exposure duration).

Residents of Point Lay use the airstrip at the radar installation and may also use the installation as an access route to traditional hunting and fishing locations (Brewster 1994). No studies have been conducted to measure the time they spend on contaminated sites at the installation. Some of the sites with levels of contamination that exceed regulatory benchmarks may not be accessed by this group. Therefore, the assumptions made regarding exposure frequency probably result in an overestimate of the true noncancer hazard and cancer risk.

Similarly, no studies have been conducted to measure the soil ingestion rate of potential receptors on the contaminated sites. Soil ingestion by potential future inhabitants at Point Lay radar installation (assuming a potential residential scenario) may be greater than the default rate of 100 mg/day for adults and 200 mg/day for children. Given the rugged, partially subsistence, lifestyle of this group, it is possible that they incidentally ingest soil at a higher rate than receptors of a similar age in the continental United States. The estimate of soil ingestion rate used in this risk assessment may over- or underestimate the true rate.

The maximum exposure duration assumed for native northerners, 55 years, is probably fairly accurate. The reasonable maximum exposure estimate for inhabitants of the continental United States is 30 years, however, native northerners are more likely to remain in their villages for a longer period. Although, the exposure duration of 55 years is an estimate, it is not expected to significantly over- or underestimate hazard or risk.

2.6 RISK ASSESSMENT SUMMARY AND CONCLUSIONS

The human health risks associated with exposure to contaminated media (soil, sediment, or surface water) at four sites at the Point Lay radar installation were evaluated in this risk assessment. The risk assessment was developed using a three step process:

- 1) The maximum concentrations of the chemicals detected in each medium (soil, sediment, or surface water) were compared to background concentrations, RBSLs, and ARARs. Chemicals present at concentrations that exceeded their background concentration and either an RBSL or an ARAR were retained as COCs for the risk assessment.
- 2) In the risk characterization, the noncancer HQ, the excess lifetime cancer risk, or both, were calculated based on the maximum concentration of each COC, and the associated toxicity values developed by EPA.
- 3) The HQs for each COC at a given site were summed, and the sum (called a hazard index) was compared to the regulatory benchmark for noncancer hazard: a hazard index of one. Sites where the hazard index exceeded one were considered to warrant either remediation or further discussion. Sites where the hazard index was less than one are considered to warrant no further action EPA 1991c).

The cancer risks for each carcinogenic COC at a given site were also summed and the sum (the total cancer risk) was compared to the regulatory benchmark for cancer risks: an excess lifetime cancer risk of 1×10^{-6} (one in one million). Sites where the total cancer risk exceeded 1×10^{-6} are considered to warrant either remediation or further discussion. Sites where the total cancer risk was less than 1×10^{-6} are considered to warrant no further action (EPA 1991c).

Table 2-11 contains a summary of the noncancer hazard and excess lifetime cancer risk for each site and medium at the Point Lay installation that exceeds a regulatory benchmark of 1.0 for noncancer hazard index or 1×10^{-6} for excess lifetime cancer risk.

No Further Action. The soil/sediment at the Deactivated Landfill (LF01), Garage (SS06), Drainage Pathway from POL Tanks (SS07), and the Crushed Drum Area (SS08) are considered to warrant no further action based on the findings of the human health risk assessment.

At the Deactivated Landfill, the noncancer hazard index for soil/sediment was below 0.01 for all receptor/exposure pathway combinations. No carcinogenic COC was identified for this medium at this site. At the Garage, the noncancer hazard index does not exceed 1 and the excess lifetime cancer risk was less than 1×10^{-6} for all receptor/exposure pathway combinations. At the Drainage Pathway from POL Tanks site, no COC was identified in soil/sediment based on a comparison of the maximum concentrations measured for each contaminant to RBSLs and ARARs (Section 2.1.5.3). The noncancer hazard index for the soil/sediment at the Crushed Drum Area was less than 0.3, and the excess lifetime cancer risk was less than 1×10^{-6} for all receptor/exposure pathway combinations.

Sites that Warrant Further Discussion. All four sites evaluated in the RI for Point Lay contain surface water contamination that warrants further discussion based on the noncancer hazard index or the excess lifetime cancer risk, or both. The noncancer hazard associated with exposure to surface water at the Deactivated Landfill is 3, at the Garage is 5, and at the Crushed Drum Area site, is 2. Manganese in surface water accounts for nearly all of the noncancer hazard at these locations. Furthermore, the excess lifetime cancer risk associated with exposure to surface water at the Deactivated Landfill, Drainage Pathway from POL Tanks, and the Crushed Drum Area sites is in the range of 1×10^{-6} to 1×10^{-4} . Tetrachloroethene, 1,1-dichloroethene, and benzene in the surface water account primarily for the excess lifetime cancer risk at these locations. These risks slightly exceed the regulatory benchmark of 1×10^{-6} or a one in one million cancer risk. EPA generally considers excess lifetime cancer risks that fall in the range of 1×10^{-6} to 1×10^{-4} not to warrant further investigation or remediation (EPA 1991c).

As noted in the exposure assessment section, however, surface water ingestion is a potentially complete pathway only under a future use scenario. The future use scenario would require retirement of the Point Lay installation, release of the installation for civilian use, and redevelopment as a residential area in which the surface water was used as the sole source of water for domestic purposes. Such a scenario is not likely because the village of Point Lay and its existing water supply system is more than adequate to support the native population in the area, and there are no population growth pressures to force the development of additional land

TABLE 2-11. SUMMARY OF NONCANCER HAZARDS AND CANCER RISKS

SITE	MEDIUM	COMMENT
Deactivated Landfill (LF01)	Surface Water*	Manganese in surface water yields noncancer hazard of 3. Tetrachloroethene in surface water yields cancer risk of 7×10^{-5} .
Garage (SS06)	Soil/Sediment	DRPH and RRPH in soil yield noncancer hazard of 1 for the native northern adult/child receptor group.
	Surface Water*	Manganese in surface water yields noncancer hazard of 5.
Drainage Pathway from POL Tanks (SS07)	Surface Water*	1,1-Dichloroethene in surface water yields cancer risk of 4×10^{-5} .
Crushed Drum Area (SS08)	Surface Water*	Manganese in surface water yields noncancer hazard of 2. Benzene in surface water yields cancer risk of 5×10^{-6} .

* Surface water noncancer hazards and cancer risks are based on potential future scenario (site water is used as sole-source drinking water supply for 180 days/year). Under current conditions hazards or risks associated with surface water at the sites are not significant.

or water resources. Therefore, based on the human health risk assessment, remediation of the surface water at the Point Lay installation does not appear to be necessary.

In conclusion, based on the human health risk assessment remediation of the four sites evaluated at Point Lay does not appear to be warranted. The estimates of noncancer hazard for each pathway/receptor combination were all below one under current conditions (site water is not used as a drinking water supply), and the estimates of cancer risk for each pathway/receptor combination were all within or below the range of cancer risk that EPA considers acceptable.

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3.0 ECOLOGICAL RISK ASSESSMENT

The objective of this ERA is to estimate potential impacts to aquatic and terrestrial plants and animals at the Point Lay DEW Line installation based on sampling and analyses conducted during the RI of the four sites located at the installation. The RI was completed during the summer of 1993 in conjunction with RIs at seven other radar installations.

Guidance documents used during preparation of this assessment include:

- Handbook to Support the IRP Statements of Work (U.S. Air Force 1991);
- Framework for ERA (EPA 1992a); and
- ERA Guidance for Superfund (EPA 1994a).

The approach used to assess potential ecological impacts is conceptually similar to that for human health risks. Potentially exposed populations (receptors) are identified, and information on exposure and toxicity are combined to derive estimates of risk. The ERA focuses, however, on potential impacts to a population of organisms rather than individual organisms (except in the case of endangered species where individuals are considered). Because ecosystems are composed of a variety of species, ERAs evaluate potential impacts to numerous species.

Ideally, ERAs should evaluate potential risks to communities and ecosystems, as well as to individual populations. Such ecosystem-wide assessments are very complex, however, and assessment methodologies have not been developed. In addition, dose-response data on community or ecosystem responses generally are lacking. Therefore, evaluations of potential impacts to communities or ecosystems are qualitative.

The degree to which potential ecological impacts can be characterized is highly dependent upon the data available to support such estimates. Such data include: information regarding contaminant release, transport and fate; characteristics of potential receptor population; and adequate supporting toxicity data for the chemicals evaluated.

This ERA is intended to be at a screening level, rather than a full scale investigation of the state of the ecosystem. No site-specific studies of the biota were undertaken. It is based on media sampling (i.e., surface water and soil/sediment samples) and is divided into six sections:

- Section 3.1 - Selection of Site Contaminants;
- Section 3.2 - Ecological Exposure Assessment;
- Section 3.3 - Ecological Toxicity Assessment;
- Section 3.4 - Risk Characterization for Ecological Receptors;
- Section 3.5 - Ecological Risk Assessment Uncertainty Analysis; and
- Section 3.6 - Summary of Ecological Risk.

3.1 SELECTION OF SITE CONTAMINANTS

A stressor in the environment is a chemical, physical, or biological action that can cause a negative impact on an ecosystem (EPA 1992a). Only chemical stressors identified as COCs are evaluated as part of this ERA. A review of the site data indicates that the chemical stressors are primarily petroleum products, solvents, and metals.

COCs are selected based on comparisons of the maximum detected concentration to background concentrations and action levels [Federal Ambient Water Quality Criteria (AWQC) for surface water (EPA 1994); ADEC Water Quality Standards (18 AAC 70.020[b]) January 1995; Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota (Suter and Mabrey 1994); ADEC determination of cleanup levels for petroleum contaminated soils (ADEC 1991); EPA sediment quality criteria (as estimated by Hull and Suter, 1994); and NOAA Sediment Effects Range (Low) (NOAA 1991)]. If no action levels were available, the maximum detected concentration of the chemical was compared to a toxicity value in the literature, derived from acute or chronic exposure tests. If the maximum concentration was above this level, the compound was considered a COC. Chemicals present onsite at concentrations in excess of background concentrations and action levels were evaluated for frequency of detection in onsite media. If a chemical was detected at a frequency of less than five percent, it was not considered representative of actual site conditions, and was eliminated from evaluation in the risk assessment. Further, an attempt was made to eliminate elements that were within the range of natural background levels. If the average concentration (exposure concentration) of a chemical was below the maximum background concentration (i.e., if the average falls within the range of background), and if the maximum detected concentration was less than twice the maximum background concentration (which is meant to approximate the 95 percent UCL of background concentrations), the chemical was not considered a COC. This criterium is used to account for outliers (i.e., hotspots) that are not representative of the distribution and concentration of chemicals to which ecological receptors may be exposed. Tables 3-1 and 3-2 present the data used in the screening process for surface water and soil/sediment. Only chemicals that were detected in at least one environmental sample are presented in these summary tables.

In summary, the decisions for selecting COCs were made using the following logic:

STEP ONE: Is the chemical detected above the maximum detected background concentration?

No: Not considered a COC.

Yes: Continue to step two.

STEP TWO: Is the chemical detected above the action level or toxicity value?

No: Not considered a COC.

Yes: Continue to step three.

TABLE 3-1. SUMMARY OF CHEMICALS OF CONCERN: SURFACE WATER

CHEMICALS OF CONCERN: POINT LAY INSTALLATION SURFACE WATER						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (µg/L)	BACKGROUND (µg/L) Organics = Point Lay Inorganics = Seven DEW Line Stations	ACTION LEVEL (µg/L)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (µg/L)	SELECTED AS COC
ORGANICS						
DRPH	181-961	<1,000	---	4/20	460	Yes
GRPH	189-223	<50	---	2/20	60	Yes
Benzene	15-21.5	<1	5,300 ^a	5/9	4.8	No
Toluene	7.3-30	<1	17,500 ^a	3/9	5.5	No
Ethylbenzene	5-12	<1	32,000 ^a	3/15	2.3	No
Xylenes (total)	12-82	<2	62,308 ^b	3/9	13	No
Dichlorodifluoromethane	33.5-58	<1	3,250 ^d	2/9	11	No
1,1-Dichloroethene	2.3	<1	2,800 ^b	1/9	0.70	No
cis-1,2-Dichloroethene	6.7-178	<1	9,538 ^b	3/8	22	No
trans-1,2-Dichloroethene	2.1-3.6	<1	9,538 ^b	2/9	1.0	No
Isopropylbenzene	1.2	<1	---	1/9	0.60	No*
p-Isopropyltoluene	1.3-1.7	<1	---	2/9	0.70	No*
Naphthalene	1.95-15	<1	620 ^b	3/4	4.9	No
n-Propylbenzene	1.3	<1	---	1/9	0.6	No*
Tetrachloroethene	2-109	<1	750 ^b	2/9	22	No
1,2,3-Trichlorobenzene	1.3	<1	50 ^a	1/4	0.8	No

* See text, Section 3.1.1.1.

^a EPA Ambient Water Quality Criteria (AWQC): Value presented is the Lowest Observed Effect Level (LOEL).

^b Based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994).

^c EPA Ambient Water Quality Criteria, Fresh water chronic criteria.

^d See text, Section 3.1.1.1, for discussion of derivation of action level.

TABLE 3-1. SUMMARY OF CHEMICALS OF CONCERN: SURFACE WATER (CONTINUED)

CHEMICALS OF CONCERN: POINT LAY INSTALLATION SURFACE WATER						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (µg/L)	BACKGROUND (µg/L) Organics = Point Lay Inorganics = Seven DEW Line Stations	ACTION LEVEL (µg/L)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (µg/L)	SELECTED AS COC
Trichloroethene	4-133	<1	21,900 ^a	2/15	9.6	No
Trichlorofluoromethane	3.0-3.8	<1	1,800 ^d	2/9	1.1	No
1,2,4-Trimethylbenzene	5.3-14	<1	---	3/9	3.5	No*
1,3,5-Trimethylbenzene	1.2-11	<1	---	4/9	2.6	No*
INORGANICS - based on total metals						
Aluminum	115-120	<100 - 350	87 ^c	2/3	95	No
Barium	205-360	<50 - 93	5,800 ^b	3/3	300	No
Calcium	52,000-82,000	4,500 - 88,000	116,000 ^b	3/3	64,000	No
Iron	5,600-32,500	180 - 2,800	1,000 ^c	3/3	15,000	Yes
Magnesium	25,000-29,000	<5,000 - 53,000	82,000 ^b	3/3	27,000	No
Manganese	610-1,700	<50 - 510	1,100 ^b	3/3	1,100	Yes
Potassium	5,400-8,050	<5,000	53,000 ^b	2/3	5,300	No
Sodium	28,000-47,000	8,400 - 410,000	680,000 ^b	3/3	40,000	No

* See text, Section 3.1.1.1.

a EPA Ambient Water Quality Criteria (AWQC): Value presented is the Lowest Observed Effect Level (LOEL).

b Based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994).

c EPA Ambient Water Quality Criteria, Fresh water chronic criteria.

d See text, Section 3.1.1.1, for discussion of derivation of action level.

TABLE 3-2. SUMMARY OF CHEMICALS OF CONCERN: SOILS AND SEDIMENTS

CHEMICALS OF CONCERN: POINT LAY INSTALLATION SEDIMENT AND SOIL						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (mg/kg)	BACKGROUND RANGE (mg/kg)	ACTION LEVEL (mg/kg)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (mg/kg)	SELECTED AS COC
ORGANICS						
DRPH	3.1 - 33,400	<50 - <100	500 ^b	25/48	2,900	Yes
GRPH	1.1 - 2,430	<3 - <4	100 ^b	20/48	150	Yes
RRPH	120 - 40,000	<100	2,000 ^b	6/33	1,500	Yes
Benzene	0.07 - 1.4	<0.02 - <0.04	0.052 ^c	4/37	0.13	Yes
Toluene	0.04 - 12.4	<0.02 - <0.04	0.786 ^c	8/37	0.70	Yes
Ethylbenzene	0.2 - 18.2	<0.02 - <0.04	4.36 ^c	10/37	1.7	Yes
Xylenes (total)	0.4 - 91.6	<0.04 - <0.08	1.21 ^c	10/37	5.7	Yes
n-Butylbenzene	3.7 - 11.6	<0.03 - <0.15	4.36 ^d	3/17	1.3	No*
sec-Butylbenzene	1.5 - 5	<0.03 - <0.15	4.36 ^d	3/17	0.58	No*
cis-1,2-Dichloroethene	0.02	<0.03 - <0.15	0.023 ^c	1/17	0.16	No
Isopropylbenzene	0.02 - 3.2	<0.03 - <0.15	4.36 ^d	4/17	0.42	No*
p-Isopropyltoluene	0.02 - 6	<0.03 - <0.15	4.36 ^d	6/17	0.64	No*
Naphthalene	0.03 - 26.2	<0.03 - <0.15	0.407 ^c	9/17	2.4	No*
n-Propylbenzene	0.02 - 7.9	<0.03 - <0.15	4.36 ^d	5/17	0.90	No*

* See text, Section 3.1.2.5.

Not available.

a NOAA 1991, sediment ER-L (Effects Range - low).

b ADEC, Interim Guidance for Non-UST Contaminated Soil Cleanup Levels, 17 July 1991.

c EPA Sediment Quality Criteria (estimated using equilibrium partitioning approach in Hull and Suter 1994). Note: action level for 1,2-dichloroethene is total (i.e., cis + trans).

d EPA Sediment Quality Criteria for Ethylbenzene (see text).

e Washington state sediment quality criteria (Hull and Suter 1994).

TABLE 3-2. SUMMARY OF CHEMICALS OF CONCERN: SOILS AND SEDIMENTS (CONTINUED)

CHEMICALS OF CONCERN: POINT LAY INSTALLATION SEDIMENT AND SOIL						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (mg/kg)	BACKGROUND RANGE (mg/kg)	ACTION LEVEL (mg/kg)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (mg/kg)	SELECTED AS COC
Tetrachloroethene	0.4 - 43	<0.03 - <0.15	2.73 ^c	4/28	1.6	Yes
Trichloroethane	0.5 - 1.0	<0.03 - <0.15	0.18 ^c	4/28	0.13	Yes
Trichloroethene	0.5 - 2.0	<0.03 - <0.15	1.07 ^c	2/28	0.11	Yes
1,2,4-Trimethylbenzene	0.06 - 48	<0.03 - <0.15	4.36 ^d	10/17	4.8	No*
1,3,5-Trimethylbenzene	0.05 - 19.6	<0.03 - <0.15	4.36 ^d	10/17	2.0	No*
Benzyl alcohol	0.68 - 1.4	<6.9 - <15	0.057 ^e	2/8	0.88	Yes
Fluoranthene	0.26	<6.9 - <15	6.2 ^c	1/4	0.70	No
2-Methylnaphthalene	1.3 - 2.4	<6.9 - <15	--	2/8	2.2	No*
Phenanthrene	0.7	<6.9 - <15	1.8 ^c	1/4	0.89	No
INORGANICS						
Aluminum	1,700 - 3,400	1,500 - 25,000	--	5/5	2,400	No
Barium	225 - 360	27 - 390	--	5/5	300	No
Calcium	1,200 - 4,450	360 - 59,000	--	5/5	2,100	No
Chromium	4.7 - 54	4.3 - 47	81 ^a	5/5	19	No
Copper	7.1 - 36	<2.7 - 45	34 ^a	3/5	16	No
Iron	14,000 - 44,500	5,400 - 35,000	--	5/5	25,000	No

* See text, Section 3.1.2.5.

Not available.

^a NOAA 1991, sediment ER-L (Effects Range - low).

^b ADEC, Interim Guidance for Non-UST Contaminated Soil Cleanup Levels, 17 July 1991.

^c EPA Sediment Quality Criteria (estimated using equilibrium partitioning approach in Hull and Suter 1994). Note: action level for 1,2-dichloroethene is total (i.e., cis + trans).

^d EPA Sediment Quality Criteria for Ethylbenzene (see text).

^e Washington state sediment quality criteria (Hull and Suter 1994).

TABLE 3-2. SUMMARY OF CHEMICALS OF CONCERN: SOILS AND SEDIMENTS (CONTINUED)

CHEMICALS OF CONCERN: POINT LAY INSTALLATION SEDIMENT AND SOIL						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (mg/kg)	BACKGROUND RANGE (mg/kg)	ACTION LEVEL (mg/kg)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (mg/kg)	SELECTED AS COC
Lead	10.4 - 195	<5.1 - 22	47 ^a	3/5	61	Yes
Magnesium	920 - 1,700	360 - 7,400	--	5/5	1,300	No
Manganese	160 - 270	25 - 290	--	5/5	210	No
Nickel	12 - 16	4.2 - 46	21 ^a	5/5	14	No
Potassium	243 - 590	<300 - 2,200	--	5/5	440	No
Selenium	17	<7.8 - <170	--	1/1	17*	Yes
Sodium	81 - 250	<160 - 680	--	5/5	120	No
Vanadium	11 - 19	6.3 - 59	--	5/5	14	No
Zinc	37 - 253	9.2 - 95	150 ^a	5/5	96	Yes

* See text, Section 3.1.2.5.

Not available.

NOAA 1991, sediment ER-L (Effects Range - low).

ADEC, Interim Guidance for Non-UST Contaminated Soil Cleanup Levels, 17 July 1991.

EPA Sediment Quality Criteria (estimated using equilibrium partitioning approach in Hull and Suter 1994). Note: action level for 1,2-dichloroethene is total (i.e., cis + trans).

EPA Sediment Quality Criteria for Ethylbenzene (see text).

Washington state sediment quality criteria (Hull and Suter 1994).

STEP THREE: Is the chemical detected at a frequency greater than five percent?

No: Not considered a COC.

Yes: Continue to step four.

STEP FOUR: Is the average concentration of the chemical greater than the maximum background concentration, and is the maximum detected concentration greater than two times the maximum background concentration?

No: Not considered a COC.

Yes: Chemical is classified as a COC.

All data for COCs were averaged (arithmetic mean) according to media. In the case of non-detects, averages were calculated using one-half of the quantitation limits. Replicate samples were averaged and treated as one sample. Total metal concentrations were used in determining COCs in surface water. This is a conservative approach because dissolved metal concentrations (the more bioavailable fraction) can be significantly lower than total metal concentrations. Section 3.1.1 describes surface water COCs, and Section 3.1.2 describes soil and sediment COCs. Any exceptions to the selection methodology are discussed in these sections.

3.1.1 Surface Water

Analytical results from the Deactivated Landfill (LF01), Garage (SS06), Drainage Pathway from POL Tanks (SS07), and Crushed Drum Area (SS08) were compiled and evaluated to identify the COCs. Surface water samples were collected and analyzed for contaminants likely to be present at the specific sites. Not all samples were analyzed for a "full suite" of parameters, but instead were analyzed for some combination of the following: DRPH, GRPH, RRPB, BTEX, HVOCs, volatile organic compounds (VOCs), semivolatile organic compounds (SVOCs), polychlorinated biphenyls (PCBs), pesticides, and metals. A summary of analytical results for all sampling conducted at the installation is presented in Appendix G. The following sections present the evaluation of the surface water data. Table 3-1 summarizes the evaluation and selection of COCs in surface water.

3.1.1.1 Organic Compounds. Twenty organic compounds were detected in surface water samples collected from the Point Lay installation: DRPH, GRPH, BTEX (total), dichlorodifluoromethane, 1,1-dichloroethene, cis-1,2-dichloroethene, trans-1,2-dichloroethene, isopropylbenzene, p-isopropyltoluene, naphthalene, n-propylbenzene, tetrachloroethene, 1,2,3-trichlorobenzene, trichloroethene, trichlorofluoromethane, 1,2,4-trimethylbenzene, and 1,3,5-trimethylbenzene. Trichloroethene and trichloroethane were detected using both the HVOC (8010 modified) and VOC (8260) analyses. In the case of these duplicate analyses, the average concentration used in the risk assessment was calculated from the analytical method that produced the highest average concentration. This is a conservative approach expected to be protective of ecological receptors. This section presents the evaluation of these compounds as COCs in surface water for the ERA.

DRPH were detected in 4 of 20 surface water samples. The range of detected concentrations was 181 to 961 $\mu\text{g/L}$. DRPH were not detected in background samples at a detection limit of 1,000 $\mu\text{g/L}$. Because of the high detection limit in background samples, detections of DRPH in site samples were not compared to the background detection limit. The average concentration of DRPH in site samples was 460 $\mu\text{g/L}$, and these levels are considered significant. Therefore, DRPH were selected as a COC.

GRPH were detected in 2 of 20 surface water samples at 189 and 223 $\mu\text{g/L}$. GRPH were not detected in background samples at a detection limit of 50 $\mu\text{g/L}$. The average concentration of GRPH was 60 $\mu\text{g/L}$. There is no established action level for GRPH, but because the concentrations exceeded the background level, GRPH were selected as a COC.

Benzene was detected in five of nine surface water samples at concentrations ranging from 15 to 21.5 $\mu\text{g/L}$. Benzene was not detected in background samples at a detection limit of 1 $\mu\text{g/L}$. Although the concentrations exceeded the background level, they were well below the action level of 5,300 $\mu\text{g/L}$. As a result, benzene was not considered a COC.

Toluene was detected in three of nine surface water samples at concentrations ranging from 7.3 to 30 $\mu\text{g/L}$. Toluene was not detected in background samples at a detection limit of 1 $\mu\text{g/L}$. Although the concentrations exceeded the background level, they were well below the action level of 17,500 $\mu\text{g/L}$. As a result, toluene was not considered a COC.

Ethylbenzene was detected in 3 of 15 surface water samples at concentrations ranging from 5 to 12 $\mu\text{g/L}$. Ethylbenzene was not detected in background samples at a detection limit of 1 $\mu\text{g/L}$. Although the concentrations exceeded the background level, they were well below the action level of 32,000 $\mu\text{g/L}$. As a result, ethylbenzene was not considered a COC.

Xylenes (total) were detected in three of nine surface water samples at concentrations ranging from 12 to 82 $\mu\text{g/L}$. Xylenes were not detected in background samples at a detection limit of 2 $\mu\text{g/L}$. Although the concentrations exceeded the background level, they were well below the action level of 62,308 $\mu\text{g/L}$. As a result, xylenes were not considered a COC.

Dichlorodifluoromethane was detected in two of nine surface water samples at concentrations of 33.5 and 58 $\mu\text{g/L}$. Dichlorodifluoromethane was not detected in background samples at a detection limit of 1 $\mu\text{g/L}$. A surface water action level was developed for dichlorodifluoromethane using aquatic toxicity information for related chloro-fluorocarbons (Dupont 1986, 1990) and QSAR equations for estimating toxicity to freshwater fish. The QSAR equation for neutral organics in Clements (1988) was used to estimate a 96-hour LC_{50} for freshwater fish of 65 mg/L. A $\log K_{ow}$ of 2.16, based on Sangster (1994), was used in the equation. A chronic No Observed Effect Level (NOEL), used as the action level, was estimated from the acute toxicity value using a UF of 20 (consistent with the methodology used to derive aquatic toxic reference values [TRVs]). The resulting action level is 3,250 $\mu\text{g/L}$. As a result, dichlorodifluoromethane was not selected as a COC because the concentrations were below the calculated action level.

1,1-Dichloroethene was detected in one of nine surface water samples at a concentration of 2.3 $\mu\text{g/L}$. 1,1-Dichloroethene was not detected in background samples at a detection limit of

1 µg/L. Although the detection exceeded the background level, it was below the action level of 2,800 µg/L. As a result, 1,1-dichloroethene was not considered a COC.

cis-1,2-Dichloroethene was detected in three of eight surface water samples at concentrations ranging from 6.7 to 178 µg/L. cis-1,2-Dichloroethene was not detected in background samples at a detection limit of 1 µg/L. Although the concentrations exceeded the background level, they were below the action level of 9,538 µg/L. As a result, cis-1,2-dichloroethene was not considered a COC.

trans-1,2-Dichloroethene was detected in two of nine surface water samples at concentrations of 2.1 and 3.6 µg/L. trans-1,2-Dichloroethene was not detected in background samples at a detection limit of 1 µg/L. Although the concentrations exceeded the background level, they were below the action level of 9,538 µg/L. As a result, trans-1,2-dichloroethene was not considered a COC.

Isopropylbenzene was detected in one of nine surface water samples at a concentration of 1.2 µg/L. Isopropylbenzene was not detected in background samples at a detection limit of 1 µg/L. Isopropylbenzene is classed as an alkylbenzene, a class of chemicals typically found in fuel oils (i.e., DRPH) (ATSDR 1993). There are no action levels for isopropylbenzene, and toxicity information is very limited. This chemical was, therefore, evaluated as one of the constituents of DRPH. Because DRPH are considered a COC (and detected at higher concentrations than isopropylbenzene), the evaluation of the toxicity of DRPH conservatively accounts for the incremental risk associated with low levels of alkylbenzenes.

p-Isopropyltoluene was detected in two of nine surface water samples at concentrations of 1.3 and 1.7 µg/L. p-Isopropyltoluene was not detected in background samples at a detection limit of 1 µg/L. p-Isopropyltoluene is classed as an alkylbenzene, a class of chemicals typically found in fuel oils (i.e., DRPH) (ATSDR 1993). There are no action levels for p-isopropyltoluene, and toxicity information is very limited. This chemical was, therefore, evaluated as one of the constituents of DRPH. Because DRPH are considered a COC (and detected at higher concentrations than p-isopropyltoluene), the evaluation of the toxicity of DRPH conservatively accounts for the incremental risk associated with low levels of alkylbenzenes.

Naphthalene was detected in three of four surface water samples at concentrations ranging from 1.95 to 15 µg/L. Naphthalene was not detected in background samples at a detection limit of 1 µg/L. Although the concentrations exceeded background levels, they were below the action level of 620 µg/L. As a result, naphthalene was not considered a COC.

n-Propylbenzene was detected in one of nine surface water samples at a concentration of 1.3 µg/L. n-Propylbenzene was not detected in background samples at a detection limit of 1 µg/L. n-Propylbenzene is classed as an alkylbenzene, a class of chemicals typically found in fuel oils (i.e., DRPH) (ATSDR 1993). There are no action levels for n-propylbenzene, and toxicity information is very limited. This chemical was, therefore, evaluated as one of the constituents of DRPH. Because DRPH are considered a COC (and detected at higher concentrations than n-propylbenzene), the evaluation of the toxicity of DRPH conservatively accounts for the incremental risk associated with low levels of alkylbenzenes.

Tetrachloroethene was detected in two of nine surface water samples at concentrations of 81 and 109 $\mu\text{g/L}$. Tetrachloroethene was not detected in background samples at a detection limit of 1 $\mu\text{g/L}$. Although the concentrations exceeded the background level, they were below the action level of 750 $\mu\text{g/L}$. As a result, tetrachloroethene was not considered a COC.

1,2,3-Trichlorobenzene was detected in one of four surface water samples at a concentration of 1.3 $\mu\text{g/L}$. 1,2,3-Trichlorobenzene was not detected in background samples at a detection limit of 1 $\mu\text{g/L}$. Although the concentration exceeded the background level, it was below the action level of 50 $\mu\text{g/L}$. As a result, 1,2,3-trichlorobenzene was not considered a COC.

Trichloroethene was detected in 2 of 15 surface water samples at concentrations of 4 and 133 $\mu\text{g/L}$. Trichloroethene was not detected in background samples at a detection limit of 1 $\mu\text{g/L}$. Although the concentrations exceeded the background level, they were below the action level of 21,900 $\mu\text{g/L}$. As a result, trichloroethene was not considered a COC.

Trichlorofluoromethane was detected in two of nine surface water samples at concentrations of 3.0 and 3.8 $\mu\text{g/L}$. Trichlorofluoromethane was not detected in background samples at a detection limit of 1 $\mu\text{g/L}$. A surface water action level was developed for trichlorofluoromethane using aquatic toxicity information for related chloro-fluorocarbons (Dupont 1986, 1990) and QSAR equations for estimating toxicity to freshwater fish. The QSAR equation for neutral organics in Clements (1988) was used to estimate a 96-hour LC_{50} for freshwater fish of 36 mg/L. A $\log K_{ow}$ of 2.53, based on Sangster (1994), was used in the equation. A chronic NOEL, used as the action level, was estimated from the acute toxicity value using a UF of 20 (consistent with the methodology used to derive aquatic TRVs). The resulting action level is 1,800 $\mu\text{g/L}$. As a result, trichlorofluoromethane was not selected as a COC because the concentrations were below the calculated action level.

1,2,4-Trimethylbenzene was detected in three of nine surface water samples at concentrations ranging from 5.3 to 14 $\mu\text{g/L}$. 1,2,4-Trimethylbenzene was not detected in background samples at a detection limit of 1 $\mu\text{g/L}$. 1,2,4-Trimethylbenzene is classed as an alkylbenzene, a class of chemicals typically found in fuel oils (i.e., DRPH) (ATSDR 1993). There are no action levels for 1,2,4-trimethylbenzene, and toxicity information is very limited. This chemical was, therefore, evaluated as one of the constituents of DRPH. Because DRPH are considered a COC (and detected at higher concentrations than 1,2,4-trimethylbenzene), the evaluation of the toxicity of DRPH conservatively accounts for the incremental risk associated with low levels of alkylbenzenes.

1,3,5-Trimethylbenzene was detected in four of nine surface water samples at concentrations ranging from 1.2 to 11 $\mu\text{g/L}$. 1,3,5-Trimethylbenzene was not detected in background samples at a detection limit of 1 $\mu\text{g/L}$. 1,3,5-Trimethylbenzene is classed as an alkylbenzene, a class of chemicals typically found in fuel oils (i.e., DRPH) (ATSDR 1993). There are no action levels for 1,3,5-trimethylbenzene, and toxicity information is very limited. This chemical was, therefore, evaluated as one of the constituents of DRPH. Because DRPH are considered a COC (and detected at higher concentrations than 1,3,5-trimethylbenzene), the evaluation of the toxicity of DRPH conservatively accounts for the incremental risk associated with low levels of alkylbenzenes.

3.1.1.2 Metals. Three surface water samples collected at the Point Lay installation were analyzed for metals. Eight inorganic analytes were detected: aluminum, barium, calcium, iron, magnesium, manganese, potassium, and sodium. This section presents the evaluation of these metals as COCs for the ERA. Analytes not detected in surface water samples were antimony, arsenic, beryllium, cadmium, chromium, cobalt, copper, lead, molybdenum, nickel, selenium, silver, thallium, vanadium, and zinc. The background ranges presented are representative of seven DEW Line installations. It is important to note that, in some cases, sample quantitation limits for certain metals were somewhat higher than ecologically relevant action levels. For example, in the case of copper, the sample quantitation limit was 50 µg/L and the current AWQC (based on a hardness value of 100 mg/L CaCO₃) is 12 µg/L. As a result, an ecological risk may exist for aquatic organisms from exposure to certain metals at their sample quantitation limits. These metals include aluminum, cadmium, chromium, copper, lead, and selenium. These issues will be addressed further in Section 3.5, Uncertainty Analysis. All concentrations of metals discussed below are results from total metal analyses.

Aluminum was detected in two of three surface water samples at concentrations of 115 and 120 µg/L. Background concentrations ranged from <100 to 350 µg/L. The EPA chronic AWQC for aluminum is 87 µg/L; this value was used as the action level. Aluminum was not present in surface water at concentrations in excess of the maximum background level, so it was not selected as a COC.

Barium was detected in all three surface water samples at concentrations ranging from 205 to 360 µg/L. Background concentrations of barium ranged from <50 to 93 µg/L. The action level for barium is 5,800 µg/L, based on data presented in Suter and Mabrey (1994). The value selected as the action level is the Lowest Chronic Value for All Organisms. Barium was not selected as a COC because it did not exceed the action level.

Calcium was detected in all three surface water samples at concentrations ranging from 52,000 to 82,000 µg/L. Background concentrations ranged from 4,500 to 88,000 µg/L. The action level is 116,000 µg/L based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994). Because calcium did not exceed background levels, it was not selected as a COC.

Iron was detected in all three surface water samples at concentrations ranging from 5,600 to 32,500 µg/L. Background concentrations ranged from 180 to 2,800 µg/L. Iron concentrations exceeded the background concentration in surface water and the 1,000 µg/L action level based on the AWQC, so iron was selected as a COC. The exposure concentration evaluated in this ERA is the average concentration of 15,000 µg/L.

Magnesium was detected in all three surface water samples at concentrations ranging from 25,000 to 29,000 µg/L. Background concentrations ranged from <5,000 to 53,000 µg/L. There are no AWQC for magnesium, but 82,000 µg/L may be used as a toxicological screening benchmark based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994). Magnesium was not selected as a COC because it is below background concentrations.

Manganese was detected in all three surface water samples at concentrations ranging from 610 to 1,700 µg/L. Background concentrations ranged from <50 to 510 µg/L. The action level for

manganese is 1,100 µg/L based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994). Because manganese was detected at concentrations exceeding background and the action level, it was selected as a COC. The average concentration used in the ERA was 1,100 µg/L.

Potassium was detected in two of three surface water samples at concentrations of 5,400 and 8,050 µg/L. Potassium was not detected in background samples at a detection limit of 5,000 µg/L. Based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994), 53,000 µg/L may be used as a toxicological screening benchmark. Potassium was not selected as a COC because it did not exceed the action level.

Sodium was detected in all three surface water samples at concentrations ranging from 28,000 to 47,000 µg/L, which are within the background range of 8,400 to 410,000 µg/L. The action level for sodium of 680,000 µg/L was based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994). Because onsite concentrations did not exceed the background levels, sodium was not selected as a COC.

3.1.2 Soils and Sediments

Soil/sediment sample analytical results from the four sites were compiled and evaluated to determine the COCs. Because most ecological receptors are principally exposed to surficial soils, only samples collected from 0 to 1.5 feet were considered in this ERA. Forty-eight of the fifty-five samples collected from the site are within this category. Of these, samples were collected and analyzed for contaminants likely to be present at the specific sites. Not all samples were analyzed for a "full suite" of parameters, but instead were analyzed for some combination of the following: DRPH, GRPH, RRPH, BTEX, HVOCs, VOCs, SVOCs, PCBs, pesticides, and metals. A summary of analytical results for all sampling conducted at the installation is presented in Appendix G. The following sections present the evaluation of the soil/sediment data for the four sites. Only compounds that were detected at the sites are discussed. Table 3-2 summarizes the evaluation and selection of COCs in soil and sediment.

3.1.2.1 Petroleum Hydrocarbons. Forty-eight soil/sediment samples were collected from the sites and selectively analyzed for a combination of DRPH and GRPH. Thirty-three soil/sediment samples were collected and analyzed for RRPH. A discussion of these petroleum hydrocarbon mixtures and their toxicity is presented in Section 3.3.1.

DRPH were detected in 25 of 48 soil/sediment samples at concentrations ranging from 3.1 to 33,400 mg/kg; these mixtures were not detected in background samples at detection limits of 50 to 100 mg/kg. The action level for DRPH in soils/sediments is 500 mg/kg. Because DRPH were detected at concentrations above the action level, they were selected as COCs. The exposure concentration used in the risk assessment was the average concentration of 2,886 mg/kg.

GRPH were detected in 20 of 48 soil/sediment samples at concentrations ranging from 1.1 to 2,430 mg/kg. The background concentrations ranged from <3 to <4 mg/kg. The action level for GRPH is 100 mg/kg. GRPH were detected at concentrations above the action level and were

considered to be a COC. GRPH were evaluated in the risk assessment as components of DRPH. Refer to Section 3.3.1 for a discussion of the toxicity of petroleum hydrocarbon mixtures.

RRPH were detected in 6 of 33 soil/sediment samples at concentrations ranging from 120 to 40,000 mg/kg. RRPH were not detected in background samples at detection limits of 100 mg/kg. The action level for RRPH is 2,000 mg/kg. RRPH were detected at concentrations above the action level, so they were considered a COC. RRPH were evaluated in the risk assessment as components of DRPH. Refer to Section 3.3.1 for a discussion of the toxicity of petroleum hydrocarbon mixtures.

3.1.2.2 Benzene, Toluene, Ethylbenzene, and Xylenes. Thirty-seven soil/sediment samples were collected from the four sites at the Point Lay installation and analyzed for BTEX using the 8020/8020 modified method. In addition, BTEX were detected using the VOC (8260) analysis. In the case of these duplicate analyses, the average concentration used in the risk assessment was calculated from the analytical method that produced the highest average concentration. This is a conservative approach expected to be protective of ecological receptors. The following paragraphs summarize the analytical results.

Benzene was detected in 4 of 37 soil/sediment samples at concentrations ranging from 0.07 to 1.4 mg/kg. Benzene was not detected in background samples at detection limits of 0.02 to 0.04 mg/kg. The estimated action level for benzene is 0.052 mg/kg, based on the equilibrium partitioning approach presented in Hull and Suter (1994). Because concentrations of benzene exceeded the action level, it was considered a COC. The average concentration used in the ERA was 0.13 mg/kg.

Toluene was detected in 8 of 37 soil/sediment samples at concentrations ranging from 0.04 to 12.4 mg/kg. Toluene was not detected in background samples at detection limits of 0.02 to 0.04 mg/kg. The estimated action level for toluene is 0.786 mg/kg, based on the equilibrium partitioning approach presented in Hull and Suter (1994). Because toluene concentrations exceeded the action level, it was considered a COC. The average concentration used in the ERA was 0.70 mg/kg.

Ethylbenzene was detected in 10 of 37 soil/sediment samples at concentrations ranging from 0.2 to 18.2 mg/kg. Ethylbenzene was not detected in background samples at detection limits of 0.02 to 0.04 mg/kg. The action level is 4.36 mg/kg based on the equilibrium partitioning approach presented in Hull and Suter (1994). Because concentrations exceeded the action level, ethylbenzene was selected as a COC. The average concentration used in the ERA was 1.7 mg/kg.

Xylenes (total) were detected in 10 of 37 samples. Xylenes concentrations ranged from 0.4 to 91.6 mg/kg. Xylenes were not detected in background samples at detection limits of 0.04 to 0.08 mg/kg. The action level is 1.21 mg/kg. Xylenes were considered a COC because concentrations were above the action level. The average concentration used in the ERA was 5.7 mg/kg.

3.1.2.3 Volatile Organic Compounds. Twelve HVOCs/VOCs were detected in soil/sediment samples (in addition to the BTEX discussed above) collected from the Point Lay installation. The compounds detected were n-butylbenzene, sec-butylbenzene, cis-1,2-dichloroethene, isopropylbenzene, p-isopropyltoluene, naphthalene, n-propylbenzene, tetrachloroethene, trichloroethane, trichloroethene, 1,2,4-trimethylbenzene, and 1,3,5-trimethylbenzene. None of these chemicals was detected in background samples (detection limits of 0.03 to 0.15 mg/kg). This section presents the evaluation of these compounds as COCs.

Toluene, ethylbenzene, sec-butylbenzene, isopropylbenzene, p-isopropyltoluene, n-propylbenzene, 1,2,4-trimethylbenzene, and 1,3,5-trimethylbenzene are alkylbenzenes, a class of chemical typically found in fuel oils (i.e., DRPH and GRPH) (ATSDR 1993). In order to evaluate the toxicity and potential for selection as COCs of chemicals without action levels (all the listed alkylbenzenes with the exception of toluene and ethylbenzene), comparisons to the known toxicities of toluene and ethylbenzene are made. The rationale and methodology for these comparisons follow.

Generally, as functional groups (e.g., methyl-, ethyl-, butyl-) are added to a benzene ring, the octanol water partition coefficient (K_{ow}), which controls the bioavailability of nonpolar organic compounds (Baudo et al. 1990), increases. In addition, as the number of functional groups increases, the toxicity of the chemical tends to increase. Compounds with a large K_{ow} , however, are more tightly sorbed to sediments, and thus are less bioavailable. This decrease in bioavailability offsets the increase in toxicity. Thus, the sediment screening values increase as functional groups are added and as K_{ow} increases. This trend may be observed in the sediment screening values: benzene, 0.052 mg/kg; toluene, 0.786 mg/kg; and ethylbenzene, 4.36 mg/kg (Hull and Suter 1994). The log K_{ow} s of benzene (2.13), toluene (2.69), and ethylbenzene (3.15) illustrate the direct relationship between the K_{ow} , toxicity, and the sediment screening values. Examining the K_{ow} of alkylbenzenes illustrates an important point; as K_{ow} increases, the expected toxicity in sediments declines. Table 3-3 presents the log K_{ow} s and estimated sediment quality criteria for benzene, toluene, and ethylbenzene.

n-Butylbenzene was detected in 3 of 17 soil/sediment samples at concentrations ranging from 3.7 to 11.6 mg/kg. There are no action levels available for this compound. n-Butylbenzene is an alkylbenzene, however, and may be compared to the known toxicity of ethylbenzene, another alkylbenzene. The log K_{ow} for n-butylbenzene is 4.2. Based on the information presented above, it appears that n-butylbenzene is less toxic than ethylbenzene. The action level for ethylbenzene is 4.36 mg/kg, and its average concentration was 1.7 mg/kg. The average concentration of n-butylbenzene was 1.3 mg/kg, below that of ethylbenzene, so the evaluation of ethylbenzene will serve to represent the risk estimate for n-butylbenzene. In addition, because DRPH are a COC (detected at higher concentrations and higher frequencies of detection) the evaluation of the toxicity of DRPH will conservatively account for the incremental risk associated with low levels of alkylbenzenes.

sec-Butylbenzene was detected in 3 of 17 soil/sediment samples at concentrations ranging from 1.5 to 5 mg/kg. There are no action levels available for this compound. sec-Butylbenzene is an alkylbenzene, however, and may be compared to the known toxicity of ethylbenzene, another alkylbenzene. The log K_{ow} for sec-butylbenzene is 4.24. Based on the information presented

TABLE 3-3. CHEMICAL/PHYSICAL PROPERTIES

Chemical	Log K _{ow} ^a	Action Level (based on Sediment Quality Criteria) (mg/kg) ^b
Benzene	2.13	0.052
Toluene	2.69	0.786
Ethylbenzene	3.15	4.36

^a EPA 1992b.

^b Hull and Suter 1994.

above, it appears that sec-butylbenzene is less toxic than ethylbenzene. The action level for ethylbenzene is 4.36 mg/kg, and its average concentration was 1.7 mg/kg. The average concentration of sec-butylbenzene was 0.58 mg/kg, below that of ethylbenzene, so the evaluation of ethylbenzene will serve to represent the risk estimate for sec-butylbenzene. In addition, because DRPH are a COC (detected at higher concentrations and higher frequencies of detection) the evaluation of the toxicity of DRPH will conservatively account for the incremental risk associated with low levels of alkylbenzenes.

cis-1,2-Dichloroethene was detected in 1 of 17 soil/sediment samples at a concentration of 0.02 mg/kg. The action level for total 1,2-dichloroethene (cis- and trans- isomers) is 0.023 mg/kg. Because the cis-1,2-dichloroethene concentration is below the action level it was not selected as a COC.

Isopropylbenzene was detected in 4 of 17 soil/sediment samples at concentrations ranging from 0.02 to 3.2 mg/kg. There are no action levels available for this compound. Isopropylbenzene is an alkylbenzene, however, and may be compared to the known toxicity of ethylbenzene, another alkylbenzene. The log K_{ow} for isopropylbenzene is 3.66. Based on the information presented above, it appears that isopropylbenzene is less toxic than ethylbenzene. The action level for ethylbenzene is 4.36 mg/kg, and its average concentration was 1.7 mg/kg. The average concentration of isopropylbenzene was 0.42 mg/kg, below that of ethylbenzene, so the evaluation of ethylbenzene will serve to represent the risk estimate for isopropylbenzene. In addition, because DRPH are a COC (detected at higher concentrations and higher frequencies of detection) it is assumed that the evaluation of the toxicity of DRPH will conservatively account for the incremental risk associated with low levels of alkylbenzenes.

p-Isopropyltoluene was detected in 6 of 17 soil/sediment samples at concentrations ranging from 0.02 to 6 mg/kg. There are no action levels available for this compound. p-Isopropyltoluene is an alkylbenzene, however, and may be compared to the known toxicity of ethylbenzene, another alkylbenzene. The log K_{ow} for p-isopropyltoluene is 4.10. Based on the information presented above, it appears that p-isopropyltoluene is less toxic than ethylbenzene.

The action level for ethylbenzene is 4.36 mg/kg, and its average concentration was 1.7 mg/kg. The average concentration of p-isopropyltoluene was 0.64 mg/kg, below that of ethylbenzene, so the evaluation of ethylbenzene will serve to represent the risk estimate for p-isopropyltoluene. In addition, because DRPH are a COC (detected at higher concentrations and higher frequencies of detection) it is assumed that the evaluation of the toxicity of DRPH will conservatively account for the incremental risk associated with low levels of alkylbenzenes.

Naphthalene was detected in 9 of 17 soil/sediment samples at concentrations ranging from 0.03 to 26.2 mg/kg. Naphthalenes are constituents of fuel oils (ATSDR 1993). As a result, naphthalene was not selected as a COC because the evaluation of the toxicity of DRPH (detected at higher concentrations than naphthalene and at a similar frequency of detection) will conservatively account for the incremental risk associated with naphthalene.

n-Propylbenzene was detected in 5 of 17 soil/sediment samples at concentrations ranging from 0.02 to 7.9 mg/kg. There are no action levels available for this compound. n-Propylbenzene is an alkylbenzene, however, and may be compared to the known toxicity of ethylbenzene, another alkylbenzene. The log K_{ow} for n-propylbenzene is 3.68. Based on the information presented above, it appears that n-propylbenzene is less toxic than ethylbenzene. The action level for ethylbenzene is 4.36 mg/kg, and its average concentration was 1.7 mg/kg. The average concentration of n-propylbenzene was 0.90 mg/kg, below that of ethylbenzene, so the evaluation of ethylbenzene will serve to represent the risk estimate for n-propylbenzene. In addition, because DRPH are a COC (detected at higher concentrations and higher frequencies of detection) it is assumed that the evaluation of the toxicity of DRPH will conservatively account for the incremental risk associated with low levels of alkylbenzenes.

Tetrachloroethene was detected in 4 of 28 soil/sediment samples at concentrations ranging from 0.4 to 43 mg/kg. The action level for this compound is 2.73 mg/kg (Hull and Suter 1994). Because the tetrachloroethene concentrations were above the action level, this compound was selected as a COC. The average concentration used in the ERA was 1.6 mg/kg.

Trichloroethane was detected in 4 of 28 soil/sediment samples at concentrations ranging from 0.5 to 1.0 mg/kg. The action level for this compound is 1.31 mg/kg (Hull and Suter 1994). Because the trichloroethane concentrations were above the action level, this compound was selected as a COC. The average concentration used in the ERA was 0.13 mg/kg.

Trichloroethene was detected in 2 of 28 soil/sediment samples at concentrations of 0.5 and 2.0 mg/kg. The action level for this compound is 0.18 mg/kg (Hull and Suter 1994). Because the trichloroethene concentrations were above the action level, this compound was selected as a COC. The average concentration used in the ERA was 0.11 mg/kg.

1,2,4-Trimethylbenzene was detected in 10 of 17 soil/sediment samples at concentrations ranging from 0.06 to 48 mg/kg. 1,2,4-Trimethylbenzene is classed as an alkylbenzene, which is a typical constituent of fuel oil (ATSDR 1993). As a result, 1,2,4-trimethylbenzene was not selected as a COC because the evaluation of the toxicity of DRPH (detected at higher concentrations than 1,2,4-trimethylbenzene and at a similar frequency of detection) will conservatively account for the incremental risk associated with 1,2,4-trimethylbenzene.

1,3,5-Trimethylbenzene was detected in 10 of 17 soil/sediment samples at concentrations ranging from 0.05 to 19.6 mg/kg. 1,3,5-Trimethylbenzene is classed as an alkylbenzene, which is a typical constituent of fuel oil (ATSDR 1993). As a result, 1,3,5-trimethylbenzene was not selected as a COC because the evaluation of the toxicity of DRPH (detected at higher concentrations than 1,3,5-trimethylbenzene and at a similar frequency of detection) will conservatively account for the incremental risk associated with 1,3,5-trimethylbenzene.

3.1.2.4 Semivolatile Organic Compounds. Four SVOCs were detected at the Point Lay installation (in addition to naphthalene, which is discussed above): benzyl alcohol, fluoranthene, 2-methylnaphthalene, and phenanthrene. These chemicals were not detected in background samples at detection limits ranging from 6.9 to 15 mg/kg. The detection limits for SVOCs in soil/sediment samples ranged from 0.22 to 2.28 mg/kg. Because incorporating non-detects (using one-half the detection limit) with unusually high detection limits skews the average (artificially inflating the average above the maximum detected concentration), samples with detection limits that were more than twice the maximum detection were not considered in the COC analysis. Soil sample 2S09 at the Crushed Drum Area (SS08) was not used in the analysis of SVOCs. The SVOCs detected in sample 2S09 are all components of petroleum products (ATSDR 1993) (with the exception of dibenzofuran, which was only detected [1.83 mg/kg] at the 2S09 location, but below the action level of 2.29 mg/kg [Hull and Suter 1994]). The risk assessment accounts for these chemicals through the analysis of DRPH. In addition, sample 2S09 was taken beneath the module train building and is not likely to be in an exposure pathway for ecological receptors.

Benzyl Alcohol was detected in two of eight soil/sediment samples at concentrations of 0.68 and 1.4 mg/kg. The action level for this compound is 0.057 mg/kg, based on the Washington State sediment standard presented in Hull and Suter (1994). Because the benzyl alcohol concentrations were above the action level, this compound was selected as a COC. The average concentration used in the ERA was 0.88 mg/kg.

Fluoranthene was detected in one of four soil/sediment samples at 0.26 mg/kg. The action level for fluoranthene is 6.2 mg/kg, based on EPA Sediment Quality Criteria (Hull and Suter 1994). Because the fluoranthene concentration did not exceed the action level, it was not selected as a COC.

2-Methylnaphthalene was detected in two of eight soil/sediment samples at concentrations of 1.3 and 2.4 mg/kg. There are no action levels available for this compound. Naphthalenes are constituents of fuel oils (ATSDR 1993). As a result, the evaluation of the toxicity of DRPH and GRPH (detected both at higher concentrations than 2-methylnaphthalene and at a higher frequency of detection) will conservatively account for the incremental risk associated with 2-methylnaphthalene (see Section 3.3.1).

Phenanthrene was detected in one of four soil/sediment samples at a concentration of 0.7 mg/kg. The action level for this phenanthrene is 1.8 mg/kg, based on EPA Sediment Quality Criteria (Hull and Suter 1994). Because the phenanthrene concentration was below the action level, this compound was not selected as a COC.

3.1.2.5 Metals. Fifteen inorganic analytes were detected in five soil/sediment samples collected from the Point Lay installation. The metals detected were aluminum, barium, calcium, chromium, copper, iron, lead, magnesium, manganese, nickel, potassium, sodium, selenium, vanadium, and zinc. This section presents the evaluation of these metals as COCs in the ERA.

Aluminum was detected in all five soil/sediment samples. Concentrations ranged from 1,700 to 3,400 mg/kg. Background concentrations ranged from 1,500 to 25,000 mg/kg. Aluminum concentrations did not exceed background concentrations, so it was not selected as a COC.

Barium was detected in all five soil/sediment samples at concentrations ranging from 225 to 360 mg/kg. The background concentrations of barium ranged from 27 to 390 mg/kg. There is no action level for barium. Barium concentrations did not exceed background concentrations, so it was not selected as a COC.

Calcium was detected in all five soil/sediment samples. Concentrations ranged from 1,200 to 4,450 mg/kg. Background concentrations ranged from 360 to 59,000 mg/kg. There is no action level for calcium. Calcium concentrations did not exceed background concentrations, so it was not selected as a COC.

Chromium was detected in all five soil/sediment samples. Concentrations ranged from 4.7 to 54 mg/kg. The maximum background concentration is 47 mg/kg. The action level for chromium is 81 mg/kg. The detected concentrations did not exceed the action level, so chromium was not selected as a COC.

Copper was detected in three of five soil/sediment samples. Detected concentrations ranged from 7.1 to 36 mg/kg. Background concentrations ranged from <2.7 to 45 mg/kg. The action level for copper is 34 mg/kg. The detected concentrations did not exceed the background concentrations, so copper was not selected as a COC.

Iron was detected in all five soil/sediment samples. Concentrations ranged from 14,000 to 44,500 mg/kg. The background concentrations ranged from 5,400 to 35,000 mg/kg. The average concentration of iron was 25,000 mg/kg, which is less than the maximum background concentration of 35,000 mg/kg, and the maximum detected concentration of 44,500 mg/kg is less than twice the maximum background concentration of 35,000 mg/kg. As a result, iron was not selected as a COC.

Lead was detected in three of five soil/sediment samples at concentrations ranging from 10.4 to 195 mg/kg. The maximum background concentration for lead is 22 mg/kg. The action level for lead is 47 mg/kg. Because the detected concentrations of lead exceeded the background and action level, it was selected as a COC. The average concentration used in the ERA was 61 mg/kg.

Magnesium was detected in all five soil/sediment samples. Concentrations ranged from 920 to 1,700 mg/kg. The background concentrations for magnesium ranged from 360 to 7,400 mg/kg. There is no action level for magnesium. Because magnesium was not detected above background concentrations, it was not selected as a COC.

Manganese was detected in all five soil/sediment samples. Concentrations ranged from 160 to 270 mg/kg. The background concentrations for manganese ranged from 25 to 290 mg/kg. There are no action levels for manganese. Because concentrations did not exceed background concentrations, manganese was not selected as a COC.

Nickel was detected in all five soil/sediment samples ranging in concentration from 12 to 16 mg/kg. The background concentrations ranged from 4.2 to 46 mg/kg. The action level for nickel is 21 mg/kg. Nickel was not selected as a COC because its concentrations did not exceed the background levels.

Potassium was detected in all five soil/sediment samples at concentrations ranging from 243 to 590 mg/kg. The background concentrations ranged from <300 to 2,200 mg/kg. There is no action level for potassium. Potassium was not selected as a COC because its concentrations were below background concentrations.

Selenium was detected in one soil/sediment sample at a concentration of 17 mg/kg. Selenium was not detected in background samples at detection limits of 7.8 to 170 mg/kg. There is no action level for selenium. Selenium was selected as a COC because its concentration was above the minimum background detection limit. The concentration used in the ERA was 17 mg/kg.

Sodium was detected in all five soil/sediment samples. Concentrations detected ranged from 81 to 250 mg/kg, which did not exceed the maximum background concentration of 680 mg/kg. There is no action level for sodium. Because sodium concentrations did not exceed the maximum background concentration, it was not selected as a COC.

Vanadium was detected in all five soil/sediment samples ranging in concentration from 11 to 19 mg/kg. The background concentrations ranged from 6.3 to 59 mg/kg. There is no action level for vanadium. Vanadium was not selected as a COC because its concentrations were below background concentrations.

Zinc was detected in all five soil/sediment samples at concentrations ranging from 37 to 253 mg/kg. The background concentrations for zinc ranged from 9.2 to 95 mg/kg. The action level for zinc is 150 mg/kg. Because the zinc concentrations are above the background and action level, zinc was selected as a COC. The average concentration used in the ERA was 96 mg/kg.

3.2 ECOLOGICAL EXPOSURE ASSESSMENT

The vegetation of the Arctic Coastal Plain and the ecosystems it characterizes have developed primarily as a result of the low relief and harsh environment. The growing season is short, typically extending from June through mid-September. Winters are long, cold, dry, and dark. Air temperatures that average below freezing for most of the year result in a permafrost layer that begins near the surface and reaches to depths as great as 610 meters. Seasonal thawing results in an active layer between ground surface and 3.7 meters below the surface (Hart Crowser 1987).

The impervious permafrost layer prevents percolation and infiltration of water below the active layer, and the generally flat terrain provides poor drainage. As a result, the ecosystems of the Arctic Coastal Plain are often defined not only by their plant associations but also by the degree of water found in and on them. Hart Crowser (1987) describes five major ecosystems for the classification of tundra and Arctic Coastal Plain communities:

- Marine zones: these include lagoons, estuaries, barrier islands, strands, and beaches. The abundance of vegetation along the marine coastal zone is inversely related to the amount of beach scouring by waves and ice. Mainland beaches support a variety of vegetation including sedges, grasses, and forbs.
- Wet sedge meadows: an association of meadows, ponds, and lakes also known as "wet tundra". This system, with its associated wetlands, is dominant in the area extending west from the Colville River to the Chukchi Sea (this area includes the Point Lonely, Point Barrow, Wainwright, Point Lay, and Cape Lisburne installations). Differences in vegetation within this ecosystem are related to moisture and microrelief.
- Tussock tundra: "moist tundra" consisting primarily of areas dominated by tussock-forming cottongrass. This system covers significant portions of the Arctic Coastal Plain.
- Riverine systems and floodplains: including riparian shrubland on recent and old alluvium. Being better drained than surrounding lands, the riparian environment supports a distinctive "shrub thicket" vegetation.
- Alpine tundra: including rocky upland areas of sparse mat-forming or fell-field vegetation.

The species associated with each ecosystem at the Point Lay DEW Line installation have the potential to be exposed to COCs if exposure pathways are complete. If pathways are complete, the representative species selected are considered receptors. Figure 3-1, Section 3.2.3, presents a schematic model of the potential exposure pathways.

The Ecological Exposure Assessment segment of the risk assessment contains the following: the most common species found at the DEW Line installations in Section 3.2.1, species of the Arctic Coastal Plain, the representative species and the rationale used for their selection in Section 3.2.2, a discussion of the exposure pathways in Section 3.2.3, and a review of the habitat suitability for representative species in Section 3.2.4. Sections 3.2.5, 3.2.6, and 3.2.7 provide the methodology of the exposure assessment for representative plants, aquatic species, and birds and mammals, respectively. Life history tables, which provide species-specific information for use in the exposure assessment, are included in Section 3.2.7.

3.2.1 Species of the Arctic Coastal Plain

The representative species used in the ERA for the Point Lay installation were selected from species characteristic of the DEW Line installations along the Arctic Coastal Plain and are detailed in Sections 3.2.2.1 through 3.2.2.5.

The Point Lay installation is located along the northwestern boundary of the Arctic Coastal Plain. Hart Crowser (1987) and Woodward-Clyde (1993) have listed the species likely to occur along the coastal plain based on site-specific studies and a review of the literature. The marine zone, wet sedge meadows, tussock tundra, and riverine/riparian are the primary ecosystems found at the Point Lay installation. Alpine tundra is minimal at the site and is not evaluated further. Site-specific surveys of the ecosystems associated with the DEW Line installations have not been conducted for this risk assessment; however, a study investigating the abundance and distribution of Steller's and spectacled eiders was used (Alaska Biological Research 1994).

3.2.1.1 Plants. Plants commonly associated with the marine zone are sedges, grasses, and forbs. *Carex subspathacea* and *C. aquatilis* are dominant plants in the coastal wetlands.

The wet sedge meadow (also known as "wet tundra") is characterized by a variety of sedges and grasses. Typical species include cottongrass, *Eriophorum* spp.; tundra grass, *Dupontia fischeri*; and mosses, *Sphagnum* spp. Marsh marigold, *Caltha palustris*; and horsetail, *Equisetum* spp. may be found in wetter areas (Hart Crowser 1987).

The tussock tundra (or moist tundra) is drier than the wet sedge meadow/wet tundra association. Tussock-forming cottongrass is the dominant plant species. Grasses, sedges, dwarf shrubs, mosses, and lichens are scattered throughout the tussock complex. These species include willows, *Salix* spp.; Labrador tea, *Ledum palustre*; blueberry and lingonberry, *Vaccinium* spp.; and lousewort, *Pedicularis* spp. (NPRA Task Force 1978; Bergman et al. 1977).

Riverine/riparian systems are composed of a diversity of habitat types and species. The dominant plants are low-growing shrubs with a scattered understory of grasses and herbs. Larkspur, *Delphinium brachycentrum*; cinquefoil, *Potentilla* spp.; bearberry, *Arctostaphylos* spp.; and wormwood, *Artemisia arctica* are common species (NPRA Task Force 1978; Bergman et al. 1977).

3.2.1.2 Aquatic Organisms. Sixty-six species of fish inhabiting marine, estuarine, and freshwater systems have been identified in the arctic region (Hart Crowser 1987). Marine species inhabiting the nearshore and offshore waters include boreal smelt, *Osmerus eperlanus*; Pacific herring, *Clupea harengus*; arctic cod, *Boreogadus saida*; and fourhorn sculpin, *Myoxocephalus quadricornis*. Anadromous species using arctic rivers for spawning include the arctic cisco, *Coregonus autumnalis*; arctic char, *Salvelinus alpinus*; and occasional pink and chum salmon, *Oncorhynchus* spp. Lack of overwintering habitat is a significant limiting condition for both anadromous and freshwater fish of the arctic region. The principal freshwater fish found in the region are grayling, *Thymallus arcticus*; lake trout, *Salvelinus namaycush*; burbot, *Lota lota*; and nine-spined stickleback, *Pungitius pungitius* (Hart Crowser 1987).

Invertebrates that may be present in the waters and wet habitats of the Arctic Coastal Plain are well represented by the crustaceans (i.e., copepods, isopods, amphipods, and decapods).

3.2.1.3 Birds. There are approximately 180 species of birds seasonally associated with the habitats of the Arctic Coastal Plain. Of these, many are shorebirds and waterfowl using migratory corridors that pass through the Point Lay area. Bird use of the coastal plain is highly seasonal and associated with typical avian breeding and migration cycles. Shoreline habitats are used significantly in association with molting, pre-migratory staging, and post breeding movement. These habitats are considered critical by the U.S. Fish and Wildlife Service (USFWS 1982). Principal species include glaucous gull, *Larus hyperboreus*; red phalarope, *Phalaropus fulicaria*; dunlin, *Calidris alpina*; loons, *Gavia* spp.; sandpipers, *Calidris* spp.; eiders, *Somateria* spp.; and geese, *Branta* spp. and *Chen* sp. Among the migratory passerine species using the coastal habitats are the Savannah sparrow, *Passerculus sandwichensis*; common and hoary redpolls, *Carduelis* spp.; snow bunting, *Plectrophenax nivalis*; and Lapland longspur, *Calcarius lapponicus* (Woodward-Clyde 1993).

3.2.1.4 Mammals. The mammalian fauna of the Arctic Coastal Plain and adjacent waters is relatively simple compared to fauna at lower latitudes. A review of species lists indicates a total of 38 species that commonly occur in the arctic; 11 of these are marine mammals (Hart Crowser 1987). A sampling of the terrestrial mammals geographically associated with the DEW Line stations, including Point Lay, consists of brown lemming, *Lemmus trimucronatus*; masked shrew, *Sorex cinereus*; arctic fox, *Alopex lagopus*; red fox, *Vulpes vulpes*; weasels, *Mustela* spp.; tundra vole, *Microtus oeconomus*; caribou, *Rangifer tarandus*; and grizzly bear, *Ursus arctos* (Hart Crowser 1987; Woodward-Clyde 1993).

Marine mammals of the arctic coast include polar bear, *Ursus maritimus*; walrus, *Odobenus rosmarus*; six species of whales; and five species of seals. The most common of the whale and seal species are beluga whale, *Delphinapterus leucas*; bowhead whale, *Balaena mysticetus*; gray whale, *Eschrichtius robustus*; ringed seal, *Phoca hispida*; and bearded seal, *Erignathus barbatus* (Hensel et al. 1984).

3.2.1.5 Threatened and Endangered Species. Species of the Arctic Coastal Plain and nearby waters that are protected by federal and state designations include bowhead whale (endangered); fin whale, *Balaenoptera physalus* (endangered); sei whale, *Balaenoptera borealis* (endangered); and humpback whale, *Megaptera novaengliae* (endangered). The gray whale was delisted by the National Marine Fisheries Service on 16 June 1994. Avian species include the spectacled eider, *Somateria fischeri* (threatened) and Steller's eider, *Polysticta stelleri* (candidate for listing). Based on the latest federal and state lists of threatened and endangered plant species (June 1995), no plant species at the DEW Line installations are currently listed as threatened or endangered.

3.2.2 Representative Species

It is impractical to evaluate all of these potential receptors individually because of the great diversity of plants and animals at a given site. Thus, for ERAs, a set of "representative species" is selected for further evaluation. The representative species are selected primarily on the

species' likelihood of exposure given their preferred habitat, feeding habits, and distribution of contaminants. Potential exposure pathways are shown in Figure 3-1 and discussed in Section 3.2.3. The abundance of a species relative to the areal extent of the sites is also considered.

The representative species encompass a range of ecological niches in order to achieve the best characterization of the ecosystems being examined. In addition, species are selected, in part, as a result of the availability of toxicity, exposure, and life history information. Species that may be sensitive to environmental impacts, such as endangered or threatened species, are also evaluated. Any endangered or threatened species discussed in the ERA are not considered representative of the Arctic Coastal Plain or the Air Force arctic radar installations. These species are evaluated to provide information about whether they face potential risks from exposure to the COCs being evaluated in the ERA.

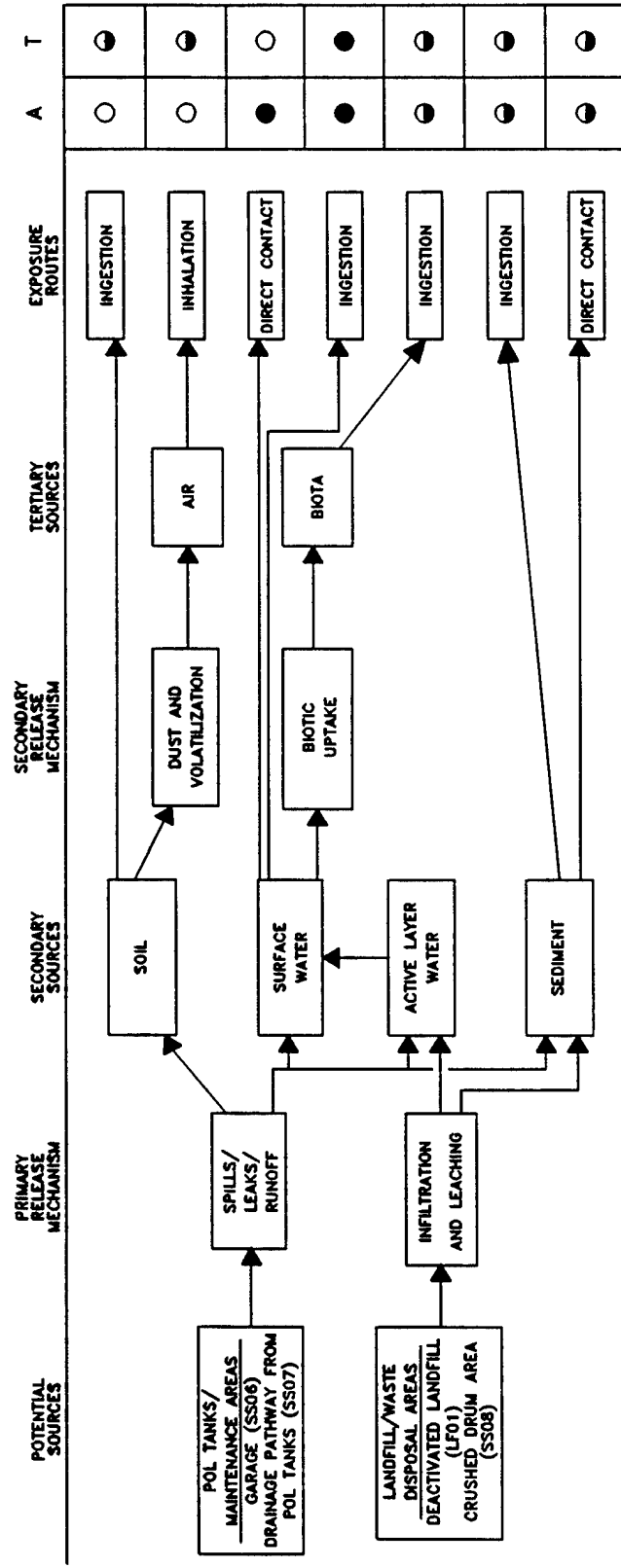
For the DEW Line installations, the groups of receptors evaluated include: plants, aquatic invertebrates, fishes, birds, and mammals. Potential risks to representative species are estimated by evaluating sampling data for the relevant exposure media (i.e., soil, sediments, and surface water). For plants, soil/sediment COC data are used to estimate potential uptake. For aquatic species, surface water COC concentrations are used to estimate exposure, for birds and mammals, exposures are estimated by evaluating their potential dietary intakes of COCs. No site-specific studies were conducted to determine exposure or toxicity levels at the installation.

The similarity of ecosystems at each of the coastal arctic DEW Line installations allows the use of the same set of representative species for all installations. It may be possible that a representative species inhabits the general area of an installation, but does not occur specifically on the installation property. When and if this situation occurs, it will be noted. The receptors selected as representative species for the Point Lay installation are listed in the paragraphs that discuss the representative groups (i.e., plants, aquatic organisms, birds, mammals, and threatened or endangered species). Table 3-4 presents the representative and sensitive species for the DEW Line installations, including endangered and threatened species that potentially may be exposed. The USFWS was consulted about the occurrence and selection of threatened and endangered species.

3.2.2.1 Representative Plants. Plants selected as representative species are sedges; willows; cottongrass; and various berry-bearing plants, *Vaccinium* spp. These species were selected because they are abundant on all the sites, are important links in the trophic structure of the ecosystems of the arctic, and represent a major percentage of the primary production along the coastal plain. In addition, the blueberry, huckleberry, and lingonberry, *Vaccinium* spp., are evaluated because of their roles as forage plants and subsistence species. All of these representative species are evaluated at the Point Lay installation.

3.2.2.2 Representative Aquatic Invertebrates and Fish. The invertebrates selected as representative species are *Daphnia* spp. (water fleas). The nine-spined stickleback is the representative fish species. The arctic char is not evaluated at the Point Lay installation because it is unlikely that the exposure pathways are complete. *Daphnia* spp. are abundant and represent a portion of the diet of the selected fish species (Johnson and Burns 1984; Wootton 1976), and toxicity information is readily available for them. The nine-spined stickleback is a freshwater

DRAWING No. LAY-FLO



**BULLEN POINT
RADAR INSTALLATION**

USAF 611th CES

FIGURE NO. 3-1

**ECOLOGICAL RISK
ASSESSMENT
POTENTIAL
EXPOSURE PATHWAYS**

- A AQUATIC RECEPTORS
- T TERRESTRIAL RECEPTORS
- COMPLETE EXPOSURE PATHWAY
- POTENTIALLY COMPLETE PATHWAY
- INCOMPLETE OR INSIGNIFICANT EXPOSURE PATHWAY

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TABLE 3-4. REPRESENTATIVE AND SENSITIVE SPECIES AT THE ARCTIC DEW LINE INSTALLATION SITES^a

COMMON NAME	GENUS AND SPECIES
PLANTS	
Sedge	<i>Carex</i> spp.
Cottongrass	<i>Eriophorum</i> spp.
Willow	<i>Salix</i> spp.
Berries	<i>Vaccinium</i> spp.
AQUATIC ORGANISMS	
Water fleas	<i>Daphnia</i> spp.
Nine-spined stickleback	<i>Pungitius pungitius</i>
Arctic char	<i>Salvelinus alpinus</i>
BIRDS	
Lapland longspur	<i>Calcarius lapponicus</i>
Brant	<i>Branta bernicla</i>
Glaucous gull	<i>Larus hyperboreus</i>
Pectoral sandpiper	<i>Calidris melanotos</i>
MAMMALS	
Brown lemming	<i>Lemmus trimucronatus</i>
Arctic fox	<i>Alopex lagopus</i>
Barren-ground caribou	<i>Rangifer tarandus</i>
ENDANGERED AND THREATENED SPECIES[*]	
Spectacled eider ^b	<i>Somateria fischeri</i>
Steller's eider ^c	<i>Polysticta stelleri</i>

* See Section 3.2.2.5 for information about endangered and threatened species.

^a Note: These representative species were selected for seven DEW Line installations (Barter Island, Bullen Point, Oliktok Point, Point Lonely, Point Barrow, Wainwright, and Point Lay) and the Cape Lisburne radar installation.

^b Threatened status.

^c Candidate for listing, see text for explanation.

species that also uses brackish habitats, nests in aquatic vegetation, and is prey for other fish and bird species (Wootton 1976). No marine mammals are evaluated in the Point Lay ERA because there are no complete pathways for COCs (at concentrations that are of concern) to reach potential marine receptors.

3.2.2.3 Representative Birds. The representative avian species are Lapland longspur; brant, *Branta bernicla*; glaucous gull; and pectoral sandpiper, *Calidris melanotos*. The Lapland longspur is a passerine belonging to a terrestrial feeding guild (including sandpipers, turnstones, and phalaropes) (Custer and Pitelka 1978). The longspur's diet of insects and seeds (Custer and Pitelka 1978) makes it an important link in the arctic trophic web. The brant nests and molts among the numerous ponds in the tussock tundra and grazes on sedges and cottongrass (Palmer 1976). It is considered to be an important subsistence resource. The glaucous gull breeds along the Arctic Coastal Plain and is a predatory scavenger that feeds on small mammals, young birds, carrion, and garbage (Farrand 1983). The pectoral sandpiper is an abundant shorebird that is primarily insectivorous and breeds on the Arctic Coastal Plain. The Lapland longspur, brant, glaucous gull, and pectoral sandpiper are in potential exposure pathways at the Point Lay installation and are evaluated in this ERA. All the avian species in this ERA are migratory, and as such, are protected under the Migratory Bird Treaty Act of 1978. This is reflected by the use of a protected species factor of 2 in the calculation of avian toxicity reference values.

3.2.2.4 Representative Mammals. The representative species of mammals are the brown lemming, arctic fox, and barren-ground caribou. The brown lemming is the predominant small mammal at all installations. The lemming consumes more vegetation than expected for an animal its size, due to its low assimilation efficiency, the low nutrient value of winter forage, and the high metabolic demands of the arctic environment (Chappell 1980). The arctic fox is selected as a representative species because it is ubiquitous along the coastal plain, and its carnivorous diet (mostly lemmings) places it near the top of the trophic structure in the arctic. Eberhardt et al. (1982) note that in fall and winter, and to a lesser extent in summer, the arctic fox frequently uses areas near development. This tendency may expose the fox to pathways of contamination. Additionally, the fox, a relatively common furbearer, can be an important subsistence resource. The caribou is selected as a representative species because it uses areas on, or near, a number of the radar installations during migration, calving, and post-calving. In addition, the caribou is a significant subsistence resource for local people along the Arctic Coastal Plain (USFWS 1982; Cuccarese et al. 1984; Hensel et al. 1984). The three mammal species discussed may be exposed to COCs at the Point Lay installation and are evaluated in this ERA.

3.2.2.5 Threatened and Endangered Species. The threatened and endangered species that potentially occur at the DEW Line installations are the spectacled eider and Steller's eider. The spectacled eider is federally listed as threatened, and Steller's eider is a candidate for listing as threatened. The U.S. Fish and Wildlife Service indicated that it was likely that Steller's eider would be listed as threatened sometime in 1995 (Ambrose 1994 pers. comm.), but a federal moratorium on additions to the threatened and endangered lists is in effect. Alaska Biological Research (1994) conducted surveys searching for spectacled and Steller's eiders on and near the DEW Line installations. The surveys report that spectacled eiders were observed near the Point Lay installation, while there is little chance that Steller's eiders will use the habitats near

Point Lay. As a result, spectacled eiders, but not Steller's eiders, will be evaluated in this ERA. Because of the ecological similarity of spectacled and Steller's eiders (i.e., relatively similar morphology, physiology, niche, and trophic status), this evaluation will serve as a surrogate indicator of risk to Steller's eiders in the event that the species is exposed to a COC pathway at Point Lay. The arctic peregrine falcon, *Falco peregrinus tundrius*, was delisted by the USFWS on 5 October 1994.

3.2.3 Exposure Pathways

This section discusses potential exposure pathways for ecological receptors. In addition, methods used to quantify exposures to selected species of plants, aquatic organisms, birds, and mammals are presented. Quantitative estimates of exposure will be compared with TRVs derived in Section 3.3 to estimate risks in the risk characterization section (Section 3.4).

Ecological receptors can be exposed to COCs through abiotic and biotic media. Potential exposure pathways for terrestrial and aquatic organisms are summarized in Figure 3-1. The following sections describe the potential exposure routes and a determination of pathways evaluated in the risk assessment.

Potential risks to representative species of plants from exposure to COCs in soil and water will be addressed. The most significant route of exposure for plants is direct contact with soil at the site, although a qualitative evaluation of the effects of COCs in surface water is presented in Section 3.4.1.

Aquatic organisms such as fish and invertebrates are primarily exposed through direct contact with surface water. Surface water is in direct contact with dermal surfaces as well as gills and other respiratory structures. Fish and invertebrates also may be exposed to COCs through ingestion of plant and animal items in the diet, direct contact with sediments, and incidental ingestion of sediments while foraging. Direct contact with surface water is the primary exposure route; therefore, these secondary routes (ingestion and direct contact) will not be evaluated for aquatic organisms.

Birds and mammals may be exposed to COCs through a variety of pathways including ingestion of surface water used for drinking, ingestion of plant and animal diet items, and incidental ingestion of surface soils and sediments while foraging. Wildlife species are not expected to be exposed to COCs via inhalation because the surface soils are well vegetated and moist during the growing season and frozen and/or snow covered the remainder of the year. Therefore, the inhalation pathway is not evaluated in the ERA.

Insufficient toxicity and exposure information is available for the representative species to allow quantification of exposures from dermal contact with soil or sediments; therefore, these pathways were not quantitatively evaluated. Because soils and sediments represent potential pathways, total exposures for the representative species could be underestimated. This represents one of the uncertainties in this risk assessment discussed in Section 3.5.

The soil and sediment samples at the Point Lay installation were taken at depths ranging from the surface to six feet deep. Only samples at depths from 0 to 1.5 feet were used in the ERA because the potential exposure pathways for the representative species are likely to be incomplete at depths greater than 1.5 feet. It is unlikely that any of the representative species would be exposed to soil or sediments much below 1 foot, although the brown lemming is reported to burrow up to 1 foot deep (Nowak 1991).

3.2.4 Habitat Suitability for Representative Species

In order to assess the representative species' degree of exposure to the COCs, the habitat suitability of each of the four sites was evaluated. The habitat suitability evaluation considered the representative aquatic, avian, and mammalian species at the Point Lay installation.

Human development and activities at the Point Lay installation have impacted the natural habitats available to the representative species. The effects of these impacts are uncertain; in some cases the activities probably deter wildlife use of the area and in other cases they may attract wildlife (e.g., arctic fox and gulls attracted to a landfill). Although these impacts may affect how and when representative species may use the habitats at Point Lay, the impacts are not subject to quantification; therefore, all the sites at the Point Lay installation are evaluated in this ERA. In some cases, the media-specific samples have been taken at locations that do not represent suitable habitat for all the representative species (e.g., under-building sample locations that are obviously not suitable for caribou, or surface water sample locations that are not suitable for fish species). This may result in an overestimate of exposure because sample data from all locations are used to calculate the average concentrations which, in turn, are used to estimate exposure for the representative species. These conditions will be noted in the risk characterization and uncertainty sections (Sections 3.4 and 3.5, respectively).

The ERA is being conducted for the entire Point Lay installation, but only a portion of the facility consists of potentially contaminated sites. The sites are estimated to total approximately 5.3 hectare (ha), or 13.2 acres. The areal extent of the potentially contaminated sites has been estimated using site maps. The spatial extent of the sites is considered when estimating the onsite dietary intake (IS) in Section 3.2.7.2. In general, based on professional judgement and onsite observations but not on site-specific surveys, the installation provides habitat less suitable than nearby areas because of the numerous roads, gravel pads, and overall development.

3.2.5 Exposure Assessment for Representative Species of Plants

The harsh environment of the Arctic Coastal Plain imposes many restrictions on plant life. The presence of permafrost limits infiltration and percolation of water, so the water table is often at or near the surface. Most plant species are perennial, with much of their biomass (50 to 98 percent) underground (Raven et al. 1986). The potential pathways of contamination for plants are through the soil/sediment and surface water.

Carex spp., *Salix* spp., and *Eriophorum* spp. all store food reserves in rhizomes. Mycorrhizal fungi play an important role in the transport and delivery of nutrients to the rhizomes and the roots of these species. This underground system probably developed in response to the harsh

above-ground arctic environment. Surface water contaminated with chemicals that are lighter than water (i.e., petroleum and its derivatives) does not present a greatly increased hazard to the below-ground portion of plants. This has been shown experimentally by exposing arctic coast vegetation to petroleum products (Walker et al. 1978). The experiments showed that sedges, willows, and cottongrass plants were not adversely affected by low to moderate amounts of petroleum (spill concentrations in the studies were up to 12 L/m²) in wet environments. Thus, soil/sediment will be considered the primary pathway of potential contamination for plants. The chemical concentration used in the risk characterization (Section 3.4) is the average concentration of the COC in the soil/sediments at the installation. A qualitative evaluation of the effect of potentially contaminated surface water on plants is presented in Section 3.4.1.

3.2.6 Exposure Assessment for Representative Aquatic Organisms

Organisms that dwell in an aquatic environment are exposed to chemicals contained in the water column. For this reason, the exposure assessment considers the concentrations in surface water to be the exposure concentrations to aquatic organisms. As described in Section 3.2.3, the primary exposure route for aquatic organisms is direct contact with surface water; therefore, the aquatic representative species are not evaluated for contact with, or ingestion, of sediments. The risk assessment compares the average concentration of the COCs found in surface waters to toxicity data for the representative aquatic species to calculate the risk estimate.

3.2.7 Exposure Estimates for Representative Bird and Mammal Species

Exposure estimates for representative species of mammals and birds (expressed as a unit of chemical ingested per unit of body weight) are based on their total exposure to COCs from diet, soils, and surface water using the following equation:

$$EE = [(FI \times CF) + (WI \times CW) + (SI \times CS \times ROA)] \times UCF \times IS / BW$$

where:

EE	=	estimated exposure (mg/kg-bw/day).
FI	=	food intake rate (g/day); rates are derived in the life history tables (Tables 3-6 through 3-10 and 3-11 through 3-14). Diets (both vegetable and animal components) are proportioned according to the diet composition information in the life history tables and are presented below.
CF	=	chemical concentration in food (mg/kg); based on concentrations for each group of food items.
WI	=	water intake rate (L/day); rates are derived in the life history tables.
CW	=	chemical concentration in water (μg/L); see Section 3.1 for calculations of concentrations.
SI	=	soil/sediment intake rate (g/day); based on a percentage of food intake.
CS	=	chemical concentration in soil/sediment (mg/kg); see Section 3.1 for calculations of concentrations.
ROA	=	relative oral availability; default to 1.0 (lack of information). This value assumes that the bioavailability of the chemical in the test medium is the same as for the medium onsite.

UCF	=	0.001; unit conversion factor used conversion of g to kg, μ g to mg, and L to ml, to ensure EE is reported in mg/kg-bw/day.
IS	=	fraction of dietary intake at potentially contaminated sites (by weight).
BW	=	body weight (kg)

In the case of species that have partial herbivorous dietary intakes, the CF x FI phrase in the equation is multiplied by the proportion of vegetation in the diet (these calculations are presented in Appendix C, Bioconcentration Factor Calculation and Appendix D, Concentration in Food Calculations). Those species and their respective proportions of vegetation are Lapland longspur, 0.25; brant, 0.90; glaucous gull, 0.10; pectoral sandpiper, 0.10; and spectacled eider, 0.25 (see the life history tables for references regarding the proportion of vegetation in the species' diets). The estimated exposure calculations for bird and mammal receptors are presented in Appendix E.

3.2.7.1 Potential Bioaccumulation of COCs in Representative Species. The potential risks from ingestion of COCs in dietary items are difficult to determine because of the complexity of the trophic web. Inputs to the exposure estimate equation include concentrations of contaminants in water and soil, ingestion rates for water, food, and soil, the relative use of the potentially contaminated sites compared to the representative species' normal range, and body weight. The food ingested, in the case of higher level consumers, may be from different levels of the trophic web. For example, a contaminant may be taken up by a plant that is consumed by a lemming, which is then eaten by an arctic fox. The amount of contaminant to which the fox is exposed is not readily quantified without supporting empirical data at each trophic level. Because the lack of data to assist in quantifying bioaccumulation, the risk assessment does not account for bioaccumulation in the animal portion of the trophic web. This uncertainty is tempered by the "hot spot" nature of the distribution of the COCs. It is possible that representative species may be exposed to these "hot spots" occasionally, but it is unlikely that their entire exposure will occur at these locations. Use of the average concentrations to estimate exposure may overestimate the potential exposure of representative species (this is discussed in more detail in the ERA Uncertainty Analysis, Section 3.5.1). Furthermore, the likelihood of predators repeatedly taking prey that were exposed to a COC "hot spot" is low. For example, the arctic fox ranges over such a wide area that any COCs to which the fox would be exposed via bioaccumulation would represent only a very small proportion of its overall exposure.

Further, most of the COCs in soils/sediments at the Point Lay installation are VOCs unlikely to bioaccumulate. For illustrative purposes, bioconcentration factors (BCFs) calculated for the organic COCs are presented in Table 3-5 (Veith et al. 1979 in Spacie and Hamelink 1985). Based on these low BCFs, the exposure estimates for organic chemicals do not include potential bioaccumulation of COCs in the animal portion of the trophic web. It is unlikely that the organic chemicals will bioaccumulate (based on the concentrations reported in the soil/sediment and water) such that the exposure estimates would exceed, or even approach, the TRVs.

The inorganic COCs at Point Lay are iron and manganese in surface water and lead and selenium in soil/sediment. The bioaccumulation of metals in the animal portion of the trophic web is not amenable to quantification without sample concentrations at each level of consumer.

TABLE 3-5. BIOCONCENTRATION FACTORS FOR SELECTED ORGANIC COMPOUNDS IN WATER

CHEMICAL	Log K _{ow}	BCF
DRPH	5.30	6,238
Naphthalene	3.36	211
1,2,4-Trimethylbenzene	3.78	439
Xylene	3.16	149

Note: BCF calculated from Log K_{ow} according to the following equation:

$$\text{Log BCF} = 0.76 \text{ Log K}_{ow} - 0.23 \text{ (Veith et al. 1979 in Spacie and Hamelink 1985)}$$

K_{ow} = octanol/water partition coefficient

The following qualitative discussions address the potential bioaccumulation of the inorganic COCs at the Point Lay installation.

Iron. Information about the bioaccumulation of iron is not available in the literature; because iron is an essential nutrient, it is likely that metabolic processes will prevent undue bioaccumulation.

Manganese. Manganese is considered an essential nutrient (ATSDR 1990). It is not likely that the concentrations of manganese found at the Point Lay installation would bioaccumulate because this mineral is regulated for metabolic use by the representative species.

Lead. Lead tends to accumulate in bone (Talmadge and Walton 1991), so ingestion of animal tissue would not contribute greatly to increased lead concentrations. Food chain biomagnification of lead is uncommon in terrestrial communities (Eisler 1988). Kraus (1989) showed that in environments high in lead, the concentration of lead in insects and the tissues of insectivorous birds was low. Thus, lead is not likely to bioaccumulate to a degree that could contribute to risk at the Point Lay installation.

Selenium. Selenium is an essential nutrient that may be toxic at levels not much greater than levels representing the required dose. Dietary requirements range from 0.1 to 0.3 parts per million (ppm) and toxic dietary levels are about 10 to 50 times greater (NAS 1980). Selenium was detected once, at a concentration of 17 mg/kg, in a soil sample (S06) at the Crushed Drum Area (SS08). Because selenium was detected at only one location, its contribution to dietary intake by the representative species is likely to be very low. Selenium is accumulated by certain "accumulator species" of plants, mainly species of *Astragalus*, *Xylorrhiza*, *Oonopsis*, and *Stanleya* spp. (Wilbur 1980), although it is unlikely that any of these species is present along the Arctic Coastal Plain. Wilbur (1980) also states that selenium has "virtually no tendency to accumulate in muscle or other organs of animals given an excess of selenium in the diet." Thus, given the

lack of plant "accumulator species" and the low likelihood of animal bioaccumulation, the biomagnification of selenium in the food chain at Point Lay does not pose a potential hazard.

Zinc. Zinc is an essential nutrient (Eisler 1993). Considering that zinc was detected above the action level at only one sample location (replicate samples SD04 and SD08 at the Deactivated Landfill, LF01), and that animal systems are conditioned to regulate essential minerals for metabolic use, it is unlikely that zinc would bioaccumulate to potentially hazardous levels.

3.2.7.2 Estimation of Percent Ingested Onsite. The size of the areas used by the representative species, and hence their potential exposure to COCs, varies greatly. Generally, a species' home range is used to characterize the size of the area it uses on a regular basis (disregarding migration and dispersal). When home range information for a species was not available, population density values have been converted to estimate the area used by the species. This information, combined with the extent of the potentially contaminated sites, can be used to estimate the percent of dietary intake that a species gets from the sites.

This estimate is referred to as the "percent of dietary intake at sites" (IS) value in the exposure estimate equation. The IS value is represented by the ratio of the total area of the sites (5.3 ha or 13.2 acres) to the reported home range size (or converted population density values) for the representative species. The representative species are most likely at Point Lay during, or directly after, the breeding season, when many species become territorial. These territories represent the area used by the species. The densities of the population may provide estimates of the size of the territories and are used as substitute values only when home range information is unavailable. This presents an added degree of uncertainty (see Section 3.5.3). If the home range (or converted population density value) is less than the total areal extent of the sites (5.3 ha), the maximum value for IS is 1.0 because it is possible that a species could meet all its dietary intake needs within the potentially contaminated areas. The IS values for the representative bird and mammal species are given below. Note that these are conservative estimates because the 5.3 ha size assumes the contaminated sites are the only areas used. Obviously, the species would use the suitable areas between the potentially contaminated sites, resulting in lower potential exposure to COCs than if the species restricted its location to contaminated sites only.

Birds. Lapland longspur. IS = 0.5; Derksen et al. (1981) report a breeding density of 38.6 birds/km². This corresponds to about 1 bird/2.6 ha. Potentially, the longspur could meet all of its dietary demands within the sites if they were contiguous. Nevertheless, an IS value of 0.5 is used because the longspur prefers drier upland habitat over the wetter areas where the majority of contaminant pathways occur.

Brant. IS = 0.27; density of breeding pairs reported by Derksen et al. (1981) is 5.0 birds/km². At this density of 1 brant/20 ha, the total extent of the potentially contaminated sites is about 27 percent of the area a brant might use.

Glaucous gull. IS = 0.04; the density for the glaucous gull is reported by Derksen et al. (1981) as 0.8 birds/km². This density, about 1 gull/125 ha, yields an IS value of 0.04, indicating that the potentially contaminated sites are about 4 percent of the area the glaucous gull uses.

Pectoral sandpiper. IS = 1.0; the density of the pectoral sandpiper along the Arctic Coastal Plain is reported by Derksen et al. (1981) as 22.4 birds/km². This density equates to one sandpiper/4.5 ha, and a corresponding IS value that defaults to the maximum of 1.0.

Spectacled eider. IS = 0.02; Derksen et al. (1981) report an average population density of 0.32 birds/km² for the spectacled eider. The resulting density of 1 bird/312.5 ha in 1981 is currently too high considering the decline in the species' population. The resulting IS value of 0.02 may result in an overestimation of potential exposure, but an overestimation may be considered acceptable in the evaluation of a sensitive species.

Mammals. Brown lemming. IS = 0.5; the lemming's home range is reported as 0.5 ha (Nowak 1991). It is possible that several lemmings may consume all their dietary needs within the bounds of a site. The lemming is not likely, however, to use the wetter sites (which constitute well over 50 percent of the total extent of the sites) where the majority of the contaminant pathways are located. The sites are mostly gravel pads that have been constructed for development purposes, support little or no vegetation, and offer a poor matrix for the lemming to use for burrowing. For these reasons the IS used for the brown lemming is 0.5 rather than 1.0.

Arctic fox. IS = 0.01; the home range of the fox is extremely variable. Eberhardt et al. (1982) report a home range of 3.7 to 20.8 km² (370 to 2080 ha) for juvenile and adult arctic foxes, respectively. Using the lower end of the reported range (370 ha) results in an IS value of 0.01.

Caribou. IS = 0.01; caribou are highly mobile, covering large distances during their movements to and from calving grounds and in their constant search for suitable forage. They may range over thousands of kilometers a year, and as a result there is no accurate estimate of their home range. Based on knowledge of the caribou's habits and professional judgement, a very conservative (likely to overestimate exposure) IS value of 0.01 is used for the caribou.

3.2.7.3 Exposure Assessment for Representative Species of Birds. In this section the methods for quantifying exposures to the selected representative species of birds are presented.

In order to estimate exposures of the representative species of birds, life history information was compiled for the selected species. This information includes: occurrence at the DEW Line sites, habitat, average body weight, estimated food intake rate, estimated water intake rate, diet composition, and home range and/or population density.

Plant uptake of contaminants has been quantified for use in the exposure estimations for herbivores (bird and mammal species). Herbivores are potentially exposed to contamination directly from ingestion of soil and water intake as well as through their diet. The dietary plant component (CF in the exposure estimate equation) is calculated by multiplying the contaminant's soil concentration by the BCF, B_v . B_v is defined as the ratio of the concentration in aboveground parts of a plant (mg of compound/kg of dry plant) to the concentration in soil (mg of compound/kg of dry soil). The B_v can be used to predict the level of a potential contaminant taken up by a plant, and this information can then be used to assess the potential transport of the contaminant in the trophic web.

The uptake of metals by plants is quantified using the B_v values in Baes et al. (1984). These values represent potential uptake to the vegetative portions of the plant. The approach for organic chemicals is basically the same, except that the B_v s for organic chemicals are derived using a regression equation (Travis and Arms 1988). The equation is:

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

where:

$$\begin{aligned} B_v &= \text{the BCF (unitless) and} \\ K_{ow} &= \text{the octanol-water partition coefficient of the chemical (mol/m}^3 \text{ / mol/m}^3\text{).} \end{aligned}$$

In order to calculate the potential uptake of DRPH by plants, the K_{ow} of diesel fuel was estimated. The estimation of the K_{ow} was conducted using equation 2-3 in Lyman et al. (1982):

$$\log S = -0.922 \log K_{ow} + 4.184$$

where:

$$\begin{aligned} S &= \text{solubility (mg/L) and} \\ K_{ow} &= \text{octanol/water partition coefficient (mol/m}^3 \text{ / mol/m}^3\text{).} \end{aligned}$$

This equation estimates the solubility of an organic chemical in water. It may also be manipulated arithmetically to calculate the $\log K_{ow}$ based on the known solubility:

$$\log K_{ow} = \frac{\log S - 4.184}{-0.922}$$

The solubility of diesel fuel (0.2 mg/L) (Custance et al. 1992) was used to calculate the $\log K_{ow}$ of diesel fuel. The $\log K_{ow}$ is calculated to be 5.3.

Life history information for the Lapland longspur, brant, glaucous gull, pectoral sandpiper, and spectacled eider (the evaluation of the spectacled eider, a threatened species, is presented with the other avian species) is presented in Tables 3-6 through 3-10.

Information is not available on the daily food intake rate (grams/day) and water intake rate (liters/day) for the representative bird species in the arctic habitat. Therefore, this information was estimated using equations associated with body weight (Calder and Braun 1983). The severity of the arctic climate may impose higher metabolic demands on animals. As a result, the food and water intake rates should be considered estimates only, and their uncertainty should be kept in mind. The food intake rate was estimated using Nagy's (1987) equations:

Passerine birds (i.e., Lapland longspur):

$$FI \text{ (kg/day dry matter)} = 0.141 \times (\text{body weight in kilograms})^{0.850}$$

All other birds:

$$FI \text{ (kg/day dry matter)} = 0.0582 \times (\text{body weight in kilograms})^{0.651}$$

TABLE 3-6. LIFE HISTORY INFORMATION FOR THE LAPLAND LONGSPUR, *Calcarius lapponicus*

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at DEW Line Sites	seasonal breeder at arctic coastal radar installations	dominant breeding passerine	USAF 1993
Habitat	breeds on arctic coastal tundra		Scott 1983
Body Weight	27.3 g (0.027 kg)	mean of 68 specimens	Dunning 1984
Food Intake Rate	6.5 g/day dry matter	FI=0.141(BWkg) ^{0.850}	Nagy 1987
Water Intake Rate	0.005 liters/day	WI=0.059(BWkg) ^{0.67}	Calder and Braun 1983
Diet Composition	during breeding (June and July): insects (crane flies); pre- and post-breeding (May and August): seeds (grasses); average 25% vegetation in diet	passerine member of insectivorous foraging guild which includes shorebirds	Custer and Pitelka 1978
Population Density	38.6/km ²	varies with changing predation pressures	Derksen et al. 1981

TABLE 3-7. LIFE HISTORY INFORMATION FOR THE BRANT, *Branta bernicla*

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at DEW Line Sites	seasonal breeder at arctic coastal radar installations	breeding, migratory sp., subsistence sp.	USAF 1993
Habitat	breeds on Arctic Coastal Plain	prefers low, barren, wet, coastal terrain	Palmer 1976
Body Weight	1,305 g (1.305 kg)	mean of 791 specimens	Dunning 1984
Food Intake Rate	69.2 g/day dry matter	FI=0.0582(BWkg) ^{0.651}	Nagy 1987
Water Intake Rate	0.07 liters/day	WI=0.059(BWkg) ^{0.67}	Calder and Braun 1983
Diet Composition	sedges, grasses; average 90% vegetation in diet	some insects during breeding (June and July)	Palmer 1976
Population Density	5.0/km ²	average from 3 coastal sites	Derksen et al. 1981

TABLE 3-8. LIFE HISTORY INFORMATION FOR THE GLAUCOUS GULL, *Larus hyperboreus*

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at DEW Line Sites	seasonal breeder at arctic coastal radar installations	relatively common along arctic coast	Woodward-Clyde 1993
Habitat	coastal tundra, lakes, ponds, and marine environment	breeds on arctic coast	Farrand 1983
Body Weight	1,445 g (1.445 kg)	mean of 65 specimens	Dunning 1984
Food Intake Rate	74 g/day dry matter	$FI=0.0582(BWkg)^{0.651}$	Nagy 1987
Water Intake Rate	0.08 liters/day	$WI=0.059(BWkg)^{0.67}$	Calder and Braun 1983
Diet Composition	small fish, birds, insects, crustaceans, mollusks, garbage; average 10% of vegetation in diet	predatory scavenger	Martin et al. 1961
Population Density	0.8/km ²	average from 3 coastal sites	Derksen et al. 1981

TABLE 3-9. LIFE HISTORY INFORMATION FOR THE PECTORAL SANDPIPER, *Calidris melanotos*

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at DEW Line Sites	seasonal breeder at arctic coastal radar installations	abundant on Arctic Coastal Plain	Woodward-Clyde 1993
Habitat	grassy margins of wet meadows, marshes, riparian areas, ponds	nests hidden on well-drained grassy sites	Scott 1983, Martin et al. 1961
Body Weight	79 g (0.079 kg)	mean of 35 specimens	Dunning 1984
Food Intake Rate	11.2 g/day dry matter	$FI=0.0582(BWkg)^{0.651}$	Nagy 1987
Water Intake Rate	0.01 liter/day	$WI=0.059(BWkg)^{0.67}$	Calder and Braun 1983
Diet Composition	insects, mollusks, crustaceans, worms, vegetable debris; average 10% vegetation in diet	crane flies are major diet component	Martin et al. 1961, Pitelka 1959
Population Density	22.4/km ²	average from 3 coastal sites	Derksen et al. 1981

TABLE 3-10. LIFE HISTORY INFORMATION FOR THE SPECTACLED EIDER, *Somateria fischeri*

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at DEW Line Sites	potential seasonal breeder at all arctic coastal radar installations	winter whereabouts unknown	Woodward-Clyde 1993 Alaska Biological Research 1994
Habitat	marine when not breeding, nests on coastal tundra	nests on islets in tundra ponds and lakes, as well as ashore	Palmer 1976
Body Weight	1,375 g (1.375 kg)	mean of 32 specimens	Dunning 1984
Food Intake Rate	71.6 g/day dry matter	$FI=0.0582(BWkg)^{0.651}$	Nagy 1987
Water Intake Rate	0.07 liters/day	$WI=0.059(BWkg)^{0.67}$	Calder and Braun 1983
Diet Composition	75% insects, mollusks, crustaceans; average of 25% plant matter in diet	mostly insects when they are abundant; June and July	Kistchinski and Flint 1974
Population Density	0.32/km ²	average of three coastal sites	Derksen et al. 1981

The water intake rate was estimated using the equation developed by Calder and Braun (1983):

All birds:

$$WI \text{ (liters/day)} = 0.059 \times (\text{body weight in kilograms})^{0.67}$$

As animals forage they may incidentally ingest soil and sediment particles. The average concentration of contaminants in soil/sediment can be multiplied by the amount of soil/sediment ingested to estimate the potential uptake of contaminants by this route. Soil intake rates have been reported for just a few wildlife species (Beyer et al. 1994). The soil ingestion rates for the representative species are extrapolated from Beyer et al. (1994) by using similar species with reported values. The percentages reported are of the total weight of dietary intake. Table 3-11 lists the representative bird species, the species used as surrogates, and the estimated percentages of soil ingested in quantifying exposure to contaminants. Species that forage directly in the soil or sediment, such as the sandpiper or goose, show relatively high percentages of soil in their diet. The Lapland longspur does not have appropriate surrogate species with soil ingestion data. Although the longspur is in the same foraging guild as sandpipers (which incidentally ingest relatively large amounts of soil), the longspur takes insects from the soil surface or gleans its prey from vegetation (Custer and Pitelka 1978), thus minimizing its soil intake; the estimate of soil ingestion (less than 2 percent of diet by weight) reflects this. The glaucous gull ingests stones and sand as a mechanical addition (to aid in digestion) to its diet (Belopol'skii 1961) and this contributes to its soil/sediment intake. For those species without a suitable surrogate or whose soil ingestion rate is reported as <2 percent, a value of 2 percent of dietary intake (by weight) was used to calculate the exposure estimates.

TABLE 3-11. SOIL INGESTION ESTIMATES FOR REPRESENTATIVE BIRD SPECIES

REPRESENTATIVE SPECIES	SURROGATE SPECIES	ESTIMATED PERCENT OF SOIL IN DIET	ESTIMATED SOIL IN DIET (g/day)
Lapland longspur	no suitable surrogate	<2.0	0.1
brant	Canada goose	8.2	5.7
glaucous gull ^a	Siberian glaucous gull	7.6	5.6
pectoral sandpiper	4 sandpiper species (average)	18.1	2.0
spectacled eider	Canada goose	8.2	5.9

Source: Beyer et al. 1994.

^a Information from Belopol'skii 1961.

3.2.7.4 Exposure Assessment for Representative Species of Mammals. This section assesses exposure to contaminants for the selected representative species of mammals. Table 3-12 (brown lemming), Table 3-13 (arctic fox), and Table 3-14 (caribou) present life history data that are used to calculate exposure estimates for the representative mammalian species. Home range and/or population density has been listed for the representative mammal species, depending on appropriateness and availability.

Information on daily food intake rates for the arctic fox and caribou was not available. The rates have been estimated using equations associated with average body weights and metabolic rates (Nagy 1987). The food intake rates for the fox and caribou were estimated using the following equations, developed for placental mammals in general and for herbivorous mammals, respectively (Nagy 1987).

arctic fox: using equation for placental mammals in general

$$FI \text{ (kg/day dry matter)} = 0.0687 \times (\text{body weight in kilograms})^{0.822}$$

caribou: using equation for mammalian herbivores

$$FI \text{ (kg/day dry matter)} = 0.0875 \times (\text{body weight in kilograms})^{0.727}$$

Because of very low assimilation efficiencies, the low nutrient content of winter forage, and the high metabolic demands in arctic habitats (Chappell 1980), the equation for food intake rate significantly underestimates the rate for the brown lemming. A more appropriate rate for the brown lemming of 45 g/day is reported by Chappell (1980) (using the highest value in the reported range of 24-45 g/day).

TABLE 3-12. LIFE HISTORY INFORMATION FOR THE BROWN LEMMING, *Lemmus trimucronatus*

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at DEW Line Sites	resident at all arctic coastal radar installations	dominant small mammal	USAF 1993
Habitat	tundra and alpine meadows	nests above ground in winter, below in summer	Burt and Grossenheider 1976
Body Weight	55 g (0.055 kg)		Chappell 1980
Food Intake Rate	24-45 g/day dry matter	has low assimilation efficiencies (31-36%), variation also related to seasons	Chappell 1980
Water Intake Rate	0.007 liters/day	$WI=0.099(BWkg)^{0.9}$	Calder and Braun 1983
Diet Composition	sedges, grasses, lichens, roots, leaves, bark, berries		Nowak 1991
Home Range Size (AVG)	0.5 ha (females) 1.0 ha (males)	0.5 ha used in assessment	Nowak 1991
Population Density	0 to 325/ha	populations have large fluctuations on a 3-5 year cycle; currently, populations are low	Nowak 1991, Snyder-Conn 1994

TABLE 3-13. LIFE HISTORY INFORMATION FOR THE ARCTIC FOX, *Alopex lagopus*

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at DEW Line Sites	resident at all arctic coastal radar installations	ubiquitous	USAF 1993
Habitat	tundra and coastal plain	dens in sandy mounds >1 m high	Chesemore 1967
Body Weight	4950 g (4.95 kg)		Burt and Grossenheider 1976
Food Intake Rate	256 g/day dry matter	$FI=0.0687(BWkg)^{0.822}$	Nagy 1987
Water Intake Rate	0.42 liters/day	$WI=0.099(BWkg)^{0.90}$	Calder and Braun 1983
Diet Composition	brown lemming (summer), nesting birds, carrion, seal pups, non-food items	brown lemming in >85% of all scats, n=224	Chesemore 1967, Nowak 1991
Home Range Size (AVG)	20.8 km ² adult 3.7 km ² juvenile (<1 yr)	adult range used in assessment	Eberhardt et al. 1982

TABLE 3-14. LIFE HISTORY INFORMATION FOR THE BARREN-GROUND CARIBOU, *Rangifer tarandus*

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at DEW Line Sites	seasonal, at or near all arctic coastal radar installations during migrations	areas near some installations used for calving	USAF 1993
Habitat	tundra in summer, open coniferous forest in winter	varies, related to migration	Burt and Grossenheider 1976
Body Weight	95,500 g (95.5 kg)	mean for adults	Nowak 1991
Food Intake Rate	2400 g/day (2.4 kg) dry matter	$FI=0.0875(BWkg)^{0.727}$	Nagy 1987
Water Intake Rate	6.0 liters/day	$WI=0.099(BWkg)^{0.90}$	Calder and Braun 1983
Diet Composition	willows, sedges, cottongrass, lichens	selection based on plant phenology	Skogland 1980, White and Trudell 1980
Population Density	1.41 km ² 0.31 km ² 4.53 km ²	undisturbed calving area within 1 km of road within 5-6 km of road	Cameron et al. 1992

The rates for water intake of the representative mammals were estimated using the equation generated by Calder and Braun (1983) because of the unavailability of species-specific information in the literature. The equation is:

$$WI \text{ (liters/day)} = 0.099 \times (\text{body weight in kilograms})^{0.90}$$

Incidental soil intake was evaluated for mammals in the same manner as for birds (Section 3.2.7.3). Table 3-15 shows the percent of soil ingested for the representative mammal species.

3.3 ECOLOGICAL TOXICITY ASSESSMENT

This section presents toxicity information for each COC in surface water and soil/sediment. The COCs identified in surface water are DRPH, GRPH, iron, and manganese (Section 3.1.1). The COCs in soils/sediments are DRPH, GRPH, RRPH, BTEX, tetrachloroethene, trichloroethane, trichloroethene, benzyl alcohol, lead, selenium, and zinc (Section 3.1.2). Sections 3.3.1 through 3.3.14 discuss the toxicity of all COCs to the receptor groups. Section 3.3.15 presents the methodology for the derivation of TRVs used for this ERA.

TABLE 3-15. SOIL INGESTION ESTIMATES FOR REPRESENTATIVE MAMMAL SPECIES

REPRESENTATIVE SPECIES	SURROGATE SPECIES	ESTIMATED PERCENT OF SOIL IN DIET	ESTIMATED SOIL IN DIET (g/day)
brown lemming	prairie dog (average of 2 species)	2.7	1.2
arctic fox	red fox	2.8	7.2
caribou	elk	<2.0	48

Source: Beyer et al. 1994

3.3.1 Petroleum Hydrocarbons

Section 3.1 presented the COCs for sites at the Point Lay installation. Petroleum hydrocarbons were identified as COCs in surface water (DRPH and GRPH) and in soil/sediment (DRPH, GRPH, and RRPB). This section is a discussion of the chemical differences between DRPH, GRPH, and RRPB and the toxicity of these three petroleum mixtures.

Crude petroleum contains thousands of different chemical compounds. Gasoline and diesel fuel are refined petroleum products. The composition of gasoline and diesel fuel depends on not only the origin of the crude oil from which the gasoline is derived, but also the process technique and the blending scheme (Von Burg 1993). Once gasoline or diesel fuel is released to the environment, weathering and volatilization further alter its composition.

Gasoline is a complex, highly variable mixture of petroleum hydrocarbons containing 3 to 21 carbon atoms; however, compounds with 4 to 12 carbon atoms predominate. Gasoline is detected with the petroleum hydrocarbon analysis as GRPH. The following chemical classes are detected as GRPH: paraffins (straight-chained alkanes), olefins (straight-chained alkenes), naphthenes (cycloalkanes and alkenes), and aromatic hydrocarbons [alkylbenzenes and polynuclear aromatic hydrocarbons (PAHs)] (Von Burg 1993). Although GRPH are generally in the range of 4 to 12 carbon atoms, the laboratory that conducted the analyses for Point Lay detected GRPH with 6 to 9 carbon atoms. As many as 140 compounds have been identified as constituents of gasoline; however, constituents such as benzene drive the toxicity. Diesel fuel is also a complex, variable mixture of the same classes of compounds containing 6 to 21 carbon atoms. Diesel fuel is detected with petroleum hydrocarbon analysis as DRPH. The laboratory that analyzed samples for Point Lay detected DRPH with 10 to 24 carbon atoms. As many as 45 compounds have been identified as constituents of diesel fuel (Von Burg 1993). The concentrations of RRPB were determined by analysis of petroleum hydrocarbons with 24 carbon atoms or more.

Table 3-16 presents the chemical classes and weight percent for GRPH and DRPH. Generally, gasoline contains more aromatic compounds and simple-chained alkanes, whereas diesel fuel is characterized by cycloparaffins (or cycloalkanes). Both gasoline and diesel fuel will be affected

TABLE 3-16. CHEMICAL CLASSES OF GRPH AND DRPH

CHEMICAL CLASS	WEIGHT PERCENT ^a
GRPH^b	
Normal paraffins (n-alkanes)	19.3-38.4 (28.8)
Isoparaffins (isoalkanes)	11.5-50.3 (30.9)
Naphthenes (cycloparaffins or cycloalkanes)	1.0-2.8 (1.9)
Aromatics (e.g., benzene, toluene, pyrene)	9.7-54.7 (32.2)
DRPH^c	
Normal paraffins (n-alkanes)	5.6
Isoparaffins (isoalkanes)	11.1
Naphthenes (cycloparaffins or cycloalkanes)	46.3
Aromatics (e.g., benzene, toluene, pyrene)	33.3
Nitrogen, sulfur and oxygen compounds	3.7

^a Average shown in parentheses.

^b Heath et al. 1993.

^c Weeks et al. 1988.

by the environment. Weathering will change the chemical composition of petroleum, and concentrations of aromatic compounds such as benzene will decrease as a result of volatilization.

Available toxicity test data have been derived from pure, fresh product, and therefore the applicability to the weathered product encountered at Point Lay is uncertain. Gasoline is the most studied of the petroleum products; however, most data are based on inhalation studies. Gasoline was classified by EPA (1992b) as a Group C (possible human) carcinogen, whereas diesel oil was classified as Group D (not classifiable as to human carcinogenicity). Presumably, this classification of gasoline is due to benzene that, under the conditions of environmental exposure, would volatilize more rapidly than any other constituent. The gasoline and diesel petroleum hydrocarbon data from surface water samples collected at Point Lay indicate that benzene was detected in surface water below action levels, and the soil/sediment samples showed concentrations above action levels. Physical-chemical data from the literature indicates that total petroleum hydrocarbon (TPH) in soil would reflect all constituents with eventual loss of aromatic (e.g., BTEX) components first, lighter alkanes second, lighter PAHs third, followed by naphthalenes. For an old diesel or petroleum spill, petroleum hydrocarbon measurements may

reflect predominantly trace amounts of high molecular-weight PAHs or higher molecular-weight and branched alkanes [Massachusetts Department of Environmental Protection (MDEP) 1993].

For the purposes of ranking the toxicity of GRPH, DRPH, and RRPB, it was assumed that BTEX and lighter-weight alkanes have been significantly weathered in the arctic environment, and that toxicity is more dependent upon noncarcinogenic endpoints associated with alkanes, alkenes, and cycloalkanes. The toxicity of DRPH and RRPB is also associated with the PAH content. At Point Lay, however, only two PAHs were detected: naphthalene (detected in surface water and soil/sediment) and 2-methylnaphthalene (detected in soil/sediment), chemicals considered to be noncarcinogenic. Other carcinogenic PAHs, such as benzo(a)pyrene, were not detected. MDEP (1993) reviewed the noncarcinogenic toxicological endpoints in laboratory animals for diesel fuel and gasoline, and determined that diesel fuel was an order of magnitude more toxic than gasoline, although other sources indicate the toxicity of alkanes and cycloalkanes is similar (Armstrong Laboratory 1994; Sax and Lewis 1989). A review of the Point Lay data indicates that DRPH are present at higher concentrations than GRPH and at similar concentrations to RRPB. Specifically, average concentrations of DRPH in surface water were approximately 8 times higher than average concentrations of GRPH in surface water, and the soil/sediment concentrations of DRPH were about 19 times greater than the GRPH concentrations and about twice the RRPB concentrations. As a result, based on the MDEP review and the chemical data reported for the Point Lay surface water and soil/sediment samples, the evaluation of DRPH is used to conservatively represent ecological risks from petroleum hydrocarbon contamination (i.e., including GRPH, RRPB, and other constituent chemicals such as naphthalene, 2-methylnaphthalene, 1,2,4-trimethylbenzene, and 1,3,5-trimethylbenzene).

As discussed above, diesel fuel is comprised of a complex mixture of paraffins (straight-chained alkanes), olefins (straight-chained alkenes), naphthenes (cycloalkanes and alkenes), and aromatic (alkylbenzenes, and polynuclear) petroleum hydrocarbons containing 6 to 21 carbon atoms. Hydrocarbons containing 8 to 18 carbon atoms predominate (Von Burg 1993). There are six grades of diesel fuel (Diesel Oil No. 1, Diesel Oil No. 2, Diesel Oil No. 4, Fuel Oil No. 1, Fuel Oil No. 2, and Home Heating Oil) (Von Burg 1993). The specific components of diesel are expected to change from source to source, so the toxicity of diesel fuels is expected to be variable. The following sections summarize the toxicity of diesel fuel to plants, aquatic organisms, birds, and mammals.

3.3.1.1 Plants. Petroleum released to the aquatic environment is expected to be toxic to aquatic plants. Toxicity tests have shown that the water-soluble components of petroleum are toxic to an algal species (*Chlorella vulgaris*) (Kauss and Hutchinson 1975); however, in this specific study, the toxicity was short term. The algal community recovered after a "lag phase". It was theorized (Kauss and Hutchinson 1975) that this trend was due to the loss of highly volatile fractions from the testing chamber over time. Exposure to water extracts of No. 2 Fuel Oil depressed algal biomass in communities and resulted in blue-green algal dominance and decreased diatom occurrence (Bott and Rogenmuser 1978).

3.3.1.2 Aquatic Organisms. Moles et al. (1979) tested the acute toxicity of Prudhoe Bay crude oil to several Alaskan freshwater and anadromous fish. Salmonids were the most sensitive species tested, and demonstrated median tolerance limits (the concentration at which one half

the organisms survive in 96 hours, same as LC_{50} [lethal concentration for 50 percent of the organisms]) ranging from 2.7 to 4.4 mg/L. The three-spined stickleback, *Gasterosteus aculeatus*, was more tolerant, with an LC_{50} of 10.4 mg/L. Klein and Jenkins (1983) studied the toxicity of the water-soluble fraction of jet fuel to fish. Growth of fry was retarded by 1.5 mg/L of the water-soluble fraction of JP-8 (jet fuel with de-icer). In a study conducted by Hedtke and Puglisi (1982), the method of introducing the oil to the test chamber was an important variable driving toxicity. Emulsified oils were substantially more toxic than either floating oils or the water-soluble fraction. The 96-hour LC_{50} for fathead minnows (*Pimephales promelas*) exposed to the emulsion of No. 2 jet fuel was 38.6 mg/L (concentration used to calculate the TRV).

Aquatic organisms other than fish may also be exposed to diesel fuel in the environment. Studies have shown that freshwater arctic zooplankton may be more sensitive to oil pollution than any other arctic freshwater organisms (O'Brien 1978). Geiger and Buikema (1981) estimated an LC_{20} (concentration lethal to 20 percent of the test organisms) of No. 2 Fuel oil to *Daphnia pulex* of 5.6 mg/L (concentration used to calculate TRV).

3.3.1.3 Birds. Petroleum hydrocarbons in the environment may affect bird reproduction. External application of Number 2 fuel oil to mallard (*Anas platyrhynchos*) and common eider (*Somateria mollissima*) eggs significantly increased embryo mortality (Albers 1977; Szaro and Albers 1977). Mallard eggs were treated with 1, 5, 10, 20, and 50 $\mu\text{g/L}$ of fuel oil. Ingestion of crude oil by mallards at a concentration of five percent by weight in the diet resulted in depressed growth (Szaro et al. 1978). Hartung (1964) demonstrated a decrease in weight gain in mallards during the first 10 days after receiving 6,000 mg/kg No. 2 fuel oil (concentration used to calculate TRV); however, after 34 days, there was no difference between treatment groups and the controls.

3.3.1.4 Mammals. The available literature does not present a great deal of information regarding the toxicity of diesel fuel to mammals, but it can be represented by the toxicity of the compound to rats. Diesel fuel is relatively nontoxic to rats based on an acute oral LD_{50} (lethal dose for 50 percent of the organisms) of 7,380 mg/kg (Beck et al. 1982) (dose used to calculate TRV).

3.3.2 Benzene

Benzene is a COC in soil/sediment at the Point Lay sites. It is a highly volatile aromatic hydrocarbon that is used as a solvent and in the synthesis of other chemicals. Benzene is typically a component of petroleum products and is found naturally in the environment (ATSDR 1993; Klaasen et al. 1986). The major toxic effect of benzene in animals is hematopoietic toxicity (i.e., blood disorders, including anemia and leukemia) (Klaasen et al. 1986). A summary of the relevant toxicity information is presented below.

3.3.2.1 Plants. No information was available concerning the toxicity of benzene to plants. A qualitative discussion of the toxicity of VOCs to plants is presented in Section 3.4.1, Risk Characterization.

3.3.2.2 Aquatic Organisms. Benzene was not selected as a COC in surface water; therefore, no aquatic toxicity information is presented.

3.3.2.3 Birds. No toxicity information was available for benzene toxicity to birds. A qualitative discussion and comparison of mammalian and avian toxicity values is presented in Section 3.4.3, Risk Characterization.

3.3.2.4 Mammals. Nawrot and Staples (1979 in Opresko et al. 1994) reported a chronic reproductive Lowest Observed Adverse Effect Level (LOAEL) for laboratory mice orally exposed to benzene at 263.6 mg/kg. Using a UF of 0.1, Opresko et al. (1994) convert the LOAEL to a chronic No Observed Adverse Effect Level (NOAEL) for mice of 26.36 mg/kg. This is the value used to calculate the benzene TRV for mammals.

3.3.3 Toluene

Toluene, identified as a COC in soil/sediment at the Point Lay sites, is an aromatic hydrocarbon classed as an alkylbenzene. It is a common constituent of petroleum products (ATSDR 1993). Toluene toxicity in animals is commonly manifest as depression of the central nervous system (Klaasen et al. 1986). A summary of the relevant toxicity information is presented below.

3.3.3.1 Plants. No information was available concerning the toxicity of toluene to plants. A qualitative discussion of the toxicity of VOCs to plants is presented in Section 3.4.1, Risk Characterization.

3.3.3.2 Aquatic Organisms. Toluene was not selected as a COC in surface water; therefore, no aquatic toxicity information is presented.

3.3.3.3 Birds. No toxicity information was available for toluene toxicity to birds. A qualitative discussion and comparison of mammalian and avian toxicity values is presented in Section 3.4.3, Risk Characterization.

3.3.3.4 Mammals. Nawrot and Staples (1979 in Opresko et al. 1994) reported a chronic reproductive LOAEL for laboratory mice orally exposed to toluene at 259.8 mg/kg. Using a UF of 0.1, Opresko et al. (1994) convert the LOAEL to a chronic NOAEL for mice of 25.98 mg/kg. This is the value used to calculate the toluene TRV for mammals.

3.3.4 Ethylbenzene

Ethylbenzene is a COC in soil/sediment at the Point Lay sites. It is a VOC, classified as an alkylbenzene, and is a common component of petroleum products (ATSDR 1993). Most toxicity information in the literature relates to its inhalation. The primary toxic effect of ethylbenzene on animals is depression of the central nervous system (Klaasen et al. 1986). A summary of the relevant toxicity information is presented below.

3.3.4.1 Plants. In a cell multiplication inhibition test using *Microcystis aeruginosa* (algae), the toxicity threshold of ethylbenzene was 33,000 µg/L. The toxicity threshold of *Scenedesmus*

quadricauda (green algae) to ethylbenzene was $>160,000 \mu\text{g/L}$ (Verschuere 1983). Galassi et al. (1988 in AQUIRE 1994) reported an EC_{50} (effective concentration for 50 percent of the organisms) (growth) of $4,600 \mu\text{g/L}$ for *Selenastrum capricornutum* (green algae).

3.3.4.2 Aquatic Organisms. Because ethylbenzene is not a COC in surface water, no toxicity information is presented for aquatic organisms.

3.3.4.3 Birds. There is no information available in the literature regarding the toxicity of ethylbenzene to birds. A discussion of comparative toxicities of avian and mammalian species is presented in Section 3.4.3, Risk Characterization.

3.3.4.4 Mammals. Budavari (1989) reported an LD_{50} of $5,460 \text{ mg/kg}$ for ethylbenzene based on oral administration to rats. This was the dose used to calculate TRV.

3.3.5 Xylene

Xylene is a COC in soil/sediment at the Point Lay sites. It is classed as an alkylbenzene and is a common constituent of petroleum products (ATSDR 1993). Most toxicity information in the literature relates to the inhalation of xylene. A summary of the relevant information is presented below.

3.3.5.1 Plants. In a study of the green algae, *Selenastrum capricornutum*, xylene decreased growth at concentrations of $72,000 \mu\text{g/L}$ (Gaur 1988 in AQUIRE 1990).

3.3.5.2 Aquatic Organisms. Xylene is not a COC in surface waters, so toxicity information for aquatic organisms is not presented.

3.3.5.3 Birds. When mallard eggs were immersed in xylene (1 percent and 10 percent) for 30 seconds, there was no significant effect on embryonic weight and length when compared to controls (Hoffman and Eastin 1981 in HSDB 1994). Japanese quail (*Coturnix japonica*) fed xylene demonstrated no sign of toxicity up to $5,000 \text{ ppm}$ (USFWS 1986). The LC_{50} was $>20,000 \text{ ppm}$ (USFWS 1986). Hill and Camardese (1986) report a maximum dietary exposure level for Japanese quail of 625 mg/kg total xylenes (dose used to calculate TRV).

3.3.5.4 Mammals. Ingestion of xylene in mammals may cause prenatal mortality, growth inhibition, and malformations, primarily cleft palate. The LD_{50} for ingestion of xylene (rat) was reported as $4,300 \text{ mg/kg}$ (Clayton and Clayton 1981) (dose used to calculate TRV).

3.3.6 Tetrachloroethene

Tetrachloroethene is a COC in soil/sediment at the Point Lay sites. It is a halogenated hydrocarbon used in commercial dry cleaning operations and as a solvent. It has demonstrated experimental carcinogenic, teratogenic, and reproductive effects (Sax and Lewis 1989). Toxicity information for this compound is limited. Available information for tetrachloroethene is summarized below.

3.3.6.1 Plants. No specific information on toxicity of tetrachloroethene to plants is available.

3.3.6.2 Aquatic Organisms. Tetrachloroethene was not selected as a COC in water, so it is not quantitatively evaluated in the risk assessment for aquatic organisms.

3.3.6.3 Birds. No avian toxicity information for tetrachloroethene was available in the literature, but a qualitative evaluation of avian species may be made using mammalian toxicity values. A comparison of avian and mammalian toxicity is presented in Section 3.4.3, Risk Characterization.

3.3.6.4 Mammals. Buben and O'Flaherty (1985 in Opresko et al 1994) conducted a six-week subchronic exposure test of tetrachloroethene on mice. A subchronic NOAEL was determined to be 20 mg/kg. Applying conversion and UFs specified in Opresko et al. (1994), a final NOAEL of 1.4 mg/kg was derived. This is the value used to calculate the TRV.

3.3.7 Trichloroethane

Trichloroethane was identified as a COC in soil/sediment at the Point Lay sites. It is a halogenated hydrocarbon that is highly toxic to the kidneys of mice and rats (Klaasen et al. 1986). Typically, this chemical is reported in one of its isomeric forms, 1,1,1-trichloroethane or 1,1,2-trichloroethane. The lowest, or most sensitive, toxicity value was selected for use in the risk assessment (see Section 3.3.7.4, Mammals). Available toxicity information is presented below.

3.3.7.1 Plants. No specific information on toxicity for trichloroethane to plants is available.

3.3.7.2 Aquatic Organisms. Trichloroethane was not selected as a COC in water; therefore, no aquatic toxicity information is presented.

3.3.7.3 Birds. No avian toxicity information for trichloroethane was available in the literature, but a qualitative evaluation of avian species may be made using mammalian toxicity values. A comparison of avian and mammalian toxicity is presented in Section 3.4.3, Risk Characterization.

3.3.7.4 Mammals. Sax and Lewis (1989) report an oral 1,1,1-trichloroethane LD₅₀ for rats of 10,300 mg/kg and an oral 1,1,2-trichloroethane LD₅₀ for rats of 580 mg/kg. The 580 mg/kg value was used to calculate the TRV.

3.3.8 Trichloroethene

Trichloroethene (trichloroethylene) was identified as a COC in soil/sediment at the Point Lay sites. It is a halogenated hydrocarbon that has been shown to be toxic to the kidneys of mice and rats (Klaasen et al. 1986). Available toxicity information is presented below.

3.3.8.1 Plants. Specific information on toxicity of trichloroethene to plants is not available.

3.3.8.2 Aquatic Organisms. Trichloroethene was not selected as a COC in water; therefore, no aquatic toxicity information is presented.

3.3.8.3 Birds. No avian toxicity information for trichloroethene was available in the literature, but a qualitative evaluation of avian species may be made using mammalian toxicity values. A comparison of avian and mammalian toxicity is presented in Section 3.4.3, Risk Characterization.

3.3.8.4 Mammals. The National Toxicology Program (NTP 1986 in ATSDR 1991b) reports a two generation NOAEL for rats exposed to 300 mg/kg trichloroethene in their food. This dose is used to calculate the TRV.

3.3.9 Benzyl Alcohol

Benzyl alcohol was identified as a COC in soil/sediment at the Point Lay sites. Toxicity information is extremely limited for this chemical and the available information is presented below.

3.3.9.1 Plants. No information on toxicity of benzyl alcohol to plants was available.

3.3.9.2 Aquatic Organisms. Benzyl alcohol was not identified as a COC in surface water; therefore, no aquatic toxicity information is presented for benzyl alcohol.

3.3.9.3 Birds. No avian toxicity information for benzyl alcohol was available in the literature, but a qualitative evaluation of avian species may be made using mammalian toxicity values. A comparison of avian and mammalian toxicity is presented in Section 3.4.3, Risk Characterization.

3.3.9.4 Mammals. Sax and Lewis (1989) report an oral LD₅₀ of 1,230 mg/kg for rats exposed to benzyl alcohol. This is the dose used to calculate the TRV.

3.3.10 Iron

Iron was identified as a COC in surface water at the Point Lay sites. Iron is an essential trace element required by both plants and animals. It plays an important role in the transport of oxygen in animals. Available information for iron is summarized below.

3.3.10.1 Plants. In a study conducted by Foy et al. (1978 in EPA 1985), concentrations of 100 to 500 ppm soluble iron in soil were toxic to rice.

3.3.10.2 Aquatic Organisms. Iron may be a threat in aquatic environments in the form of precipitates that can destroy habitat, coat gills, and inhibit oxygen uptake. The EPA uses 1,000 µg/L as the chronic AWQC protective of aquatic life (dose used to calculate TRV) (EPA

1986). Doudoroff and Katz (1953) found iron concentrations of 1,000 to 2,000 µg/L toxic to *Esox lucius* (northern pike) and trout (species not reported).

3.3.10.3 Birds. There are few studies available that address the toxicity of iron to species of wild birds. There were no adverse effects produced in turkeys at concentrations of 440 ppm (Woerpel and Balloun 1964 in NAS 1980). NAS (1980) recommends that the maximum tolerable level of dietary iron of 1,000 ppm be used for poultry. The 1,000 ppm dose converts to 70.0 mg/kg for a maximum tolerable dietary level for a chicken (dose used to calculate TRV).

3.3.10.4 Mammals. At high concentrations, iron is toxic to livestock and interferes with phosphorus metabolism (NAS 1974 in EPA 1976). Cattle fed 477 µg/g iron demonstrated a slight decrease in weight gain; concentrations of 1,677 µg/g of iron produced a significant decline in growth rate (EPA 1985). Shanas and Boyd (1969 in NAS 1980) report an acute LD₅₀ dose of 1000 mg/kg for the rat (dose used to calculate TRV for brown lemming and arctic fox). The maximum tolerable dietary level of 500 ppm of iron (converted to 20 mg/kg) for sheep is used to calculate the TRV for caribou (NAS 1980).

3.3.11 Lead

Lead was selected as a COC in soil/sediment. Lead is a trace element naturally found in environmental media (e.g., soil, water, etc.); however, it is neither essential nor beneficial to living organisms (Eisler 1988). Available information for lead is summarized below.

3.3.11.1 Plants. Lead inhibits plant growth and reduces photosynthesis, mitosis, and water absorption (Eisler 1988). Concentrations of 500 mg/kg in soils were found to result in reduced pollen germination in several weed species, but the same study found that 46 mg/kg lead concentrations in soil did not have adverse effects on pollen germination (USACOE 1991).

3.3.11.2 Aquatic Organisms. Lead was not found to be a COC in surface water; therefore, no toxicity information is presented for aquatic organisms.

3.3.11.3 Birds. The bulk of the toxicity information in the literature regarding avian exposure to lead concerns waterfowl that have ingested spent lead shot and died. There is, however, limited dose-response information available for some species. Mautino and Bell (1987) reported neurological effects in mallards that had ingested and absorbed lead shot for a total intake of 423.8 mg/kg body weight. Young American kestrels (*Falco sparverius*) (1 day old) that ingested 125 and 625 mg/kg body weight of lead showed significantly depressed growth and hematocrit values (Hoffman et al. 1985). Based on a review of several studies, 12.0 mg/kg (USACOE 1991) is the highest chronic NOAEL lead dose for many species of birds. This is the dose used to calculate the TRV.

3.3.11.4 Mammals. Lead may affect the survival, growth, development, and metabolism of animal species. Rats are affected by 5 to 108 mg/kg body weight (acute oral dose); dogs by 0.32 mg/kg body weight daily (chronic oral dose); and horses by chronic dietary concentrations of 1.7 mg/kg (Eisler 1988). Azar et al. (1973 in Opresko et al. 1994) report an oral dose of 8.0 mg/kg-bw/day as a chronic NOAEL for laboratory rats (dose used to calculate TRV for brown

lemming and arctic fox). Fick et al. (1976 in NAS 1980) report a chronic NOAEL for sheep of 10 ppm (converted to 0.4 mg/kg) in diet. This value is used to calculate the TRV for caribou.

3.3.12 Manganese

Manganese was determined to be a COC in surface water. Manganese is considered to be an essential nutrient for animals (ATSDR 1990), and it is important for growth and reproduction. The toxicity of manganese can be affected by pH and water hardness, although these interactions were not analyzed for the Point Lay sites. Available information for manganese is summarized below.

3.3.12.1 Plants. In a four-day study conducted using duckweed (*Lemna minor*), an EC₅₀ (reduction in growth) of 31,000 µg/L was reported (Wang 1986 in AQUIRE 1990). Lewis et al. (1979) studied the species composition of freshwater phytoplankton populations when exposed to manganese. Population composition was altered at 0.1 mg/L manganese. Soil concentrations of 1,500 to 3,000 mg/kg were reported as phytotoxic to all plant species (Kabata-Pendias and Pendias 1984).

3.3.12.2 Aquatic Organisms. In a study conducted by Doudoroff and Katz (1953), brook trout were killed within 24 hours when exposed to concentrations of manganese of 6,250 µg/L. *Daphnia* spp. have a reported 16 percent reproductive impairment in water with concentrations of 4,100 µg/L (Biesinger and Christensen 1972 in Lewis et al. 1979) (dose used to calculate TRV for *Daphnia* spp).

3.3.12.3 Birds. Vohra and Kratzer (1968 in NAS 1980) exposed young turkeys to dietary manganese for 21 days. A NOEL of 4,080 ppm was derived. The maximum tolerable levels of manganese recommended by the NAS are 2,000 ppm (250 mg/kg body weight) for poultry. This is the value used to calculate the avian TRV.

3.3.12.4 Mammals. When fed 9,000 ppm manganese, sheep demonstrated reduced feed intake (Puls 1988). NAS (1980) recommends maximum tolerable levels of 1,000 ppm for cattle (15 mg/kg body weight) and sheep (40 mg/kg body weight). The value for sheep is used to calculate the TRV for caribou. A NOAEL of 930 mg/kg-bw/day is reported for rats in ATSDR (1990). The TRVs for brown lemming and arctic fox are based on the 930 mg/kg dose.

3.3.13 Selenium

Selenium was selected as a COC in soil/sediment at the Point Lay sites. It is an essential trace mineral with toxic levels about 10-50 times the recommended dietary levels (NAS 1980). Selenium, in toxic concentrations, has been associated with malformations, reduced survival, and decreased numbers of offspring in birds (Heinz et al. 1989 in USACOE 1991). Available information for selenium is summarized below.

3.3.13.1 Plants. Kabata-Pendias and Pendias (1984) report concentrations of selenium ranging from 5 to 10 mg/kg in soil as phytotoxic to crop plants (species unreported). The symptoms of selenium phytotoxicity included leaf chlorosis and growth reduction.

3.3.13.2 Aquatic Organisms. Selenium was not found to be a COC in surface water; therefore, no toxicity information is presented for aquatic organisms.

3.3.13.3 Birds. Heinz et al. (1989 in USACOE 1991) reported adverse reproductive effects in birds exposed to concentrations of selenium of 8 mg/kg. Opresko et al. (1994) report a chronic reproductive selenium NOAEL for mallards of 0.5 mg/kg, and this is the value used to calculate the avian TRV.

3.3.13.4 Mammals. Schroeder and Mitchner (1971 in Opresko et al. 1994) studied laboratory mice orally dosed with selenium. They reported a chronic reproductive LOAEL of 0.75 mg/kg, and this dose is used to calculate the mammalian TRV for selenium.

3.3.14 Zinc

Zinc was determined to be a COC in soil/sediment. Zinc is considered to be an essential nutrient for animals (Eisler 1993) and is necessary for plant growth. Deficiencies of zinc in the diet may retard growth in animals (Eisler 1993). Toxicity information for zinc, and the groups of representative species, is presented below.

3.3.14.1 Plants. According to information presented in Eisler (1993), plants that are sensitive to zinc concentrations die when soil levels are in excess of 100 mg/kg or if plant zinc content exceeds 178 mg/kg. The amount of zinc absorbed from soil by plants is dependent upon soil-specific characteristics. USACOE (1991) reports that several species of plants find average concentrations of zinc of 270 mg/kg in soil to be phytotoxic.

3.3.14.2 Aquatic Organisms. Zinc was not identified as a COC in surface water at the Point Lay sites; therefore, no toxicity information is presented for aquatic organisms.

3.3.14.3 Birds. When ducks were fed 2,500 to 3,000 mg/kg ration of zinc, or alternately, force-fed zinc at 742 mg/kg body weight, survival was reduced (Eisler 1993). Chickens were more resistant to zinc exposure; zinc/kg ration was lethal to chicks (Eisler 1993). Elevated levels of zinc (20 g zinc/kg ration) are given to poultry to induce molting and subsequently reduce egg deposition (Eisler 1993). A four-week study conducted by Roberson and Schaible (1960 in NAS 1980) calculated a NOAEL of 1,000 ppm for one-day-old chicks. This value was used to calculate the avian TRV.

3.3.14.4 Mammals. According to Eisler (1993), zinc is relatively non-toxic to mammals (as might be expected for an essential trace element). There is a large range in concentrations between normal dietary intakes and concentrations expected to cause harm. Adult male rats, when fed zinc at levels of 500 mg/kg diet, were adversely affected; spermatogenesis was arrested, and testes enlarged (Eisler 1993). Zinc concentrations of 6.82 mg/kg ration suppressed rat growth and produced changes in the pancreas (Eisler 1993). A chronic NOAEL for laboratory rats of 160 mg/kg-bw/day is reported by Schlicker and Cox (1968 in Opresko et al. 1994). The TRVs for the brown lemming, arctic fox, and caribou are derived from this toxicity value.

3.3.15 Characterization of Effects

In this section toxicity information is presented for representative ecological receptors evaluated in the risk characterization section (Section 3.4). Potential impacts to aquatic receptors are evaluated by comparing exposure concentrations to TRVs. TRVs for the representative aquatic species are presented in Table 3-17. Potential impacts to terrestrial wildlife are evaluated for the representative species based on comparisons of estimated exposures to TRVs. Exposure to COCs for the representative terrestrial species is primarily through diet, which may include plants, fish, aquatic invertebrates, soils, and surface water. TRVs are derived for COCs in surface water and soil/sediment. TRVs for the representative bird species are presented in Table 3-18, and for the representative mammal species in Table 3-19.

3.3.15.1 Toxicity Reference Values. TRVs are derived by selecting toxicity values from the literature and extrapolating to the species of concern. UF and body scaling factors are used in the extrapolation process as described below.

- (1) The first step is to select an appropriate toxicity value from the scientific literature for each combination of chemical and representative or protected species. Test species most similar to the species of concern are preferred. A secondary emphasis is given to tests conducted over a significant portion of the animal's natural lifespan (e.g., chronic tests) when available.
- (2) The second step is to modify the toxicity value, if necessary, through application of UFs associated with the quality of toxicity data to derive a NOAEL (the highest concentration of a material in a toxicity test that has no statistically significant adverse effect on the exposed population of test organisms as compared with the next highest dose tested). If a chronic NOAEL or NOEL is available, it is used with an UF of one (i.e., no adjustment) because these values have the lowest uncertainty. If chronic data are unavailable, acute or subchronic toxicity data are modified by UFs to extrapolate to chronic effects. Based on Harding Lawson Associates (1992), the following strategy was derived for UFs for extrapolating study results to chronic NOAELs: 10 for chronic lowest observed effect level (LOEL) values, 10 for subchronic NOEL values, and 20 for subchronic LOEL values. LC_{50} and LD_{50} values are extrapolated to chronic NOAELs by a factor of 20.
- (3) The third step is applicable only to terrestrial receptors. This step extrapolates the estimated NOAEL from the test species to a NOAEL for the species of concern using a body scaling factor. Klaasen et al. (1986) have indicated that dose expressed per unit surface area may be more appropriate than dose per unit body weight. The underlying assumption is that a toxicant acts on a physiologic surface, and the toxic effect increases as the ratio of chemical to surface area increases. The scaling factor (SF) accounts for differences in the mass to surface area ratios between species. In this assessment the scaling factor is calculated

TABLE 3-17. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF AQUATIC ORGANISMS AT THE POINT LAY INSTALLATION

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	CONCENTRATION ($\mu\text{g/L}$)	TEST SPECIES	NOAEL UF	INTERSPECIES UF	PROTECTED SPECIES UF	TRV ($\mu\text{g/L}$)	REFERENCE
DRPH	nine-spined stickleback	LC ₅₀	38,600	fathead minnow	20	2	1	965	Hedke and Puglisi 1982
	<i>Daphnia</i> spp.	LOAEL	5,600	<i>D. pulex</i>	20	1	1	280	Hedke and Puglisi 1982
Iron	nine-spined stickleback	EPA chronic water quality criteria	1,000	all aquatic life	1	1	1	1,000	EPA 1986
	<i>Daphnia</i> spp.	EPA chronic water quality criteria	1,000	all aquatic life	1	1	1	1,000	EPA 1986
Manganese	nine-spined stickleback	28-day LC ₅₀	2,910	rainbow trout	20	2	1	73	Pickering et al. 1983
	<i>Daphnia</i> spp.	chronic reproductive impairment NOAEL	4,100	<i>Daphnia</i> spp.	10	1	1	410	Beisinger and Christensen 1972 in Lewis et al. 1979

TABLE 3-18. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE AND SENSITIVE SPECIES OF BIRDS AT THE POINT LAY INSTALLATION

CHEMICAL OF CONCERN ^a	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Iron	Lapland longspur	NOAEL; 28 day growth study	70.0	chicken	1	0.32	2	2	54.7	McGhee et al. 1965 in NAS 1980
	brant	NOAEL; 28 day growth study	70.0	chicken	1	1.18	2	2	14.8	McGhee et al. 1965 in NAS 1980
	glaucous gull	NOAEL; 28 day growth study	70.0	chicken	1	1.22	2	2	14.3	McGhee et al. 1965 in NAS 1980
	pectoral sandpiper	NOAEL; 28 day growth study	70.0	chicken	1	0.46	2	2	38.0	McGhee et al. 1965 in NAS 1980
	spectacled eider	NOAEL; 28 day growth study	70.0	chicken	1	1.20	2	2	14.6	McGhee et al. 1965 in NAS 1980
Lead	Lapland longspur	chronic growth and reproduction NOAEL	12	all birds	1	na	na	2	6	USACOE 1991
	brant	chronic growth and reproduction NOAEL	12	all birds	1	na	na	2	6	USACOE 1991
	glaucous gull	chronic growth and reproduction NOAEL	12	all birds	1	na	na	2	6	USACOE 1991
	pectoral sandpiper	chronic growth and reproduction NOAEL	12	all birds	1	na	na	2	6	USACOE 1991
	spectacled eider	chronic growth and reproduction NOAEL	12	all birds	1	na	na	2	6	USACOE 1991

^a Toxicity information was not available for the following COCs: benzene, toluene, ethylbenzene, tetrachloroethane, trichloroethane, and benzyl alcohol. See Section 3.4.3, Risk Characterization, for discussion of avian toxicities.

TABLE 3-18. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE AND SENSITIVE SPECIES OF BIRDS AT THE POINT LAY INSTALLATION (CONTINUED)

CHEMICAL OF CONCERN ^a	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Manganese	Lapland longspur	systemic LOAEL	250	chicken	10	0.32	2	2	19.5	NAS 1980
	brant	systemic LOAEL	250	chicken	10	1.18	2	2	5.3	NAS 1980
	glaucous gull	systemic LOAEL	250	chicken	10	1.22	2	2	5.1	NAS 1980
	pectoral sandpiper	systemic LOAEL	250	chicken	10	0.46	2	2	13.6	NAS 1980
	spectacled eider	systemic LOAEL	250	chicken	10	1.20	2	2	5.2	NAS 1980
Selenium	Lapland longspur	chronic reproduction NOAEL	0.5	mallard	1	0.32	2	2	0.4	Heinz et al. 1987 in Opresko et al. 1994
	brant	chronic reproduction NOAEL	0.5	mallard	1	1.18	2	2	0.1	Heinz et al. 1987 in Opresko et al. 1994
	glaucous gull	chronic reproduction NOAEL	0.5	mallard	1	1.22	2	2	0.1	Heinz et al. 1987 in Opresko et al. 1994
	pectoral sandpiper	chronic reproduction NOAEL	0.5	mallard	1	0.46	2	2	0.3	Heinz et al. 1987 in Opresko et al. 1994
	spectacled eider	chronic reproduction NOAEL	0.5	mallard	1	1.08	2	2	0.1	Heinz et al. 1987 in Opresko et al. 1994

^a Toxicity information was not available for the following COCs: benzene, toluene, ethylbenzene, tetrachloroethene, trichloroethane, trichloroethene, and benzyl alcohol. See Section 3.4.3, Risk Characterization, for discussion of avian toxicities.

TABLE 3-18. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE AND SENSITIVE SPECIES OF BIRDS AT THE POINT LAY INSTALLATION (CONTINUED)

CHEMICAL OF CONCERN ^a	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Zinc	Lapland longspur	subchronic dietary NOAEL	175	chicken	10	0.32	2	2	14	Roberson and Schaible 1960 in NAS 1980
	brant	subchronic dietary NOAEL	175	chicken	10	1.18	2	2	3.7	Roberson and Schaible 1960 in NAS 1980
	glaucous gull	subchronic dietary NOAEL	175	chicken	10	1.22	2	2	3.6	Roberson and Schaible 1960 in NAS 1980
	pectoral sandpiper	subchronic dietary NOAEL	175	chicken	10	0.46	2	2	9.5	Roberson and Schaible 1960 in NAS 1980
	spectacled eider	subchronic dietary NOAEL	175	chicken	10	1.20	2	2	3.6	Roberson and Schaible 1960 in NAS 1980
DRPH	Lapland longspur	decreased weight gain LOAEL	6,000	mallard	10	0.29	2	2	517	Hartung 1964
	brant	decreased weight gain LOAEL	6,000	mallard	10	1.07	2	2	140	Hartung 1964
	glaucous gull	decreased weight gain LOAEL	6,000	mallard	10	1.10	2	2	136	Hartung 1964
	pectoral sandpiper	decreased weight gain LOAEL	6,000	mallard	10	0.42	2	2	357	Hartung 1964
	spectacled eider	decreased weight gain LOAEL	6,000	mallard	10	1.08	2	2	139	Hartung 1964

^a Toxicity information was not available for the following COCs: benzene, toluene, ethylbenzene, tetrachloroethene, trichloroethane, trichloroethene, and benzyl alcohol. See Section 3.4.3, Risk Characterization, for discussion of avian toxicities.

TABLE 3-18. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE AND SENSITIVE SPECIES OF BIRDS AT THE POINT LAY INSTALLATION (CONTINUED)

CHEMICAL OF CONCERN ^a	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Xylenes (total)	Lapland longspur	Maximum dietary exposure	608	Japanese quail	10	0.60	2	2	25	Hill and Camardese 1986
	brant	Maximum dietary exposure	608	Japanese quail	10	2.16	2	2	7	Hill and Camardese 1986
	glaucous gull	Maximum dietary exposure	608	Japanese quail	10	2.23	2	2	7	Hill and Camardese 1986
	pectoral sandpiper	Maximum dietary exposure	608	Japanese quail	10	0.85	2	2	18	Hill and Camardese 1986
	spectacled eider	Maximum dietary exposure	608	Japanese quail	10	2.39	2	2	6	Hill and Camardese 1986

^a Toxicity information was not available for the following COCs: benzene, toluene, ethylbenzene, tetrachloroethene, trichloroethane, trichloroethene, and benzyl alcohol. See Section 3.4.3, Risk Characterization, for discussion of avian toxicities.

TABLE 3-19. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE POINT LAY INSTALLATION

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
DRPH	brown lemming	LD ₅₀	7,380	rat	20	0.60	2	1	308	Beck et al. 1982
	arctic fox	LD ₅₀	7,380	rat	20	2.70	2	1	68	Beck et al. 1982
	caribou	LD ₅₀	7,380	rat	20	7.24	2	1	25	Beck et al. 1982
Benzene	brown lemming	reproductive NOAEL	26.36	mouse	1	1.30	2	1	10	Nawrot and Staples 1979 in Opreko et al. 1994
	arctic fox	reproductive NOAEL	26.36	mouse	1	5.82	2	1	2	Nawrot and Staples 1979 in Opreko et al. 1994
	caribou	reproductive NOAEL	26.36	mouse	1	15.59	2	1	1	Nawrot and Staples 1979 in Opreko et al. 1994
Toluene	brown lemming	reproductive NOAEL	25.98	mouse	1	1.30	2	1	10	Nawrot and Staples 1979 in Opreko et al. 1994
	arctic fox	reproductive NOAEL	25.98	mouse	1	5.82	2	1	2	Nawrot and Staples 1979 in Opreko et al. 1994
	caribou	reproductive NOAEL	25.98	mouse	1	15.59	2	1	1	Nawrot and Staples 1979 in Opreko et al. 1994

**TABLE 3-19. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE POINT LAY INSTALLATION
(CONTINUED)**

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg- bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg- bw/day	REFERENCE
Ethylbenzene	brown lemming	LD ₅₀	5,460	rat	20	0.60	2	1	228	Budavari 1989
	arctic fox	LD ₅₀	5,460	rat	20	2.70	2	1	51	Budavari 1989
	caribou	LD ₅₀	5,460	rat	20	7.24	2	1	19	Budavari 1989
Xylenes (total)	brown lemming	LD ₅₀	4,300	rat	20	0.60	2	1	179	Clayton and Clayton 1981
	arctic fox	LD ₅₀	4,300	rat	20	2.70	2	1	40	Clayton and Clayton 1981
	caribou	LD ₅₀	4,300	rat	20	7.24	2	1	15	Clayton and Clayton 1981
Tetrachloroethene	brown lemming	hepatic NOAEL	1.4	mouse	1	1.30	2	1	0.53	Buben and O'Flaherty 1985 in Opreko et al. 1994
	arctic fox	hepatic NOAEL	1.4	mouse	1	2.70	2	1	0.12	Buben and O'Flaherty 1985 in Opreko et al. 1994
	caribou	hepatic NOAEL	1.4	mouse	1	7.24	2	1	0.05	Buben and O'Flaherty 1985 in Opreko et al. 1994

**TABLE 3-19. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE POINT LAY INSTALLATION
(CONTINUED)**

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Trichloroethane	brown lemming	LD ₅₀	580	rat	20	0.60	2	1	24	Sax and Lewis 1989
	arctic fox	LD ₅₀	580	rat	20	2.70	2	1	5	Sax and Lewis 1989
	caribou	LD ₅₀	580	rat	20	7.24	2	1	2	Sax and Lewis 1989
Trichloroethene	brown lemming	chronic reproductive NOAEL	300	rat	1	0.60	2	1	250	ATSDR 1991
	arctic fox	chronic reproductive NOAEL	300	rat	1	2.70	2	1	56	ATSDR 1991
	caribou	chronic reproductive NOAEL	300	rat	1	7.24	2	1	21	ATSDR 1991
Benzyl alcohol	brown lemming	LD ₅₀	1,230	rat	20	0.60	2	1	51	Sax and Lewis 1989
	arctic fox	LD ₅₀	1,230	rat	20	2.70	2	1	11	Sax and Lewis 1989
	caribou	LD ₅₀	1,230	rat	20	7.24	2	1	4	Sax and Lewis 1989
Iron	brown lemming	Acute LD ₅₀	1,000	rat	20	0.60	2	1	42	Shanas and Boyd 1969 in NAS 1980
	arctic fox	Acute LD ₅₀	1,000	rat	20	2.70	2	1	9	Shanas and Boyd 1969 in NAS 1980
	caribou	NOAEL; maximum tolerable dietary level	20	sheep	1	1.17	2	1	9	NAS 1980

**TABLE 3-19. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE POINT LAY INSTALLATION
(CONTINUED)**

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Lead	brown lemming	reproductive NOAEL	8	rat	1	0.60	2	1	7	Azar et al. 1973 in Opresko et al. 1994
	arctic fox	reproductive NOAEL	8	rat	1	2.70	2	1	1	Azar et al. 1973 in Opresko et al. 1994
	caribou	chronic reproductive NOAEL	0.4	sheep	1	1.17	2	1	0.2	Fick et al. 1976 in NAS 1980
Manganese	brown lemming	chronic systemic NOAEL	930	rat	1	0.60	2	1	780	Hejtmannick et al. in ATSDR 1990
	arctic fox	chronic systemic NOAEL	930	rat	1	2.70	2	1	170	Hejtmannick et al. 1987 in ATSDR 1990
	caribou	NOAEL; maximum tolerable dietary level	40	sheep	1	1.17	2	1	17	NAS 1980
Selenium	brown lemming	chronic reproductive LOAEL	0.75	mouse	10	1.30	2	1	0.03	Schroeder and Mitchner 1971 in Opresko et al. 1994
	arctic fox	chronic reproductive LOAEL	0.75	mouse	10	5.82	2	1	0.01	Schroeder and Mitchner 1971 in Opresko et al. 1994
	caribou	chronic reproductive LOAEL	0.75	mouse	10	15.59	2	1	0.002	Schroeder and Mitchner 1971 in Opresko et al. 1994

**TABLE 3-19. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE POINT LAY INSTALLATION
(CONTINUED)**

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg- bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg- bw/day	REFERENCE
Zinc	brown lemming	chronic reproductive LOAEL	160	rat	1	0.60	2	1	133	Schlicker and Cox 1968 in Opresko et al. 1994
	arctic fox	chronic reproductive LOAEL	160	rat	1	2.70	2	1	30	Schlicker and Cox 1968 in Opresko et al. 1994
	caribou	chronic reproductive LOAEL	160	rat	1	7.24	2	1	11	Schlicker and Cox 1968 in Opresko et al. 1994

using the following equation (Mantel and Schneiderman 1975) (scaling factors are presented in Appendix F):

$$SF = (\text{weight of representative species/weight of test species})^{1/3}.$$

- (4) A UF of 2 is used to account for interspecies variation in sensitivity. This value is based on the methodology used in Harding Lawson Associates (1992).
- (5) A UF of 2 was used to account for additional sensitivity of state and/or federally protected species. This value is based on Harding Lawson Associates (1992). Migratory birds are federally protected and include all the representative avian and protected species (i.e., spectacled eider) selected for this assessment.

The methods of calculating the TRV for the terrestrial and aquatic receptors are as follows:

TERRESTRIAL:

- a) Convert test dose to a NOAEL:

$$\text{DOSE} \div \text{NOAEL UF} = \text{Estimated NOAEL}$$

- b) Adjust for body size difference between test species and ROC:

$$\text{Estimated NOAEL} \div \text{SCALING FACTOR} = \text{Scaled, estimated NOAEL}$$

- c) Adjust for interspecies differences:

$$\text{Scaled, estimated NOAEL} \div \text{INTERSPECIES UF} = \text{Species-specific, scaled, estimated NOAEL.}$$

- D) Account for protected species status:

$$\text{Species-specific, scaled, estimated NOAEL} \div \text{PROTECTED SPECIES UF} = \text{TRV}$$

AQUATIC:

$$\text{EFFECTIVE CONCENTRATION} \div \text{NOAEL UF} \div \text{INTERSPECIES UF} = \text{TRV}$$

3.4 RISK CHARACTERIZATION FOR ECOLOGICAL RECEPTORS

HQs, for the representative species, are presented in this section. Potential risks to plants are evaluated based on the contaminant concentrations in the soil/sediment and information from the literature. Potential risks to aquatic organisms, birds, and mammals are estimated by comparing estimated exposures to TRVs (i.e., quotient method). The quotient method divides the estimated exposure concentration by the associated TRV to derive the HQ. If the HQ is less than 1.0 adverse effects are not expected. Conversely, if the HQ is equal to or greater than 1.0, a potential for adverse effects exists. The confidence level of the risk estimate is increased as the magnitude of the HQ departs from 1.0. For example, there is greater confidence in a risk estimate where the HQ is 0.1 or 10, than in a HQ such as 0.9 or 1.1. The confidence level is also

dependent on the uncertainty associated with the estimated exposure and the TRV for a given chemical-receptor combination.

The characterization of risk focuses on the assessment endpoints. These endpoints are selected and discussed in keeping with the Framework for Ecological Risk Assessment guidance (EPA 1992a). The assessment endpoints for the Point Lay ERA are changes in:

- the populations of the plant representative species (*Carex* spp., *Salix* spp., *Eriophorum* spp., and *Vaccinium* spp.);
- the populations of aquatic representative species (*Daphnia* spp. and nine-spined stickleback);
- the populations of avian representative and sensitive species (Lapland longspur, brant, glaucous gull, pectoral sandpiper, and spectacled eider); and
- the populations of mammalian representative species (brown lemming, arctic fox, and barren-ground caribou).

The measurement endpoints used to evaluate potential changes in populations of the representative species were based on the endpoints used to derive the TRVs. These endpoints included physiological effects, growth, reproduction, and mortality.

Potential ecological risks to representative species are presented in the following sections: Section 3.4.1 addresses plants; Section 3.4.2 considers aquatic organisms; Section 3.4.3 addresses birds; and Section 3.4.4 discusses mammals. A discussion of potential future risks to ecological receptors is presented in Section 3.4.5. Toxicity information and the HQs that represent potential risk estimates are summarized in Tables 3-20 through 3-22.

3.4.1 Potential Risks to Representative Species of Plants

In determining the risks to plants at the Point Lay sites, a qualitative comparison was made of soil and surface water contaminant concentrations and plant toxicity information in the literature.

Table 3-20 summarizes these comparisons. There is a great deal of uncertainty in this phase of the assessment because of the differences in degree of uptake between plant species (Walker et al. 1978); however, the concentrations of contaminants onsite can be compared on the level of orders of magnitude, which can identify broad trends and determine whether a potential risk may exist.

Information generally is lacking concerning the toxicity of the COCs at Point Lay and how they relate to the representative species of plants. As a result, when comparisons of TRVs for site-specific species and chemicals are not possible, comparisons of related chemicals with other plant species are made.

TABLE 3-20. COMPARISON OF CONCENTRATIONS OF POTENTIAL CONTAMINANTS TO TOXICITY INFORMATION FOR PLANTS AT THE POINT LAY INSTALLATION

CHEMICAL (COC media)	PLANT	EXPOSURE LEVEL	EFFECT ON PLANT	POINT LAY EXPOSURE	REFERENCE
IRON (COC in water)	rice	100,000-500,000 µg/L; >500,000 µg/L	toxic; highly toxic	14,800 µg/L	USACOE 1991
LEAD (COC in soil/sediment)	weed spp.	500 mg/kg in soil	reduced pollen germination	60.6 mg/kg	USACOE 1991
	weed spp.	46 mg/kg	normal germination	60.6 mg/kg	USACOE 1991
MANGANESE (COC in water)	duckweed	31,000 µg/L in water	EC ₅₀	1,087 µg/L	USACOE 1991
SELENIUM (COC in soil/sediment)	unspecified crop species	5 to 10 mg/kg	phytotoxic	17 mg/kg	Kabata-Pendias and Pendias 1984
ZINC (COC in soil/sediment)	several spp.	270 mg/kg (avg) in soil	phytotoxic	95.5 mg/kg	USACOE 1991
VOCs (COCs as DRPH in water; as DRPH, BTEX, and other reported VOCs in soil/sediment)	green algae	4,600 µg/L for ethylbenzene 2,290 µg/L for methylene chloride, in water	EC ₅₀	DRPH = 461 µg/L benzene = 0.13 toluene = 0.7 ethylbenzene = 1.66 xylenes = 5.68 (BTEX in mg/kg)	USACOE 1991

TABLE 3-21. RISK CHARACTERIZATION OF REPRESENTATIVE SPECIES OF AQUATIC ORGANISMS AT THE POINT LAY INSTALLATION

SPECIES	ESTIMATED EXPOSURE CONCENTRATION (µg/liter)	TRV (µg/liter)	HAZARD QUOTIENT
IRON			
<i>Daphnia</i> spp.	14,800	1,000	15
nine-spined stickleback	14,800	1,000	15
MANGANESE			
<i>Daphnia</i> spp.	1,087	410	2.7
nine-spined stickleback	1,087	73	15
DRPH			
<i>Daphnia</i> spp.	461	280	1.6
nine-spined stickleback	461	965	0.5

TABLE 3-22. RELATIVE TOXICITY RANKINGS FOR COMPARISON OF MAMMALIAN AND AVIAN TOXICITY

COC	STUDY TYPE*	DOSE* mg/kg	RELATIVE TOXICITY
benzene	oral rat LD ₅₀	3,400	slightly toxic
toluene	oral rat LD ₅₀	5,000	slightly toxic
ethylbenzene	oral rat LD ₅₀	5,460	relatively nontoxic
tetrachloroethene	oral rat LD ₅₀	8,850	relatively nontoxic
trichloroethane	oral rat LD ₅₀	580	moderately toxic
trichloroethene	oral rat LD ₅₀	3,670	slightly toxic
benzyl alcohol	oral rat LD ₅₀	1,230	slightly toxic

* Sax and Lewis 1989

As seen in Table 3-20, the concentrations of iron and manganese in the surface water at Point Lay are both below the reported toxicity values. Zinc concentrations in soil/sediment are also below the reported toxicity value. The concentrations of lead and selenium in soil/sediment indicate that plants may be subject to adverse effects as a result of exposure to these COCs. Note that the lead concentration is driven by elevated levels at two sample locations, S03 (92 mg/kg) and S08 (195 mg/kg) at the Garage (SS06), and the elevated selenium concentration is found at one sample location, S06 (17 mg/kg) at the Crushed Drum Area (SS08). Kabata-Pendias and Pendias (1984) report phytotoxicity levels of lead in soil ranging from 100 to 400 mg/kg and selenium phytotoxicity in soil at concentrations of 5 to 10 mg/kg. The number of samples (both the total number of samples analyzed for metals [5] and with detected concentrations of lead [3] and selenium [1]) introduces uncertainty concerning the extent of potential contamination. The lead and selenium concentrations reported in soil/sediment may pose a low level of risk to plants, but the uncertainty regarding these risk estimates is large. Refer to Section 3.5, Uncertainty Analysis, for further discussion.

The risk posed to plants by VOCs, including components of DRPH and BTEX, is not expected to be significant. Toxicity values for terrestrial plants were not available in the literature, but VOCs are not expected at significant levels in most plants because of their volatility, absorption to soil particles, metabolism, or degradation rates in soil (Kostecki and Calabrese 1989).

Considering the discussions above, the overall risk to plants from COCs at the Point Lay sites is not significant.

3.4.2 Potential Risks to Representative Species of Aquatic Organisms

Estimates of exposure for aquatic organisms are based on the average concentrations of each COC in surface water samples (Section 3.1). The TRVs for aquatic species are presented in Table 3-17. The HQ is calculated by dividing the estimated exposure concentration by the TRV. Table 3-21 presents the results of the risk characterization for aquatic organisms. The following paragraph summarizes the potential risks to aquatic organisms from iron, the only COC identified in surface water.

The HQs for iron in surface water were 15 for *Daphnia* spp. and the nine-spined stickleback. These HQs indicate potential risk to aquatic organisms from iron concentrations in surface water. These risks are mitigated, however, by the fact that "total" metal concentrations were used to estimate risk and iron is an essential nutrient. The HQs are based on total concentrations of iron (i.e., 14,800 µg/L), but the dissolved concentrations are approximately one-half (7,810 µg/L) of the total concentrations. In addition, the site-specific risks at the Point Lay facility are likely to be lower than the risk estimates presented, due to the nature of the drainage areas at the installation. The surface water sample collected from the Deactivated Landfill (SW04 and SW08, duplicate samples), where the elevated iron concentration is located, are not significant routes of exposure for the nine-spined stickleback, as they are collected from a shallow drainage ditch that does not provide habitat for the stickleback. As a result, the only representative aquatic species that are exposed in surface water at the Deactivated Landfill are *Daphnia* spp. There is uncertainty associated with the evaluation of metals in surface waters, which is discussed in Section 3.5, Uncertainty Analysis.

Manganese HQs are elevated; HQs are 2.7 for *Daphnia* spp. and 15 for the nine-spined stickleback. These risk estimates may be mitigated by several factors: manganese is an essential nutrient, there are a limited number of samples, and the TRVs calculated for the aquatic species (410 and 73 $\mu\text{g/L}$ for *Daphnia* spp. and the stickleback, respectively) are below the maximum background concentration of 510 $\mu\text{g/L}$. In addition, the sample location that drives the elevated manganese concentration (1,700 $\mu\text{g/L}$ at SW02 at the Garage, SS06) is in an ephemeral tundra pond. The pond is likely to freeze to the bottom and thus not provide suitable habitat for the nine-spined stickleback.

The DRPH HQ for *Daphnia* spp. is slightly elevated (1.6), largely due to the high non-detect levels ($<1,000 \mu\text{g/L}$), rather than extensive DRPH contamination. The two water samples with detected DRPH were 2SW09 and 2SW10 at the Deactivated Landfill, and the average DRPH in the detected samples was 210 $\mu\text{g/L}$. This is below the DRPH TRV of 280 $\mu\text{g/L}$ for *Daphnia* spp. The two other detections of DRPH in surface water were reported by the laboratory to not be consistent with middle distillate fuel (i.e., may be biogenic in nature).

Based on the factors described above, the risks to the representative aquatic organisms at the Point Lay installation are not estimated to be significant.

3.4.3 Potential Risks to Representative Species of Birds

The HQs for the pectoral sandpiper are elevated for selenium (HQ = 2) and zinc (HQ = 2): the calculated HQs for all other representative avian species and COCs are below 1. The avian HQs are presented in Table 3-23. HQs were not calculated for several of the organic COCs because no toxicity information was available for avian species; however, a qualitative discussion of these COCs follows.

The pectoral sandpiper forages in surface soil, and its incidental ingestion of soil/sediment is relatively high (estimated to be about 18 percent). Realistically, the likelihood of the sandpiper repeatedly foraging at the one sample location where selenium was detected (S06 at the Crushed Drum Area, SS08) or at the one location where zinc was elevated above background levels (the SD04 and SD08 duplicate sample at the Deactivated Landfill, LF01) is very low. This mitigates the HQ, and results in a non-significant risk estimate for the pectoral sandpiper's exposure to selenium and zinc.

Quantitative evaluations of benzene, toluene, 1,4-dichlorobenzene, 4-methylphenol, and 1,2,4-trimethylbenzene were not possible because avian toxicity studies for these COCs were not available. A discussion of the relative toxicity of these chemicals provides a basis for making qualitative statements concerning their toxicity to avian species. Smith (1987) provides the following ranking of relative toxicities based on median lethal doses (LD_{50} s).

- I. Extremely toxic ($\text{LD}_{50} \leq 40 \text{ mg/kg}$)
- II. Highly toxic ($\text{LD}_{50} 41\text{-}200 \text{ mg/kg}$)
- III. Moderately toxic ($\text{LD}_{50} 201\text{-}1,000 \text{ mg/kg}$)
- IV. Slightly toxic ($\text{LD}_{50} 1,001\text{-}5,000 \text{ mg/kg}$)
- V. Relatively nontoxic ($\text{LD}_{50} > 5,000 \text{ mg/kg}$)

TABLE 3-23. HAZARD QUOTIENTS FOR REPRESENTATIVE BIRDS AND MAMMALS AT THE POINT LAY INSTALLATION

CHEMICAL OF CONCERN	ESTIMATED EXPOSURE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
IRON			
Lapland longspur	6×10^{-1}	55	1×10^{-2}
brant	2×10^{-2}	15	2×10^{-3}
glaucous gull	8×10^{-3}	14	6×10^{-4}
pectoral sandpiper	2×10^{-0}	38	5×10^{-2}
spectacled eider	2×10^{-1}	15	1×10^{-2}
brown lemming	9×10^{-1}	42	2×10^{-2}
arctic fox	1×10^{-2}	9	1×10^{-3}
caribou	9×10^{-3}	9	1×10^{-3}
LEAD			
Lapland longspur	9×10^{-2}	6	1×10^{-2}
brant	8×10^{-3}	6	1×10^{-3}
glaucous gull	2×10^{-3}	6	4×10^{-4}
pectoral sandpiper	2×10^0	6	3×10^{-1}
spectacled eider	7×10^{-2}	6	1×10^{-2}
brown lemming	1×10^0	7	2×10^{-1}
arctic fox	9×10^{-4}	1	9×10^{-4}
caribou	3×10^{-4}	0.2	2×10^{-3}
MANGANESE			
Lapland longspur	4×10^{-2}	20	2×10^{-3}
brant	2×10^{-3}	5	4×10^{-4}
glaucous gull	6×10^{-4}	5	1×10^{-4}
pectoral sandpiper	1×10^{-1}	14	1×10^{-2}
spectacled eider	1×10^{-2}	5	3×10^{-3}
brown lemming	7×10^{-2}	780	9×10^{-5}
arctic fox	9×10^{-4}	170	5×10^{-6}
caribou	7×10^{-4}	17	4×10^{-5}

TABLE 3-23. HAZARD QUOTIENTS FOR REPRESENTATIVE BIRDS AND MAMMALS AT THE POINT LAY INSTALLATION (CONTINUED)

CHEMICAL OF CONCERN	ESTIMATED EXPOSURE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
SELENIUM			
Lapland longspur	2×10^{-2}	0.4	5×10^{-2}
brant	2×10^{-3}	0.1	2×10^{-2}
glaucous gull	7×10^{-4}	0.1	7×10^{-3}
pectoral sandpiper	5×10^{-1}	0.3	2×10^0
spectacled eider	2×10^{-2}	0.1	2×10^{-1}
brown lemming	3×10^{-1}	0.03	9×10^0
arctic fox	3×10^{-4}	0.01	3×10^{-2}
caribou	9×10^{-5}	0.002	4×10^{-2}
ZINC			
Lapland longspur	2×10^0	14	1×10^{-1}
brant	2×10^{-2}	4	5×10^{-3}
glaucous gull	5×10^{-3}	4	1×10^{-3}
pectoral sandpiper	2×10^1	10	2×10^0
spectacled eider	6×10^{-1}	4	1×10^{-1}
brown lemming	3×10^1	133	2×10^{-1}
arctic fox	1×10^{-3}	30	5×10^{-5}
caribou	8×10^{-4}	11	8×10^{-5}
DRPH			
Lapland longspur	4×10^0	520	7×10^{-3}
brant	3×10^{-5}	140	3×10^{-3}
glaucous gull	1×10^{-1}	140	8×10^{-4}
pectoral sandpiper	9×10^1	360	2×10^{-1}
spectacled eider	3×10^0	140	2×10^{-2}
brown lemming	5×10^1	310	2×10^{-1}
arctic fox	4×10^{-2}	68	6×10^{-4}
caribou	2×10^{-2}	25	6×10^{-4}

TABLE 3-23. HAZARD QUOTIENTS FOR REPRESENTATIVE BIRDS AND MAMMALS AT THE POINT LAY INSTALLATION (CONTINUED)

CHEMICAL OF CONCERN	ESTIMATED EXPOSURE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
BENZENE			
Lapland longspur	3×10^{-3}	NA	NC
brant	3×10^{-5}	NA	NC
glaucous gull	7×10^{-6}	NA	NC
pectoral sandpiper	4×10^{-2}	NA	NC
spectacled eider	1×10^{-3}	NA	NC
brown lemming	6×10^{-2}	10	6×10^{-3}
arctic fox	2×10^{-6}	2	9×10^{-7}
caribou	1×10^{-6}	1	1×10^{-6}
TOLUENE			
Lapland longspur	8×10^{-3}	NA	NC
brant	1×10^{-4}	NA	NC
glaucous gull	3×10^{-5}	NA	NC
pectoral sandpiper	1×10^{-1}	NA	NC
spectacled eider	3×10^{-3}	NA	NC
brown lemming	2×10^{-1}	10	2×10^{-2}
arctic fox	1×10^{-5}	2	5×10^{-6}
caribou	5×10^{-6}	1	5×10^{-6}
ETHYLBENZENE			
Lapland longspur	1×10^{-2}	NA	NC
brant	3×10^{-4}	NA	NC
glaucous gull	7×10^{-5}	NA	NC
pectoral sandpiper	2×10^{-1}	NA	NC
spectacled eider	5×10^{-3}	NA	NC
brown lemming	2×10^{-1}	230	1×10^{-3}
arctic fox	2×10^{-5}	51	5×10^{-7}
caribou	1×10^{-5}	19	6×10^{-7}

TABLE 3-23. HAZARD QUOTIENTS FOR REPRESENTATIVE BIRDS AND MAMMALS AT THE POINT LAY INSTALLATION (CONTINUED)

CHEMICAL OF CONCERN	ESTIMATED EXPOSURE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
XYLENES (total)			
Lapland longspur	6×10^{-2}	26	2×10^{-3}
brant	1×10^{-3}	7	1×10^{-4}
glaucous gull	3×10^{-4}	7	4×10^{-5}
pectoral sandpiper	9×10^{-1}	18	5×10^{-2}
spectacled eider	2×10^{-2}	6	4×10^{-3}
brown lemming	1×10^0	180	7×10^{-3}
arctic fox	8×10^{-5}	40	2×10^{-6}
caribou	4×10^{-5}	15	3×10^{-6}
TETRACHLOROETHENE			
Lapland longspur	2×10^{-2}	NA	NC
brant	3×10^{-4}	NA	NC
glaucous gull	7×10^{-5}	NA	NC
pectoral sandpiper	3×10^{-1}	NA	NC
spectacled eider	9×10^{-3}	NA	NC
brown lemming	5×10^{-1}	0.53	9×10^{-1}
arctic fox	2×10^{-5}	0.12	2×10^{-4}
caribou	1×10^{-5}	0.05	3×10^{-4}
TRICHLOROETHANE			
Lapland longspur	3×10^{-3}	NA	NC
brant	3×10^{-5}	NA	NC
glaucous gull	7×10^{-6}	NA	NC
pectoral sandpiper	5×10^{-2}	NA	NC
spectacled eider	1×10^{-3}	NA	NC
brown lemming	7×10^{-2}	24	3×10^{-3}
arctic fox	2×10^{-6}	5	4×10^{-7}
caribou	2×10^{-6}	2	7×10^{-7}

TABLE 3-23. HAZARD QUOTIENTS FOR REPRESENTATIVE BIRDS AND MAMMALS AT THE POINT LAY INSTALLATION (CONTINUED)

CHEMICAL OF CONCERN	ESTIMATED EXPOSURE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
TRICHLOROETHENE			
Lapland longspur	2×10^{-3}	NA	NC
brant	2×10^{-5}	NA	NC
glaucous gull	5×10^{-6}	NA	NC
pectoral sandpiper	3×10^{-2}	NA	NC
spectacled eider	7×10^{-4}	NA	NC
brown lemming	4×10^{-2}	250	1×10^{-4}
arctic fox	2×10^{-6}	56	3×10^{-8}
caribou	1×10^{-6}	21	5×10^{-8}
BENZYL ALCOHOL			
Lapland longspur	9×10^{-2}	NA	NC
brant	5×10^{-4}	NA	NC
glaucous gull	8×10^{-5}	NA	NC
pectoral sandpiper	1×10^0	NA	NC
spectacled eider	3×10^{-2}	NA	NC
brown lemming	2×10^0	51	4×10^{-2}
arctic fox	1×10^{-5}	11	1×10^{-6}
caribou	3×10^{-5}	4	7×10^{-6}

Table 3-22 shows the relative toxicity rankings of benzene, toluene, ethylbenzene, tetrachloroethene, trichloroethane, trichloroethene, and benzyl alcohol (Smith 1987).

Using the relative toxicities of these COCs, the exposure estimates for avian and mammalian species, and the HQs that were calculated for mammalian species (see Table 3-23 for exposure estimates and HQs), it is possible to make inferences concerning the potential risk to avian species.

There is limited information available on the relative toxicologic sensitivities of birds compared to mammals. Based upon a review of the species and chemicals tested (Smith 1987, Hudson et al. 1979, and Tucker and Leitzke 1979), it appears in general that avian and mammalian sensitivities (via oral exposure) fall within the same range, with birds being slightly more sensitive than mammals. There are, of course, exceptions to this general observation and for a number of chemicals mammals are more sensitive than birds. For cases where birds are more sensitive, most avian toxicity values fall well within one order of magnitude of the mammalian toxicity values.

As noted, avian toxicity values are not available for some COCs in this ERA. However, based on the information presented above, these chemicals are not expected to be significantly more toxic to birds than to mammals. Birds are not expected to be at risk given that there are no HQs for these COCs above one for mammals, and the estimated exposures for birds are sufficiently lower than those for mammals to offset the possibility that some birds may be more sensitive than mammals to these selected COCs. This qualitative discussion, however, introduces additional elements of uncertainty to the risk assessment. See Section 3.5, Uncertainty Analysis, for more discussion of this topic.

Based on the above discussions and the HQs presented in Table 3-23, the risk estimates for the avian representative species at the Point Lay installation are not significant.

3.4.4 Potential Risks to Representative Species of Mammals

Mammalian HQs are presented in Table 3-23. HQs for the brown lemming, arctic fox, and the barren-ground caribou are below 1.0 for all COCs with the exception of the brown lemmings' selenium HQ of 9. This could indicate potential risk to the brown lemming, but this is mitigated because selenium was detected at only one location, sample S06 at the Crushed Drum Area, site SS08. It is unlikely that the population of lemmings at the Point Lay installation would be exposed to the selenium concentration at that one sample location, although the one reported concentration (17 mg/kg) was used in calculating the HQ. Given the realistic distribution of the brown lemming population and the probability for exposure to selenium at the one detected location, the risk to the lemming posed by selenium is not significant.

Based on the calculated HQs and the discussion above, the estimated risks for mammals at the Point Lay installation are not significant. As noted in the risk characterizations for aquatic and avian species, the analysis for metals is limited and introduces uncertainty to the risk estimates. Refer to Section 3.5, Uncertainty Analysis, for a more detailed discussion of this topic.

3.4.5 Potential Future Risks

Estimates of future risk at the Point Lay installation are based on the assumption that the gravel pads will remain in place and that the sites will remain suitable habitat for the representative species. Future risks at the Deactivated Landfill (LF01), Garage (SS06), Drainage Pathway from POL Tanks (SS07), and Crushed Drum Area (SS08) are expected to be as low as, or lower than, current risks (i.e., not significant) because the exposure pathways are not likely to change, and the concentrations of COCs are likely to diminish over time.

3.5 ECOLOGICAL RISK ASSESSMENT UNCERTAINTY ANALYSIS

As with any risk assessment, there is great uncertainty associated with the estimates of ecological risk for the sites at the Point Lay installation. The risk estimates are based on a number of assumptions regarding exposure and toxicity. In general, the primary sources of uncertainty are the following:

- Environmental Sampling and Analysis;
- Selection of COCs;
- Selection of Representative Species;
- Exposure Parameter Estimation; and
- Toxicological Data.

A complete understanding of the uncertainties associated with risk estimates is critical to placing the predicted risks in proper perspective. The most significant sources of uncertainty associated with the estimates of risk for the Point Lay installation sites are summarized in the following sections.

3.5.1 Environmental Sampling and Analysis

The principal source of uncertainty in the analytical data (for the ERA) stems from the sampling approach and the subsequent calculation of exposure concentrations. Sampling at the Point Lay installation was conducted in a systematic manner, designed to characterize localized contaminated areas or "hot spots". Therefore, the average concentrations of COCs tend to be biased high because sampling was generally concentrated in areas of the installation where significant contamination exists or was suspected. In order to partially compensate for this non-random sampling methodology in the calculation of exposure concentrations, the exposure assessment used the average concentration of COCs across the site.

The methods of calculating the average concentrations were the same for organic and inorganic data. In calculating the average concentration of chemicals at the site, non-detected chemicals were entered at one-half of the quantitation limit, as per EPA guidance (EPA 1989a). The use of total metal concentrations in surface water to estimate risk is a conservative approach because dissolved metal concentrations are generally significantly less than total metal concentrations. Therefore, the average concentrations of total metals used to estimate exposure in surface water may overestimate potential risk.

The number of metal analyses is a factor that also contributes to uncertainty. Of the nineteen surface water samples, three were analyzed for metals, and the soil/sediment samples were analyzed for metals in five of fifty-five instances. The selection of the inorganic COCs was based on these analyses and may under- or overestimate the true nature and extent of metals contamination at the Point Lay installation. It is difficult to assess the impact that this uncertainty has on the overall risk estimates, although the available data indicate that metals contamination is not likely to present risks at Point Lay.

In addition, the sample quantitation limits for several metals were higher than the action levels used to screen the chemicals. For example, the sample quantitation limit of aluminum was 100 µg/L, and the action level is 87 µg/L. Therefore, non-detected concentrations of aluminum and other metals, including beryllium, cadmium, copper, chromium, lead, selenium, silver, and thallium may be present in quantities sufficient to elicit adverse effects in aquatic organisms. This probably contributes a low level of uncertainty to the overall risk estimate because surface water pathways at the Point Lay installation are not likely to be significant routes of exposure to representative species other than *Daphnia* spp.

There is uncertainty inherent in using measurements of DRPH, GRPH, and RRPH for risk assessments. The analytical techniques are not specific to petroleum (i.e., they detect other organics, including naturally-occurring ones) (Von Burg 1993). Moreover, the toxicity of these groups of petroleum hydrocarbons is determined by the toxicity of their individual constituents. When petroleum compounds are released to the environment, they tend to weather or transform readily. For example, the lighter fractions (such as BTEX) will volatilize to the atmosphere more readily than the heavier fractions (such as decane, pyrene, or benzo(a)pyrene). The lighter fractions are thought to be the more toxic (Wong et al. 1981; O'Brien 1978; Kauss and Hutchinson 1975; Soto et al. 1975). Therefore, the toxicity of DRPH, GRPH, and RRPH is expected to change over time depending upon the attenuation mechanisms occurring in the environment. As a result, the toxicity of the petroleum hydrocarbons detected at the Point Lay sites is unknown. Use of toxicity values reported in the literature probably contributes to an overestimation of the risk because it is likely that the most toxic components of the mixtures detected have volatilized to the atmosphere over time.

3.5.2 Selection of Chemicals for Evaluation

The selection of COCs in the ERA was based upon a comparison to background concentrations and action levels, and an evaluation of the frequency of detection. For certain chemicals, no action levels were available, and action levels for related compounds were used. This introduces some uncertainty into the risk assessment as actual toxicity may be different from the toxicity of the surrogate chemical. Overall, however, the process provided a conservative screen of COCs, and it is unlikely that any chemicals presenting an ecological risk were omitted.

3.5.3 Selection of Representative Species

The selection of representative species in the ERA introduces some uncertainty into the risk estimates. No site-specific biological surveys were conducted at the Point Lay installation, with the exception of a survey for spectacled and Steller's eiders (Alaska Biological Research 1994).

As a result, it is not known whether or how often the representative species are actually found at the site; however, the uncertainty introduced into the risk estimate by this route is likely to be low. The purpose of ERAs is not to survey the biota at a site, but to estimate the risks to species that may inhabit the area. Surrogate species are commonly used, and even if the representative species do not reside specifically at the Point Lay installation, the risk estimates in this report provide a sound measure of the potential risks to the species that do inhabit the area.

3.5.4 Exposure Assessment

Exposures were estimated using literature-based life history information for the selected representative species. There is moderate uncertainty associated with the exposure information. Food and water ingestion rates were not available for some animals and had to be estimated from regression equations. Incidental ingestion of soils and sediments may occur while animals are foraging, and it is uncertain how much is actually ingested. In addition, there is uncertainty associated with the habitat at the site. Samples were collected around buildings and other structures that are likely to provide habitat of limited quality. As a result, this tends to overestimate exposure. Further, there are significant uncertainties associated with the estimates of how extensively a receptor will use the site, which were based on home range information. As noted in the discussion of Estimation of Percent Ingested Onsite, Section 3.2.7.2, the conversion of population density values as substitutes for home ranges adds uncertainty to the risk assessment. The conversion was necessary because home range data are lacking for some of the representative species.

There is some uncertainty associated with the diet compositions estimated from the literature. A good example of this type of uncertainty is the unpredictable fluctuation in the populations of the brown lemmings and their predators (i.e., arctic fox, glaucous gull). As the numbers of prey increase, predator populations may experience numerical and density increases well beyond the values reported in the literature. When prey populations decrease, predation pressure can shift to diet items not considered "normal", that do not represent dietary intakes reported in the literature. Wildlife and their interactions with the environment around them are dynamic. Stochastic events, natural or anthropogenic, may cause behavior and/or habits to differ markedly from the "expected or norm". Deviations from typical behavior cause uncertainty when evaluating wildlife and ecosystems.

There is uncertainty associated with exposure estimates for plants. Plant uptake of COCs was derived from a regression equation using the K_{ow} of the COC (Table 3-6). This calculation estimates the concentration of chemicals in the vegetative portion of plants. Actual concentrations of the COC in plant tissue will vary depending upon actual chemical uptake, species of plant, and other site-specific factors (such as soil organic carbon). It is important to note that screening level tissue concentrations in plants were not available for comparison with these estimated concentrations. The overall effect of this source of uncertainty in the risk assessment is low, as is the ecological risk to plants.

The only component in the diet of representative species evaluated quantitatively was the ingestion of plants. Ingestion of animal prey (e.g., the diet of the arctic fox and the insectivorous

portion of some avian diets) was not quantified. This may slightly underestimate risk for species that rely on animal items in their diet.

3.5.5 Toxicological Data

One of the largest sources of uncertainty in risk assessment is from the toxicological data. Often there are not relevant studies for the specific representative species or endpoints. As a result, extrapolations are made, which introduce uncertainty into the risk estimate. These extrapolations incorporate UFs into the calculation of TRVs. The purpose of the UFs is to incorporate some margin of error into the risk estimate, in order to arrive at a "safe" level of exposure to which onsite exposure concentrations may be compared. These techniques introduce into the risk assessment a tendency to overestimate rather than underestimate the risk, as conservative estimates were made in estimating toxicity values.

For some chemicals, no toxicity information was available (e.g., avian toxicity values for BTEX and several VOCs for birds). As a result, these compounds were not evaluated quantitatively in the risk assessment, and the risk may be somewhat underestimated. Based on the low concentrations and low frequency of detection of these compounds (as discussed in Section 3.1), the uncertainty associated with this factor is low.

Toxicity values for plants, water, soils, and sediments are based on literature values. Toxicity in soils and sediments is affected by the bioavailability of a given chemical. Toxicity of metals in water is based, in part, upon the speciation of the element. As a result, site-specific bioavailability or toxicity may differ from that in the studies used to estimate potential toxic effects. Therefore, actual toxicities of chemicals at the Point Lay sites may be different from the values reported in the literature. In addition, the sensitivity of receptors on site may be different from the sensitivity of the species reported in the literature.

There is a great deal of uncertainty in assessing the toxicity of a mixture of chemicals. In this ERA, the effects of exposure from each contaminant have been considered separately. These substances occur together at the site, and organisms may be exposed to mixtures of the chemicals. Prediction of how these mixtures of toxicants will interact must be based on an understanding of the mechanisms of such interactions. Interactions of the individual components of chemical mixtures may occur during absorption, distribution, metabolism, excretion, or activity at the receptor site. Individual compounds may interact chemically, yielding a new toxic component or causing a change in the biological availability of an existing component, or may interact by causing different effects at different receptor sites. Suitable data are not currently available to characterize the effects of chemical mixtures rigorously, so chemicals present at the site were evaluated independently. This approach of assessing risk associated with mixtures of chemicals does not account for any additive, synergistic, or antagonistic interactions among the chemicals considered. However, as discussed in Section 3.6, the risk assessment yielded a low potential for ecological risks, and it is unlikely that additive effects of chemicals are a concern.

3.6 SUMMARY OF ECOLOGICAL RISK

The potential risks to ecological receptors are summarized in this section based on the information presented in Sections 3.1 through 3.4. The reader is referred to these sections for more details on the assessment. Conclusions regarding potential risks must be viewed in the context of the uncertainties associated with the assessment (Section 3.5) and the available risk information. The available risk information includes chemical data, exposure estimates, and literature-based toxicity information.

Table 3-24 summarizes the ecological risks at the Point Lay installation. The table shows the COCs associated with potential risks on a site-by-site basis for current and future conditions.

3.6.1 Potential Risks to Representative Plants

A qualitative comparison was conducted of onsite soil and surface water concentrations with plant toxicity information. The risk to plants is characterized using comparative information from the literature and BCF (B_v). Based on the qualitative comparison, the risks to plants are not significant.

3.6.2 Potential Risks to Representative Aquatic Species

Potential risks to aquatic species were evaluated by comparing toxicity information from the literature with the average exposure concentrations of potential contaminants in surface water. Although the HQs for aquatic organisms indicate that potential for risks may exist, a qualitative discussion in Section 3.4.2, Risk Characterization, indicates that there are circumstances that mitigate the potential risks. Therefore, considering site and COC-specific factors, the overall risk to aquatic organisms at the Point Lay installation is not considered significant.

3.6.3 Potential Risks to Representative Species of Birds and Mammals

The risks to representative species of birds and mammals were evaluated using the quotient method. This method compares the estimated exposures with TRVs, resulting in a calculated

TABLE 3-24. SUMMARY OF ECOLOGICAL RISK ESTIMATES AT THE POINT LAY SITES

SITE	COC CONTRIBUTING TO RISK	CURRENT RISK POTENTIAL	FUTURE RISK POTENTIAL
Deactivated Landfill -- LF01	None	not significant	not significant
Garage -- SS06	None	not significant	not significant
Drainage Pathway from POL Tanks -- SS07	None	not significant	not significant
Crushed Drum Area --SS08	None	not significant	not significant

HQ. In some cases, HQs were not calculated for avian species because of the lack of COC-specific toxicity information; they were evaluated by a qualitative comparison with the mammalian toxicities in Section 3.4.3, Risk Characterization. The resulting risk estimates for all of the avian and mammalian representative species were not significant. In addition, the risks resulting from potential future exposure to COCs at the Point Lay installation are estimated to be as low as, or lower than (i.e., not significant), the current estimates.

The objective of this ERA is to evaluate the potential risk to the representative plant, aquatic, and terrestrial species at the Point Lay DEW Line installation. The assessment indicates that, overall, the potential ecological risks presented by the COCs at the Point Lay sites are not significant.

3.6.4 Potential Ecological Risks

The objective of this ERA is to evaluate the potential risk to the representative species at the sites at the Point Lay DEW Line installation. This assessment indicates that, although there are a few instances of minimal potential risk to individual species, overall the potential risks presented by the COCs are very low.

4.0 REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological Profile for Zinc. Draft Report. U.S. Department of Health and Human Services.
- Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological Profile for Manganese. Draft Report. U.S. Department of Health and Human Services.
- Agency for Toxic Substances and Disease Registry (ATSDR). 1991a. Toxicological Profile for 2,3-Benzofuran. Draft Report. U.S. Department of Health and Human Services.
- Agency for Toxic Substances and Disease Registry (ATSDR). 1991b. Toxicological Profile for Trichloroethylene. Draft Report. U.S. Department of Health and Human Services.
- Agency for Toxic Substances and Disease Registry (ATSDR). 1993. Toxicological Profile for Fuel Oils. Draft Report. U.S. Department of Health and Human Services.
- Alaska Biological Research. 1994. Spectacled and Steller's Eiders Surveys at 11 CEOS Remote Sites in Alaska, 1994. Fairbanks, Alaska. 30 September 1994.
- Alaska Department of Environmental Conservation. 1991. Interim Guidance for Non-UST Contaminated Soil Cleanup Levels. Guidance Number 001, Revision 1. Alaska Department of Environmental Conservation, Juneau, Alaska. July 17, 1991.
- Albers, P.H. 1977. Effects of External Applications of Fuel Oil on Hatchability of Mallard Eggs. Pages 158-163 in D.A. Wolfe (Ed). Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms. Pergamon Press, New York.
- American Petroleum Institute (API). 1994. Results of Toxicological Studies Conducted for the American Petroleum Institute. API Publication No. 45591. January 1994.
- Ambrose, S. 1994. Personal communication with S. Ambrose, a threatened and endangered species specialist with the U.S. Fish and Wildlife Service. Don Kellett, ICF Kaiser Engineers, Lakewood, Colorado. 31 March 1994.
- Aquatic Information Retrieval (AQUIRE). 1990. Computerized Database. Chemical Information System, Inc. Baltimore, Maryland.
- Aquatic Information Retrieval (AQUIRE). 1994. Computerized Database. Chemical Information System, Inc. Baltimore, Maryland.
- Armstrong Laboratory. 1994. Evaluation of the Total Petroleum Hydrocarbon Standard at Jet Fuel Contaminated Air Force Sites. Prepared by EA Engineering, Science and Technology Inc. for Armstrong Laboratory, Brook AFB, Texas. January.

- Baes, C.F., III, R.D. Sharp, A.L. Sjoreen, and R.W. Shor. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides Through Agriculture. Oak Ridge National Laboratory. Prepared for the U.S. Department of Energy, Contract No. DE-AC05-84OR21400.
- Baudo, R., J. Giesy, and H. Muntau. 1990. Sediments: Chemistry and Toxicity of In-Place Pollutants. Lewis Publishers. Chelsea, Michigan.
- Beck, L.S., D.I. Hepler, and K.L. Hansen. 1982. The Acute Toxicology of Selected Petroleum Hydrocarbons. Pages 1-12 in H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, and M.L. Kaen (Eds). Proceedings of the Symposium - The Toxicology of Petroleum Hydrocarbons. American Petroleum Institute. Washington D.C.
- Belopol'skii, L.O. 1961. Ecology of Sea Colony Birds of the Barents Sea. Translated from the Russian Original. Israel Program for Scientific Translations. Jerusalem, Israel.
- Bergman, R.D., R.L. Howard, K.F. Abraham, and M. W. Weller. 1977. Water Birds and Their Wetland Resources in Relation to Oil Development at Storkersen Point, Alaska. Resource Publication 129. U.S. Fish and Wildlife Service. Washington D.C.
- Beyer, N., E. Conner, and S. Gerould. 1994. Estimates of Soil Ingestion by Wildlife. J. Wildl. Manage. 58(2):375-382.
- Brewster, K. 1994. Personal communication with North Slope Borough oral historian.
- Bott, T.L. and K. Rogenmuser. 1978. Effects of No. 2 Fuel Oil, Nigerian Crude Oil, and Used Crankcase Oil on Attached Algal Communities: Acute and Chronic Toxicity of Water-soluble Constituents. Applied and Environmental Microbiology. November 1978:673-682.
- Budavari, S. (Ed). 1989. The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Merck and Co., Inc. Rahway, New Jersey. Pp. 595.
- Burt, W.H. and R.P. Grossenheider (Eds). 1976. A Field Guide to the Mammals. Third Edition. Houghton-Mifflin Co., Boston, Massachusetts.
- Calder, W.A. and E.J. Braun. 1983. Scaling of Osmotic Regulation in Mammals and Birds. Am J. Physiol. 244:R601-R606.
- Cameron, R.D., D.J. Reed, J.R. Dau, and W.T. Smith. 1992. Redistribution of Calving Caribou in Response to Oil Field Development on the Arctic Slope of Alaska. Arctic 45(4):338-342.
- Chance, N. 1990. The Inupiat and Arctic Alaska: An Ethnography of Development. Holt, Rinehart, and Winston, New York. Pp. 241.
- Chappell, M.A. 1980. Thermal Energetics and Thermoregulatory Costs of Small Arctic Mammals. J. Mamm. 61(2):278-291.

- Chesemore, D.L. 1967. Ecology of the Arctic Fox in Northern and Western Alaska. M.S. Thesis, University of Alaska, Fairbanks.
- Clayton, G.D. and F.E. Clayton (Eds). 1981-1982. Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. Third Edition. John Wiley Sons, New York, New York. Pp. 3292.
- Clements, R.G. 1988. Estimating Toxicity of Industrial Chemicals to Aquatic Organisms Using Structure Activity Relationships. U.S. Environmental Protection Agency, Office of Toxic Substances, Washington D.C. EPA-560-6-88-001.
- Cuccarese, S.V., M.F. Arend, R.J. Hensel, and P.O. McMillan. 1984. Biological and Socioeconomic Systems of the BAR-M, POW-1, LIZ-3A, and SI-1 North Warning System Sites, Alaska. AEIDC. University of Alaska, Anchorage.
- Custance, S.R., P.A. McCaw, A.C. Kopf, and M.J. Sullivan. 1992. Environmental Fate of the Chemical Mixtures: Crude Oil, JP-5, Mineral Spirits and Diesel Fuel. Journal of Soil Contamination 1(4):379-386.
- Custer, T.W. and F.A. Pitelka. 1978. Seasonal Trends in Summer Diet of the Lapland Longspur near Barrow, Alaska. Condor 80:295-301.
- Delos, C. 1995. Personal communication with Charles Delos, United States Environmental Protection Agency. Karen Prochnow, ICF Kaiser Engineers. 25 April 1995.
- Derksen, D.V., T.C. Rothe, and W.D. Eldridge. 1981. Use of Wetland Habitats by Birds in the National Petroleum Reserve, Alaska. U.S. Fish and Wildlife Service, Resource Publication 141. Washington D.C.
- Doudoroff, P. and M. Katz. 1953. Critical Review of Literature on the Toxicity of Industrial Wastes and their Components to Fish. II. The Metals. Sewage and Industrial Wastes. 25(7)802-839.
- Dunning, J.B. 1984. Body Weights of 686 Species of North American Birds. Western Bird Banding Association. Monograph No. 1. Cave Creek, Arizona.
- Dupont De Nemours and Company, Inc. (Dupont). 1986. Flow-Through Acute 96-hour LC₅₀ of Freon 113 to Rainbow Trout. November 14, 1986.
- Dupont De Nemours and Company, Inc. (Dupont). 1990. Flow-Through Acute 96-hour LC₅₀ of Freon 21 to Rainbow Trout. September 14, 1990.
- Eberhardt, L.E., W.C. Hanson, J.L. Bengtson, R.A. Garrott, and E.E. Hanson. 1982. Arctic Fox Home Range Characteristics in an Oil-Development Area. J. Wildl. Manage. 46(1):183-190.

- Eisler, R. 1988. Lead Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service. Contaminant Hazard Reviews Report No. 14. Biological Report 85 (1.14).
- Eisler, R. 1993. Zinc Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service. Biological Report 10. Laurel, Maryland.
- Farrand, J., Jr. (Ed). 1983. The Audubon Society Master Guide to Birding, Volume 2. Alfred A. Knopf, New York, New York. 398 pp.
- Geiger, J.G. and A.L. Buikema, Jr. 1981. Oxygen Consumption and Filtering Rate of *Daphnia Pulex* After Exposure to Water-Soluble Fractions of Naphthalene, Phenanthrene, No. 2 Fuel Oil and Coal-Tar Creosote. Bulletin of Environmental Contamination and Toxicology 27:783-789.
- Harcharek, R.C. 1994. North Slope Borough 1993/94 Economic Profile and Census Report, Volume VII. North Slope Borough, Department of Planning and Community Services, Barrow, Alaska.
- Harding Lawson Associates. 1992. Offpost Operable Unit Endangerment Assessment/ Feasibility Study. Final Report. Technical Support for Rocky Mountain Arsenal. 24 November 1992. Prepared for Program Manager for Rocky Mountain Arsenal.
- Hart Crowser. 1987. Environmental Assessment for North Warning System. Alaska.
- Hartung, R. 1964. "Some Effects of Oils on Waterfowl." PhD Thesis. University of Michigan, Ann Arbor. Pages 426-436 in R.C. Szaro, M.P. Dieter, G.H. Heinz, and J.F. Ferrell. 1978. Effects of Chronic Ingestion of South Louisiana Crude Oil on Mallard Ducklings. Environmental Research 17:426-436.
- Hazardous Substance Data Bank (HSDB). 1994. National Institute of Health. Bethesda, Maryland.
- Heath, J.S., K. Koblis, and S.L. Sager. 1993. Review of Chemical, Physical, and Toxicologic Properties of Components of Total Petroleum Hydrocarbons. J. of Soil Contamination. 2(i): 1-25.
- Hedtke, S. and F.A. Puglisi. 1982. Short-Term Toxicity of Five Oils to Four Freshwater Species. Archives of Environmental Contamination and Toxicology 11:425-430.
- Hensel, R., M.F. Arend, J. Thiele, P.O. McMilland, and S.V. Cuccarese. 1984. Living Resources of the Point Barrow, Oliktok Point and Boulder Creek Areas, Alaska: A Literature Survey. AEIDC. University of Alaska, Anchorage.

- Hill, E.F. and M.B. Camardese. 1986. Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to *Coturnix*. U.S. Fish and Wildlife Service. Technical Report 2. Washington D.C. 147 p.
- Hoffman, D.J., J.C. Franson, O.H. Pattee, C.M. Bunck, and A. Anderson. 1985. Survival, Growth and Accumulation of Ingested Lead in Nestling American Kestrels (*Falco sparverius*). Arch. Environ. Contam. Toxicol. 14:89-94.
- Hudson, R.H., M.A. Haegle, and R.K. Tucker. 1979. Acute Oral and Percutaneous Toxicity of Pesticides to Mallards: Correlations with Mammalian Toxicity Data. Toxicology and Applied Pharmacology 47:451-460.
- Hull, R.N. and G.W. Suter II. 1994. Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Sediment Associated Biota: 1994 Revision. ORNL Environmental Restoration Program. ES/ER/TM-95/R1.
- Integrated Risk Information System (IRIS). 1994. Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Cincinnati, Ohio.
- Johnson, L. and B. Burns (Eds). 1984. Biology of the Arctic Char: Proceedings of the International Symposium on Arctic Char. University of Manitoba Press, Winnipeg, Man., Canada. 584 pp.
- Kabata-Pendias, A. and H. Pendias. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.
- Kauss, P.B. and T.C. Hutchinson. 1975. The Effects of Water-Soluble Petroleum Components on the Growth of *Chlorella vulgaris*, Beijerinck. Environmental Pollution (9):157-174.
- Kistchinski, A.A. and V.E. Flint. 1974. On the Biology of the Spectacled Eider. Wildfowl 25:5-15.
- Klaassen, C.D., M.O. Amdur, and J. Doull. 1986. Casarett and Doull's Toxicology, the Basic Science of Poisons. Third Edition. MacMillan Publishing Company. New York. 974 pp.
- Klein, S.A. and D. Jenkins. 1983. The Toxicity of Jet Fuels to Fish - II. Water Research 17 (10):1213-1220.
- Kostecki, P.T. and E.J. Calabrese. 1989. Petroleum Contaminated Soils. Volume 1. Lewis Publishers, Chelsea, Michigan. 357 pp.
- Kraus, M.L. 1989. Bioaccumulation of Heavy Metals in Pre-fledgling Tree Swallows, *Tachycineta Bicolor*. Bull. Environ. Contam. Toxicol. 43:407-414.
- Lewis, M.A., Evans, D.W., and J.G. Wiener. 1979. Manganese. In: R.V. Thurston, R.C. Russo, C.M. Ferrerolf Jr., T.A. Edsall, and Y.M. Barber Jr. (Eds.) 1979. A Review of the EPA Red

Book: Quality Criteria for Water. American Fisheries Society. Bethesda, Maryland. Pp. 137-144.

Lindsay, W.L. 1979. Chemical Equilibria in Soils. John Wiley & Sons. New York.

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt (Eds). 1982. Research and Development Methods for Estimating Physio-Chemical Properties of Organic Compounds of Environmental Concern. Report No. C-82426 by Arthur D. Little, Inc., under contract DAMD-17-78-C-0873, U.S. Army Medical R&D Command. Fort Detrick, Maryland.

Mantel, N. and M.A. Schneiderman. 1975. Estimating Safe Levels: A Hazardous Undertaking. Cancer Res. 35:1379-1386.

Martin, A.C., H.S. Zim, and A.L. Nelson. 1961. American Wildlife and Plants: A Guide to Wildlife Food Habits. Dover Publications, New York. 500 pp.

Massachusetts Department of Environmental Protection (MDEP). 1993. Petroleum Policy: Development of Health-Based Alternative to the TPH Parameter. Prepared by ABB Environmental Services, Inc. Wakefield, Massachusetts. Project No. 06979-00. August.

Mautino, M. and J.U. Bell. 1987. Hematological Evaluation of Lead Intoxication in Mallards. Bull. Environ. Contam. Toxicol. 38:29-34.

MITRE. 1990. General Guidance for Ecological Risk Assessment at Air Force Installation. J.M. DeSessa, Ph.D. and F.T. Prince, Ph.D., December 1990.

Moles, A., S.D. Rice, and S. Korn. 1979. Sensitivity of Alaskan Freshwater and Anadromous Fishes to Prudhoe Bay Crude Oil and Benzene. Transactions of the American Fisheries Society 108:408-414.

Nagy, K.A. 1987. Field Metabolic Rate and Food Requirement Scaling in Mammals and Birds. Ecol. Mono. 57:111-128.

National Academy of Sciences. 1980. Mineral Tolerance of Domestic Animals. Subcommittee on Mineral Toxicity in Animals. National Research Council. Washington D.C.

National Oceanic and Atmospheric Administration (NOAA). August 1991. The Potential for Biological Effects of Sediment Sorbed Contaminants Tested in the National Status and Trends Program. Technical Memorandum NOS OMA 52. NOAA, Seattle, Washington.

National Petroleum Reserve in Alaska Task Force (NPRA). 1978. Land Use Study, Volume 2. Values and Resource Analysis. U.S. Department of the Interior. Anchorage, Alaska.

Nowak, R.M. (Ed). 1991. Walker's Mammals of the World. Fifth Edition. Johns Hopkins University Press, Baltimore, Maryland.

- O'Brien, J.W. 1978. Toxicity of Prudhoe Bay Crude Oil to Alaskan Arctic Zooplankton. *Arctic* 31(3):219-228.
- Opresko, D.M., B.E. Sample, and G.W. Suter II. 1994. Toxicological Benchmarks for Wildlife: 1994 Revision. ORNL Environmental Restoration Program. ES/ER/TM-86/R1.
- Palmer, R.S. (Ed). 1976. Handbook of North American Birds, Volume 2, pp. 244-273. Yale University Press, New Haven, Connecticut. 521 pp.
- Pickering, Q.H., Hunt, E.P., Phipps, G.L., Roush, T.H., Smith, W.E., Spehar, D.L., Stephens, C.E., and D.K. Tanner. 1983. Effects of Pollution on Freshwater Fish and Amphibians. *JWPCF*. 55(6):840-862.
- Pitelka, F.A. 1959. Numbers, Breeding Schedule, and Territoriality in Pectoral Sandpipers of Northern Alaska. *Condor* 62(4):233-264.
- Puls, R. 1988. Mineral Levels in Animal Health. Sherpa International. Clearbrook, British Columbia.
- Raven, P.H., R.H. Evert, and F.E. Eichorn. 1986. Biology of Plants. Worth Publishers, Inc. New York, New York. 775 pp.
- Sangster Research Laboratories and Technical Database Services, Incorporated. 1994. Log Kow Database. Accessed August 21, 1995.
- Sax, I. and R.J. Lewis, Sr. 1989. Dangerous Properties of Industrial Materials. Seventh Edition. Van Nostrand, Reinhold, New York.
- Scott, S.L. 1983. Field Guide to the Birds. National Geographic Society. Washington D.C. 464 pp.
- Sims, R.C. and R. Overcash. 1983. Fate of Polynuclear Aromatic Compounds (PNAS) in Soil-Plant Systems. *Residue Rev.* 88:1-68. In: R. Eisler, 1987. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. USFWS Biological Report 85(1.11). May 1987.
- Skogland, T. 1980. Comparative Summer Feeding Strategies of Arctic and Alpine Rangifer. *Journal of Animal Ecology*. 49:81-98.
- Smith, G.J. 1987. Pesticide Use and Toxicology in Relation to Wildlife: Organophosphorus and Carbamate Compounds. U.S. Fish and Wildlife Service, Resource Publication 170. Washington D.C.
- Snyder-Conn, E. 1994. Personal communication with E. Snyder-Conn, Wildlife Biologist with the U.S. Fish and Wildlife Service, Fairbanks, Alaska. Don Kellett, ICF Kaiser Engineers. 31 March 1994.

- Soto, C., J. Hellebust, and T.C. Hutchinson. 1975. Effect of Napthalene and Aqueous Crude Oil Extracts on the Green Flagellate *Chlamysomanas Angulosa*. Canadian J. of Botany 53(2):118-126.
- Spacie, A. and J.L. Hamelink. 1985. Bioaccumulation, Chapter 17, pp. 495-525 in Fundamentals of Aquatic Toxicology. G.M. Rand and S.R. Petrocelli (Eds). Hemisphere Publishing Corporation, New York.
- Suter, G.W and J.B. Mabrey. 1994. Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision. Environmental Sciences Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee.
- Szaro, R.C. and P.H. Albers. 1977. Effects of External Applications of No. 2 Fuel Oil on Common Eider Eggs. Pp. 164-167. In: D.A. Wolfe (Ed). Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms. Pergamon Press, New York.
- Szaro, R.C., M.P. Dieter, G.H. Heinz, and J.F. Ferrell. 1978. Effects of Chronic Ingestion of South Louisiana Crude Oil on Mallard Ducklings. Environmental Research 17:426-436.
- Talmadge, S.S. and B.T. Walton. 1991. Small Mammals as Monitors of Environmental Contaminants. Reviews of Environ. Contam. Toxicol. 119:47-145.
- Travis, C.C. and A.D. Arms. 1988. Bioconcentration of Organics in Beef, Milk, and Vegetation. Environ. Sci. Technol. 22(3):271-274.
- Tucker, R.K. and J.S. Leitzke. 1979. Comparative Toxicology of Insecticides for Vertebrate Wildlife and Fish. Pharmac. Ter. 6:167-220.
- U.S. Air Force. 1991. Handbook to Support the Installation Restoration Program (IRP) Statements of Work. Volume 1, Remedial Investigation/Feasibility Studies (RI/FS). Human Systems Division, Brooks Air Force Base, Texas.
- U.S. Air Force. 1993. Sampling and Analysis Plan for DEW Line and Cape Lisburne Radar Stations. Prepared for USAF Center for Environmental Excellence, Environmental Restoration Program Office, Brooks AFB, Texas. Prepared by ICF Technology Inc.
- U.S. Air Force. 1996. Final Point Lay RI/FS Report. Prepared for USAF Center for Environmental Excellence, Environmental Restoration Program Office, Brooks AFB, Texas. Prepared by ICF Technology Inc. 04 March 1996.
- U.S. Army Corps of Engineers (USACOE). 1991. Baseline Risk Assessment for Eight Selected Study Areas at Aberdeen Proving Ground. Appendix C, Draft Document.
- U.S. Environmental Protection Agency. 1976. Quality Criteria for Water. Office of Water and Hazardous Materials. Washington D.C.

- U.S. Environmental Protection Agency. 1985. Environmental Profiles and Hazard Indices for Constituents of Municipal Sludge: Iron. Office of Water Regulations and Standards. Washington D.C.
- U.S. Environmental Protection Agency. 1986a. Guidelines for Carcinogen Risk Assessment. Federal Register 51:33992-34013.
- U.S. Environmental Protection Agency. 1986b. Guidelines for Health Risk Assessment of Chemical Mixtures. Federal Register 51:34014-34025.
- U.S. Environmental Protection Agency. 1986c. Quality Criteria for Water. Office of Water Regulations and Standards. Washington D.C. EPA 440/5-86-001.
- U.S. Environmental Protection Agency. 1989a. Risk Assessment Guidance for Superfund, Human Health Evaluation Manual, Part A. Office of Solid Waste and Emergency Response. Washington D.C.
- U.S. Environmental Protection Agency. 1989b. Risk Assessment Guidance for Superfund: Volume 2, Environmental Evaluation Manual. Office of Solid Waste and Emergency Response. Washington D.C.
- U.S. Environmental Protection Agency. 1991a. Region 10 Supplemental Risk Assessment Guidance for Superfund. Seattle, Washington. 16 August 1991.
- U.S. Environmental Protection Agency. 1991b. Human Health Evaluation Manual, Part B: Development of Risk/Based Preliminary Remediation Goals. Office of Solid Waste and Emergency Response. Washington D.C. December 13, 1991.
- U.S. Environmental Protection Agency. 1991c. Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions. Office of Solid Waste and Emergency Response. Washington D.C. 22 April 1991.
- U.S. Environmental Protection Agency. 1991d. Update on OSWER Soil Lead Guidance (OSWER Directive 9355.4-02). Office of Solid Waste and Emergency Response, Washington D.C. August 29, 1991.
- U.S. Environmental Protection Agency. 1992a. Framework for Ecological Risk Assessment. EPA/630/R-92/001. NTIS #PB93-102192. Washington D.C. February 1992.
- U.S. Environmental Protection Agency. 1992b. Oral Reference Doses and Oral Slope Factors for JP-4, JP-5, Diesel fuel, and Gasoline. Environmental Criterion Assessment Office, Office of Research and Development. Cincinnati, Ohio. March 24, 1992.
- U.S. Environmental Protection Agency. 1992c. Handbook of RCRA Ground-Water Monitoring Constituents: Chemical and Physical Properties. Office of Solid Waste Permits and State Programs Division. September 1992.

- U.S. Environmental Protection Agency (EPA). 1994. Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments. USEPA Environmental Response Team, Edison, New Jersey. September 26, 1994. Review Draft.
- U.S. Fish and Wildlife Service. 1982. Arctic National Wildlife Refuge Coastal Plain Resource Assessment-Initial Report. Baseline Study of Fish, Wildlife, and Their Habitats. U.S. Department of the Interior, Anchorage Alaska.
- U.S. Fish and Wildlife Service. 1986. Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to Coturnix. Fish and Wildlife Technical Report 2. Washington D.C. E.F. Hill and M.B. Camardese.
- University of Alaska, Arctic Environmental Information and Data Center. 1978. Point Lay.
- Upicksoun. 1995. Personal communication with Upicksoun, Point Lay Alaska. Dr. Scott Dwyer, ICF Kaiser Engineers. 1995.
- Verschueren, K. 1983. Handbook of Environmental Data of Organic Chemicals. Second Edition. New York: Van Nostrand Reinhold Co., p. 629.
- Von Burg, R. 1993. Evaluation of TPH as a Determinant for Petroleum Hydrocarbon Cleanup in Soil. ICF Kaiser Engineers, Oakland, California.
- Walker, D.A., P.J. Webber, K.R. Everett, and J. Brown. 1978. Effects of Crude and Diesel Oil Spills on Plant Communities at Prudhoe Bay, Alaska, and the Derivation of Oil Spill Sensitivity Maps. Arctic 31(3):242-259.
- Weeks, J.A., G.H. Drendel, R.S. Jagan, T.E. McManus, and P.J. Sczerzenie. 1988. Diesel Oil and Kerosene Background Statement. Prepared by Labat-Anderson, Inc. for the U.S. Department of Agriculture, Forest Service. February.
- White, R.G. and J. Trudell. 1980. Habitat Preference and Forage Consumption by Reindeer and Caribou Near Atkasook, Alaska. Arctic and Alpine Research 12(4):511-529.
- Wilber, C.G. 1980. Toxicology of Selenium: A Review. Clinical Toxicology 17(2):171-230.
- Wong, C.K., F.R. Engelhardt, and J.R. Strickler. 1981. Survival and Fecundity of *Daphnia Pulex* on Exposure to Particulate Oil.
- Woodward-Clyde Corporation. 1993. Natural Resources Plan: North Coastal Long Range Radar Sites. Final Draft. Prepared for the United States Air Force.
- Wootton, R.J. 1976. The Biology of the Sticklebacks. Academic Press, New York. 387 pp.

APPENDIX A

RISK CHARACTERIZATION SPREADSHEETS

Deactivated Landfill (LF01)	A-1
Garage (SS06)	A-4
Drainage Pathway from POL Tanks (SS07)	A-7
Crushed Drum Area (SS08)	A-9

TABLE A-1. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion
 Endpoint: Noncancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Deactivated Landfill (LF01)
 File: LF01SONC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	0.000001	0.000001	0.000001
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
DRPH	0.08	624	3.66e-05	7.33e-05	6.84e-04	4.58e-04	9.46e-03
HAZARD INDEX							0.009

TABLE A-2. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
 Endpoint: Noncancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Deactivated Landfill (LF01)
 File: LF01WANC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult
Water Ingestion	(L/day)	2	2
Exposure Frequency	(days/year)	180	180
Exposure Duration	(years)	10	55
Conversion Factor	(kg/mg)	1	1
Body Weight	(kg)	70	70
Averaging Time	(ED x 365 days/year)	3,650	20,075

Chemical	Oral RfD	Concentration Water (mg/L)	ADD by Receptor Group (mg/kg-day)		Hazard Quotient	
			DEW Line Worker	Native Northern Adult	DEW Line Worker	Native Northern Adult
Manganese	0.005	0.96	1.35e-02	1.35e-02	2.71e+00	2.71e+00
GRPH	0.2	0.223	3.14e-03	3.14e-03	1.57e-02	1.57e-02
Tetrachloroethene	0.01	0.109	1.54e-03	1.54e-03	1.54e-01	1.54e-01
Dichlorodifluoro-methane	0.2	0.058	8.17e-04	8.17e-04	4.09e-03	4.09e-03
			HAZARD INDEX		2.88e+00	2.88e+00

TABLE A-3. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
 Endpoint: Cancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Deactivated Landfill (LF01)
 File: LF01WACA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult
Water Ingestion	(L/day)	2	2
Exposure Frequency	(days/year)	180	180
Exposure Duration	(years)	10	55
Conversion Factor	(kg/mg)	1	1
Body Weight	(kg)	70	70
Averaging Time	(lifetime in days)	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Water (mg/L)	LADD by Receptor Group (mg/kg-day)		Cancer Risk	
			DEW Line Worker	Native Northern Adult	DEW Line Worker	Native Northern Adult
GRPH	0.0017	0.223	4.49e-04	2.47e-03	7.63e-07	4.20e-06
Benzene	0.029	0.023	4.63e-05	2.55e-04	1.34e-06	7.38e-06
Tetrachloroethene	0.052	0.109	2.19e-04	1.21e-03	1.14e-05	6.27e-05
Trichloroethene	0.011	0.003	6.03e-6	3.33e-5	6.633e-08	3.66e-07
CANCER RISK					1e-05	7e-05

TABLE A-4. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion
 Endpoint: Noncancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Garage (SS06)
 File: SS06SONC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3,650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
DRPH	0.08	33,400	1.96e-03	3.92e-03	3.66e-02	2.45e-02	5.07e-01
GRPH	0.2	937	5.50e-05	1.10e-04	1.03e-03	2.75e-04	5.68e-03
RRPH	0.08	40,000	2.35e-03	4.70e-03	4.38e-02	2.94e-02	6.07e-01
Tetrachloroethene	0.01	43	2.52e-06	5.05e-06	4.71e-05	2.52e-04	5.22e-03
1,1,2-Trichloroethane	0.004	1.8	1.06e-07	2.11e-07	1.97e-06	2.64e-05	1.00e-3
HAZARD INDEX						0.054	1.124

TABLE A-5. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion
 Endpoint: Cancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Garage (SS06)
 File: SS06SOCA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	0.000001	0.000001	0.000001
Body Weight	(kg)	70	70	15
Averaging Time	(lifetime in days)	25,550	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Soil (mg/kg)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
GRPH	0.0017	937	7.86e-06	7.70e-05	8.80e-05	1.34e-08	2.81e-07
Tetrachloroethene	0.052	43	3.61e-07	3.53e-06	4.04e-06	1.88e-08	3.94e-07
1,1,2-Trichloroethane	0.057	1.8	1.51e-08	1.48e-07	1.69e-07	7.86e-10	1.81e-08
CANCER RISK							7e-07

TABLE A-6. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
 Endpoint: Noncancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Garage (SS06)
 File: SS06WANC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult
Water Ingestion	(L/day)	2	2
Exposure Frequency	(days/year)	180	180
Exposure Duration	(years)	10	55
Conversion Factor	(kg/mg)	1	1
Body Weight	(kg)	70	70
Averaging Time	(ED x 365 days/year)	3,650	20,075

Chemical	Oral RfD	Concentration Water (mg/L)	ADD by Receptor Group (mg/kg-day)		Hazard Quotient	
			DEW Line Worker	Native Northern Adult	DEW Line Worker	Native Northern Adult
Manganese	0.005	1.7	2.40e-02	2.40e-02	4.79e+00	4.79e+00
Barium	0.07	0.36	5.07e-03	5.07e-03	7.25e-02	7.25e-02
			HAZARD INDEX		4.86e+00	4.86e+00

TABLE A-7. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
 Endpoint: Noncancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Drainage Pathway from POL Tanks (SS07)
 File: SS07WANC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult
Water Ingestion	(L/day)	2	2
Exposure Frequency	(days/year)	180	180
Exposure Duration	(years)	10	55
Conversion Factor	(kg/mg)	1	1
Body Weight	(kg)	70	70
Averaging Time	(ED x 365 days/year)	3,650	20,075

Chemical	Oral RfD	Concentration Water (mg/L)	ADD by Receptor Group (mg/kg-day)		Hazard Quotient	
			DEW Line Worker	Native Northern Adult	DEW Line Worker	Native Northern Adult
DRPH	0.08	0.961	1.35e-02	1.35e-02	1.69e-01	1.69e-01
GRPH	0.2	0.189	2.66e-03	2.66e-03	1.33e-02	1.33e-02
Tetrachloroethene	0.01	0.002	2.82e-05	2.82e-05	2.82e-03	2.82e-03
1,1-Dichloroethene	0.009	0.0023	3.24e-05	3.24e-05	3.60e-03	3.60e-03
cis-1,2-Dichloroethene	0.01	0.178	2.51e-03	2.51e-03	2.51e-01	2.51e-01
HAZARD INDEX					4.40e-01	4.40e-01

TABLE A-8. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
 Endpoint: Cancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Drainage Pathway from POL Tanks (SS07)
 File: SS07WACA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult
Water Ingestion	(L/day)	2	2
Exposure Frequency	(days/year)	180	180
Exposure Duration	(years)	10	55
Conversion Factor	(kg/mg)	1	1
Body Weight	(kg)	70	70
Averaging Time	(lifetime in days)	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Water (mg/L)	LADD by Receptor Group (mg/kg-day)		Cancer Risk	
			DEW Line Worker	Native Northern Adult	DEW Line Worker	Native Northern Adult
GRPH	0.0017	0.189	3.80e-04	2.09e-03	6.47e-07	3.56e-06
Benzene	0.029	0.0017	3.42e-06	1.88e-05	9.92e-08	5.46e-07
1,1-Dichloroethene	0.6	0.0023	4.63e-06	2.55e-05	2.78e-06	1.53e-05
Tetrachloroethene	0.052	0.002	4.03e-06	2.21e-05	2.09e-07	1.15e-06
Trichloroethene	0.011	0.133	2.68e-04	1.47e-03	2.94e-06	1.62e-05
CANCER RISK					7e-06	4e-05

TABLE A-9. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion
 Endpoint: Noncancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Crushed Drum Area (SS08)
 File: SS08SONC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	0.000001	0.000001	0.000001
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3,650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
DRPH	0.08	17,500	1.03e-03	2.05e-03	1.92e-02	1.28e-02	2.65e-01
GRPH	0.2	2,430	1.43e-04	2.85e-04	2.66e-03	7.13e-04	1.47e-02
HAZARD INDEX							0.280

TABLE A-10. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion
 Endpoint: Cancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Crushed Drum Area (SS08)
 File: SS08SOCA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	0.000001	0.000001	0.000001
Body Weight	(kg)	70	70	15
Averaging Time	(lifetime in days)	25,550	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Soil (mg/kg)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
GRPH	0.0017	2,430	2.04e-05	2.00e-04	2.28e-04	3.46e-08	7.28e-07
Benzene	0.029	1.4	1.17e-08	1.15e-07	1.32e-07	3.41e-10	7.15e-09
CANCER RISK							7e-07

TABLE A-11. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
 Endpoint: Noncancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Crushed Drum Area (SS08)
 File: SS08WANC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult
Water Ingestion	(L/day)	2	2
Exposure Frequency	(days/year)	180	180
Exposure Duration	(years)	10	55
Conversion Factor	(kg/mg)	1	1
Body Weight	(kg)	70	70
Averaging Time	(ED x 365 days/year)	3,650	20,075

Chemical	Oral RfD	Concentration Water (mg/L)	ADD by Receptor Group (mg/kg-day)		Hazard Quotient	
			DEW Line Worker	Native Northern Adult	DEW Line Worker	Native Northern Adult
Manganese	0.005	0.61	8.59e-03	8.59e-03	1.72e+00	1.72e+00
Barium	0.07	0.33	4.65e-03	4.65e-03	6.64e-02	6.64e-02
			HAZARD INDEX		1.79e+00	1.79e+00

TABLE A-12. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
 Endpoint: Cancer
 Assumptions: Site-specific
 Installation: Point Lay
 Site: Crushed Drum Area (SS08)
 File: SS08WACA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult
Water Ingestion	(L/day)	2	2
Exposure Frequency	(days/year)	180	180
Exposure Duration	(years)	10	55
Conversion Factor	(kg/mg)	1	1
Body Weight	(kg)	70	70
Averaging Time	(lifetime in days)	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Water (mg/L)	LADD by Receptor Group (mg/kg-day)		Cancer Risk	
			DEW Line Worker	Native Northern Adult	DEW Line Worker	Native Northern Adult
Benzene	0.029	0.015	3.02e-05	1.66e-04	8.76e-07	4.82e-06
			CANCER RISK			
					9e-07	5e-06

APPENDIX B

TOXICITY PROFILES

GASOLINE (GRPH)	B-1
DIESEL FUEL (DRPH)	B-6
BENZENE	B-11
WASTE OIL (RRPH)	B-13
1,1,2-TRICHLOROETHANE	B-19
TRICHLOROETHENE	B-21
TETRACHLOROETHENE	B-23
BARIUM	B-25
1,1-DICHLOROETHENE	B-28
cis-1,2-DICHLOROETHENE	B-30
DICHLORODIFLUOROMETHANE	B-32
MANGANESE	B-34

TOXICOLOGY PROFILE FOR GASOLINE (GRPH)

GENERAL DATA

The chemical composition of gasoline is extremely variable, depending upon the crude oil starting material, types of processing and refining, blending and additives employed. Gasolines are formulated to meet fuel performance specifications, not to achieve a specific chemical composition. Volatility must be within a certain range to avoid vapor lock (too high) or sluggish acceleration (too low). In addition, the air-fuel mixture within the cylinder must burn uniformly to prevent "pinging" or "knocking." Often small quantities of butanes, pentanes, organo lead compounds or branched chain hydrocarbons are added to achieve uniform burning rates. McDermott and Killiany (1978) published a detailed gas chromatographic analysis of a premium grade gasoline listing 21 components that accounted for 92 percent of the gasoline vapors (Table 1). Low-volatility hydrocarbons (high carbon numbers) were not well represented.

Gasoline additives include organic lead (tetraethyl lead and tetramethyl lead) to a concentration of 0.1 g/gallon (7 ppm). Alkyl lead vapors have low volatility (vapor pressure = 0.4 mm Hg) compared to gasoline (400-775 mm Hg), so lead compounds should not be acutely hazardous by inhalation. To prevent accumulation of lead deposits, scavenging agents ethylene dichloride (EDC) and ethylene dibromide (EDB) are added to fuels, usually in a molar ratio EDC/EDB/Pb = 2:2:1.

FATE AND TRANSPORT

Gasoline released into the environment would be expected to evaporate rapidly because of its high vapor pressure (400-477 mm Hg). Studies of gasoline fate when added to soils show that the main clearance mechanism was evaporation, which can account for up to 75 percent removal from surface soils (Donaldson 1990). Microbial degradation plus evaporation can remove up to 90 percent of the added gasoline (Song 1988). Benzene, a volatile gasoline component of major toxicological interest, has a half life in the air of less than one day (Korte and Klein 1982). Gasoline has appreciable water solubility (12 to 16 percent) so it would be transported in ground water and may be found in well water.

TOXICITY DATA

Human Toxicological Profile

Like other solvents, gasoline has potent CNS depressant activity. Breathing vapors at concentrations achieved during "huffing" or occupational overexposures has led to a variety of neurological symptoms: hallucinations, encephalopathy, ataxia, convulsions, Tourette's Disease, vertigo and nystagmus, and peripheral neuropathy (Von Burg 1989). Many of these symptoms may be attributed to n-hexane or alkyl lead compounds.

Ingestion of gasoline can occur during siphoning or abuse situations, or from contaminated well-water. Ingestion is accompanied by a burning sensation in the mouth, pharynx and chest. Swallowing large amounts of gasoline leads to coma and death by respiratory depression. A serious complication is the aspiration of hydrocarbons into the lung which produces a potentially lethal hemorrhagic pneumonitis (Lee and Seymour 1979).

TABLE 1. COMPOSITION OF A PREMIUM-GRADE GASOLINE

COMPOUND	VOL %
Propane	0.8
n-Butane	38.1
Isobutane	5.2
n-Pentane	7.0
Cyclopentane	0.7
2,3-DM-butane	0.7
2-M-pentane	2.1
3-M-pentane	1.6
n-Hexane	1.5
M-cyclopentane	1.3
2,4-DM-pentane	0.4
2,3-DM-pentane	0.7
2,2,4-TM-pentane	0.5
Isobutylene	1.1
2-M-1-butane	1.6
c-2-Pentene	1.2
2-M-2-butene	1.7
Benzene	0.7
Toluene	1.8
Xylene (m,p,o)	0.5
Total %	92.1

Inhalation exposure to concentrations of 1,000 - 5,000 ppm for 15-60 minutes can produce CNS depression. A 5-minute exposure to 20,000 ppm (20 percent) has been reported to be fatal (Von Burg 1989).

Three epidemiologic studies showed no increased cancer risk in refinery workers (Hanis et al. 1982; Kaplan 1986; and Wong 1987). In an epidemiological study of refinery workers and gasoline handlers, Thomas et al. (1982), found a significant increase in stomach and brain cancer with a trend to increased leukemia and cancer of the skin, prostate and pancreas.

Animal Toxicology and Significant Studies

The acute dermal LD₅₀ of gasoline in rabbits is reported to be <5 ml/kg (Von Burg 1989). Liquid gasoline is considered a primary skin irritant because of the defatting and fissuring that occurs with repeated contact. Hypersensitivity response to gasoline can occur. Dermal absorption of gasoline is unlikely to result in systemic toxicity, but chronic exposure to alkyl lead additives may be toxic. Gasoline is acutely irritating to the eye, but animal studies indicate no effect lasting longer than seven days.

MacFarland (1982), reported a chronic inhalation study of gasoline in Fischer 344 rats and B6C3F₁ mice. Exposure levels were 0, 67, 292, and 2056 ppm for 6 hours/day, 5 days/week for 103 to 113 weeks. Male (but not female) rats exhibited a progressive renal tubular disease and renal carcinomas in all dose groups; renal effects in mice were not significantly different from controls. High dose female mice had an increased incidence of hepatocellular tumors (48 percent); the spontaneous incidence of these tumors is also high (14 percent). Males showed no increase (44 percent high dose vs. control 45 percent).

Reproductive Toxicity

Male rats exposed intermittently to about 650 ppm unleaded gasoline for 2 months showed endocrine changes which were attributed to stress. Pregnant females exposed to 0, 400, and 1,600 ppm unleaded gasoline on days 6-15 of gestation for 6 hours/day did not show any teratogenic or fetotoxic effects. Mental retardation has been reported among offspring of gasoline-sniffing mothers.

Genotoxicity

Several common fuels gave negative results in the Ames Salmonella typhimurium assay, mouse lymphoma, and the rat bone marrow chromosomal aberration assay (Lebowitz et al. 1979). Unleaded gasoline did not induce unscheduled DNA synthesis in the male rat kidney at doses known to be nephrotoxic.

Carcinogenicity

As indicated earlier, chronic gasoline exposures produces renal tumors in rats.

REGULATIONS AND STANDARDS

The American Conference of Governmental Industrial Hygienists (ACGIH 1990) adopted a threshold limit value (TLV) of 300 ppm (mg/m³) for gasoline vapors. Because of the complexity and variability in the composition of gasoline, OSHA has no standard; it regulates the toxic components by their respective PELs (e.g., n-hexane, benzene, alkyl lead).

Gasoline is not mentioned in HEAST (1990) as having a specific cancer slope factor (CSF) or RfD. However, individual components such as benzene, other aromatics, and n-hexane have CSF or RfD values and should be evaluated individually.

COMPOUND CAS NO.	ACGIH TLV ppm	RfD (inhal) mg/kg/day	RfD (oral) mg/kg/day	SLOPE FACTOR (inhal) kg-day/mg	SLOPE FACTOR (oral) kg-day/mg
Benzene 71-43-2	0.1	N/A	N/A	2.9E-2	2.9E-2
Ethylene Dibromide 106-93-4	A2 ¹	N/A	N/A	7.7E-1	8.5E+1
Ethylene Dichloride 107-06-2	10	N/A	N/A	9.1E-2	9.1E-2
n-Hexane 110-54-3	50	6E-1	2E-1	N/A	N/A
Tetraethyl Lead 78-00-2	0.1 ²	1E-7	2.9E-8	N/A	N/A

¹ A2 - Substance classed as a suspected human carcinogen, no ACGIH TLV listed.
² mg/m³, not ppm.

REFERENCES

- ACGIH. 1990. Threshold Limit Values for Chemical Substances and Biological Exposure Indices for 1990-1991. Cincinnati, Ohio: American Conference of Governmental Industrial Hygienists.
- Donaldson, S.G. 1990. Volatilization of Gasoline from Contaminated Soil. M.S. Thesis, University of Reno.
- Hanis, N.M., T.M. Holmes, L.G. Shallenberger, and K.E. Jones. 1982. Epidemiologic Study of Refinery and Chemical Plant Workers. J. Occup. Med. 24:203-212.
- HEAST. 1990. Health Effects Assessment Summary Tables, Environmental Protection Agency. Office of Emergency and Remedial Response. EPA, OERR 9200 6-303 (90-4).
- Kaplan, S. 1986. Update of a Mortality Study of Workers in Petroleum Refineries. J. Occup. Med. 28:514-516.
- Korte, F. and W. Klein. 1982. Degradation of Benzene in the Environment. Ecotox. Environ. Safety 6:311-327.

- Lee, T. and W. Seymour. 1979. Pneumonitis Caused by Petrol Siphoning. *Lancet* 8134:149.
- Lebowitz, H., D. Brusick, and D. Matheson et al. 1979. Commonly Used Fuels and Solvents Evaluated in a Battery of Short-Term Bioassays. *Environ. Mutagen.* 1:172-173.
- MacFarland, H.N. 1982. Chronic Gasoline Toxicology. In: *Proceedings of the Symposium: The Toxicology of Petroleum Hydrocarbons*, American Petroleum Institute, Washington D.C.
- McDermott, H. and S. Killiany. 1978. Quest for a Gasoline TLV. *Am. Ind. Hyg. Assoc. J.* 39:110-117.
- Song, H.G. 1988. Petroleum Hydrocarbons in Soil: Biodegradation and Effects on the Microbial Community. Ph.D. Thesis, University of Medicine and Dentistry of New Jersey.
- Thomas, T.L., R.J. Waxweiler, and R. Moure-Eraso et al. 1982. Mortality Patterns Among Workers in Three Texas Oil Refineries. *J. Occup. Med.* 24:135-141.
- Wong, O. 1987. An Industry Wide Mortality Study of Chemical Workers Occupationally Exposed to Benzene. *Br. J. Ind. Med.* 44:365-381.
- Von Burg, R. 1989. Toxicology Update for Gasoline. *J. Appl. Toxicol.* 9:203-210.

TOXICOLOGY PROFILE FOR DIESEL FUEL (DRPH)

GENERAL DATA

Petroleum fuels are classified into light, middle and heavy distillate fuels. Gasoline is a typical light distillate fuel and diesel fuel is considered to be a middle distillate material obtained from the distillation of crude oil. Included in this category of middle distillate fuels are jet fuel, kerosene and No. 2 fuel oils; many of the ecological and toxicological effects of these materials are very similar.

The chemical composition of diesel fuel is extremely variable and depends upon the crude oil source, types of processing and refining, blending and additives employed. These fuels are formulated to meet physical characteristics and not a specific chemical composition. Viscosity and volatility are the principal determinants of the fuel specifications. Diesel #1 is primarily a kerosene type of fuel produced mainly from straight run middle distillates. Diesel #2 also contains straight run middle distillates but is blended with straight run kerosene, straight run gas oils, light vacuum distillate and light thermally and/or catalytically cracked streams (IARC 1989).

Like other petroleum-derived fuels, diesel fuels consist of paraffins, olefins, cycloparaffins, isoparaffins and aromatics, as well as additives. Additives can include amyl nitrates and alcohols, n-hexyl nitrate and octyl nitrate at levels of 0.1 to 0.2 percent (Kirk-Othmer 1984). The total aromatic content of diesel fuel is variable, but levels between 23 to 38 percent have been reported. The average total aromatic content is probably in the range of 25 percent. The concentrations of the principal aromatic species of toxicological significance are presented in Table 2.

TABLE 2. REPRESENTATIVE VALUES FOR TOXICOLOGICALLY SIGNIFICANT AROMATIC CONTENT FOR DIESEL FUEL #2.

COMPONENT	APPROXIMATE CONCENTRATION
Benzene	<50 ppm with an average of 10 ppm
Ethylbenzene	300 ppm
Toluene	200 ppm (max)
Xylene (mixed)	2,400 ppm

(personal communication, Chevron Corp.)

The odor threshold of diesel fuel is approximately 0.8 ppm.

FATE AND TRANSPORT

Microbial degradation plus evaporation can remove up to 90 percent of the added diesel fuel to **soil**. Depending on the soil characteristics, the half-life of diesel fuel in soil ranges from one to eight weeks (Song 1988). Volatilization to the **air** occurs and diesel fuel can be detected by its odor in the air. However, a vapor pressure value could not be located in the literature. Diesel fuel will percolate through the soil and float on the ground **water**. When spilled onto surface water, diesel fuels can be toxic to fish, waterfowl and algae.

TOXICITY DATA

Human Toxicological Profile

Like other solvents, diesel fuel can be expected to have central nervous system (CNS) depressant activity. However, since this fuel is not as volatile as gasoline, breathing vapors at concentrations sufficient to achieve a level of intoxication is not likely at normal temperatures and pressures. An attempt to generate a kerosene (diesel) laden atmosphere resulted in an ambient concentration of only 14 ppm (Carpenter et al. 1976). However, under certain occupational settings like tank cleaning, it may be possible to generate mists or aerosols that can lead to symptoms of overexposure. As with kerosene, these symptoms may include headache, dizziness, weakness, confusion, drowsiness, and possibly death (HSDB 1991).

Ingestion of diesel fuel can occur during siphoning, abuse situations, or from contaminated well-water. Ingestion may be accompanied by a burning sensation in the mouth, pharynx and chest, gastrointestinal hypermotility and diarrhea (Gosselin et al. 1984), and possibly nausea and vomiting. A serious complication is the aspiration of hydrocarbons into the lung, which produces a potentially-lethal hemorrhagic pneumonitis (Lee and Seymour 1979).

There have been reports of acute renal failure in people following exposure to diesel fuel (Barrientos et al. 1977; Crisp et al. 1979). Kryzanovskij (1971) reports that workers cleaning diesel storage tanks have an increased general incidence of disease, and specifically cardiovascular disease and bronchitis, over control shipyard workers.

Animal Toxicology and Significant Studies

The acute oral and dermal LD₅₀ of diesel fuel is in the range of 9 ml/kg body weight. Eye irritation properties were minimal, but the primary skin irritation score of a marketplace sample was 6.8 indicating that this material is a strong skin irritant (Beck et al. 1982). Chronic skin contact can be expected to produce defatting, fissuring and cracking. There are no readily available reports on hypersensitivity. Response to diesel fuels can be expected because products on either side of diesel fuels distillation range have been reported to produce hypersensitivity reactions (Beck et al. 1982). Dermal absorption of gasoline is unlikely to result in systemic toxicity, but chronic poisoning of the readily absorbable alkyl lead additives is possible.

Exposure of CD-1 mice to diesel vapor for eight hours per day on five consecutive days resulted in a decrement of performance on the roto-rod test, square box activity test and hot plate test. However, the corneal reflex and inclined plane test were unaffected. General observations noted vasodilation, ataxia, poor grooming and, in some cases, tremor (Kainz and White 1982).

Exposure of rats to aerosolized diesel fuel at concentrations up to 6 mg/L produced direct toxic effects on the lungs but did not produce any neurotoxicity (Dalbey et al. 1987).

Reproductive Toxicity

Female rats were exposed 6 hours per day to air concentrations of 0, 100, and 400 ppm during days 6 through 15 of gestation. Neither jet fuel or number 2 fuel oil produced any significant detrimental effects on the reproductive parameters of the experimental animals (Beliles and Mecler 1982). Neither Jet Fuel A nor diesel fuel at exposure levels of 400 ppm, 6 hours per day, 5 day per week for 8 weeks reduced the fertility of CD-1 male mice (API 1980a,b).

Genotoxicity

Kerosene, jet fuel and diesel fuel all tested negatively in the standard Ames bioassay. However, the "Modified Ames Assay" (Blackburn et al. 1988) on two straight run gas oils did demonstrate mutagenicity straight run gas oil can be considered similar to diesel oils). Diesel fuel was also negative in the mouse lymphoma assay but positive on the rat bone marrow cytogenetics assay when administered by intraperitoneal injection (Conaway et al. 1982). Heating oil #2 did produce a positive Ames test as well as positive results in two other short term bioassays (Rothman and Emmett 1988). Dominant lethal testing of Jet fuel A and diesel fuel was negative at 400 ppm to male CD-1 mice (API 1980a,b).

Carcinogenicity

In a classical mouse skin-painting bioassay, all petroleum fractions derived from a crude oil source that boiled between 120 and 700°F showed a low level of tumorigenic activity (Lewis et al. 1982). Home heating oil also showed a low level of tumorigenicity in a more recent mouse skin-painting assay (Witschi et al. 1987).

In a case referent study, Seimietycki et al. (1987), reported an increase of several specific cancers associated with exposures to different petroleum products. Leaded gasoline was associated with stomach cancer; aviation gasoline with kidney cancer; diesel fuel with non adenocarcinoma of the lung and prostate cancer; and mineral spirits with squamous cell lung cancer. However, not all parameters of concern were properly controlled, excluded or assessed, making conclusions from this study inappropriate.

IARC (1989) has classified diesel fuel as having limited evidence of carcinogenicity in animals. Light diesel fuels are not classifiable as to their carcinogenicity to humans (Group 3).

REGULATIONS AND STANDARDS

Neither the American Conference of Governmental Industrial Hygienists (ACGIH) nor OSHA have recommended or established permissible exposure standards (PELs) for diesel fuels. However, NIOSH has recommended a 10 hour time-weighted average (TWA) of 100 mg/m³ or 14 ppm for kerosene (NIOSH 1977). Because of the complexity and variability in composition, OSHA regulates the toxic components by their respective PELs (i.e., n-hexane, benzene, etc.).

Diesel fuels, as such, are not mentioned in HEAST 1990, nor identified for a specific cancer Potency Factor (CPF) or reference dose (RfD). However, individual components such as

benzene, other aromatics and n-hexane that have CPF or RfD values should be evaluated individually.

REFERENCES

- API. 1980a. Mutagenicity Evaluation of Jet Fuel A in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-03. American Petroleum Institute Medical Research Publications, Washington D.C.
- API. 1980b. Mutagenicity Evaluation of Diesel Fuel in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-04. American Petroleum Institute Medical Research Publications, Washington D.C.
- Barrientos, A., M.T. Ortuno, and J.M. Morales et al. 1977. Acute Renal Failure After Use of Diesel Fuel as a Shampoo. *Arch. Intern. Med.* 137:1217-1219.
- Beck, L.S., D.I. Hepler, and K.L. Hansen. 1982. The Acute Toxicology of Selected Petroleum Hydrocarbons. In: *Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons*. H.N. MacFarland et al. (Editors), American Petroleum Institute, Publisher. Washington D.C. May.
- Beliles, R.P. and F.J. Mecler. 1982. Inhalation Teratology of Jet Fuel, Fuel Oil and Petroleum Naphtha in Rats. In: *Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons*. H.N. MacFarland et al. (Editors), American Petroleum Institute, Publisher. Washington D.C. May.
- Blackburn, G.R., R.A. Deitch, and T.A. Roy et al. 1988. Estimation of the Dermal Carcinogenic Potency of Petroleum Fractions Using a Modified Ames Assay. In: *Polynuclear Aromatic Hydrocarbons: A Decade of Progress, Proceedings of the Tenth International Symposium*. M. Cooke and A.J. Dennis, Editors. Battelle Press, Columbus Ohio, 99 83-97.
- Carpenter, C.P., D.L. Geary, and R.C. Meyers et al. 1976. Petroleum Hydrocarbon Toxicity Studies XI. Animal and Human Response to Vapors of Deodorized Kerosene. *Toxicol. Appl. Pharmacol.* 36:443-456.
- Conaway, C.C., C.A. Schreiner, and S.T. Cragg. 1982. Mutagenicity Evaluation of Petroleum Hydrocarbons. In: *Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons*. H.N. MacFarland et al. (Editors), American Petroleum Institute, Publisher. Washington D.C. May.
- Crisp, A.J., A.K. Bhalla, and B.I. Hoffbrand. 1979. Acute Tubular Necrosis After Exposure to Diesel Oil. *Brit. Med J.* 2:177-178.
- Dalbey, W., M. Henry, and R. Holmberg et al. 1987. Role of Exposure Parameters in the Toxicity of Aerosolized Diesel Fuel in the Rat. *J. Appl. Toxicol.* 7:265-275.
- Gosselin, R.E., R.P. Smith, and H.C. Hodge. 1984. *Clinical Toxicology of Commercial Products*. 5th Ed. Baltimore, Maryland: Williams & Wilkins.

- HEAST. 1990. Health Effects Assessment Summary Tables, Office of Emergency and Remedial Response, Environmental Protection Agency. OERR 9200 6-303 (90-4).
- HSDB. 1991. Hazardous Substances Data Bank. National Library of Medicine. Washington D.C.
- IARC. 1989. Monographs on the Evaluation of Carcinogenic Risks to Humans. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels. International Agency for Research on Cancer, Lyon France. Vol 45.
- Kainz, R.J. and L.E. White. 1982. Consequences Associated with the Inhalation of Uncombusted Diesel Vapor. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (Editors), American Petroleum Institute, Publisher. Washington D.C. May.
- Kirk Othmer. 1984. Diesel Fuel. In: Kirk-Othmer Encyclopedia of Chemical Technology. F. Mark, M. Grayson, and D. Eckroth et al., Eds. 3rd Ed. John Wiley and Sons, New York.
- Kryzanovskij, N.V. 1971. Occupational Health Conditions Associated with the Cleaning of Oil Tankers and Their Effect on Worker's Health. Gig. Tr. Prof. Zabol. 15:14-17.
- Lee, T. and W. Seymour. 1979. Pneumonitis Caused by Petrol Siphoning. (Letter). The Lancet 8134:149.
- Lewis, S.C., R.W. King, S.T. Cragg, and D.W. Hillman. 1982. Skin Carcinogenic Potential of Petroleum Hydrocarbons. 2. Carcinogenesis of Crude Oil, Distillate Fractions and Chemical Class Subfractions. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (Editors), American Petroleum Institute, Publisher. Washington D.C. May.
- NIOSH. 1977. Criteria for a Recommended Standard...Occupational Exposure to Refined Petroleum Solvents. National Institute for Occupational Safety and Health, Cincinnati, Ohio, NIOSH Publication #77-192.
- Rothman, N. and E.A. Emmett. 1988. The Carcinogenic Potential of Selected Petroleum-Derived Products. Occup. Med.: State Art Rev. 3:475-482.
- Siemiatycki, J., R. Dewar, and L. Nadon et al. 1987. Associations Between Several Sites of Cancer and Twelve Petroleum Derived Liquids. Scan. J. Work Environ. Health. 13:493-504.
- Song, H.G. 1988. Petroleum Hydrocarbons in Soil: Biodegradation and Effects on the Microbial Community. Ph.D. Thesis, University of Medicine and Dentistry of New Jersey.
- Witschi, H.P., L.H. Smith, and E.L. Frome et al. 1987. Skin Tumorigenic Potential of Crude and Refined Coal Liquids and Analogous Petroleum Products. Fund. Appl. Toxicol. 9:297-303.

BENZENE

Benzene is readily absorbed following oral and inhalation exposure (EPA 1985). The toxic effects of benzene in humans and other animals following exposure by inhalation include central nervous system effects, hematological effects, and immune system depression. In humans, acute exposures to high concentrations of benzene vapors have been associated with dizziness, nausea, vomiting, headache, drowsiness, narcosis, coma, and death (NAS 1976). Chronic exposure (at least 20 years of worker exposure) to benzene vapors [1 to 100 ppm 8-hour time-weighted-average (TWA)] can produce reduced leukocyte, platelet, and red blood cell counts (EPA 1993). Benzene induced tumors of the zymal gland, oral cavity, leukemia, and lymphoma in rodents chronically exposed by gavage to doses in the range of 25-500 mg/kg/day (Huff et al. 1989; NTP 1986; Maltoni et al. 1989). Many studies have also described a causal relationship between exposure to benzene by inhalation (either alone or in combination with other chemicals) and leukemia in humans (IARC 1982; Rinsky et al. 1981; Ott et al. 1978; Wong et al. 1983).

Applying EPA's criteria for evaluating the overall evidence of carcinogenicity to humans, benzene is classified in Group A (Human Carcinogen) based on adequate evidence of carcinogenicity from epidemiological studies. EPA (1993) derived an oral cancer slope factor of 2.9×10^{-2} (mg/kg/day)⁻¹ and an inhalation unit risk of 8.3×10^{-6} (ug/m³)⁻¹ for benzene. These values were based on several studies in which increased incidence of nonlymphocytic leukemia were observed in humans occupationally exposed to benzene principally by inhalation (Rinsky et al. 1981; Ott et al. 1978; Wong et al. 1983). Equal weight was given to cumulative dose and weighted cumulative dose as well as to relative and absolute risk model forms (EPA 1993). EPA (1993) is currently reviewing both oral and inhalation RfDs for benzene, for which the status is pending.

The National Research Council's Committee on Toxicology has set a one-hour Emergency Exposure Guidance Level (EEGL), for benzene at 50 ppm (200 mg/m³) (NRC 1986). Formerly known as EEL, the EEGL is defined as a ceiling limit for an unpredicted single exposure lasting one to 24 hours, which is expected to be rare in the lifetime of any person. It is designed to avoid substantial decrements in performance during emergencies and takes into account the statistical likelihood of a non-incapacitative, reversible effect in exposed populations (NRC 1986). A health criterion for acute inhalation exposure to benzene of 20 mg/m³ can be derived from the EEGL by combining it with a safety factor of 10 to account for the healthy worker effect, which assumes employed persons are generally healthier than the general population.

REFERENCES

- Environmental Protection Agency (EPA). 1985. Drinking Water Criteria Document for Benzene (Final Draft). Office of Drinking Water, Washington D.C. April 1985.
- Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.
- Huff, J.E., J.K. Haseman, and D.M. Demarini et al. 1989. Multiple-Site Carcinogenicity of Benzene in Fischer 344 Rats and B6C3F1 Mice. Environ. Health Perspect. 82:125-163.

- International Agency for Research On Cancer (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France. Vol. 27.
- Maltoni, C., B. Conti, and G. Cotti et al. 1989. Benzene: A Experimental Multipotential Carcinogen. Results of Long-Term Bioassays Performed at the Bologna Institute of Oncology. Environ. Health Perspect. 82:109-124.
- National Academy of Science (NAS). 1976. Health Effects of Benzene: A Review Committee on Toxicology, Assembly of Life Sciences. National Research Council, Washington D.C.
- National Research Council (NRC). 1986. Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants. Volume 6. Prepared by the Committee on Toxicology. National Academy Press, 1986.
- National Toxicology Program (NTP). 1986. NTP-Technical Report Series No. 289. Toxicology and Carcinogenesis Studies of Benzene in F344/N Rats and B6C3F1 Mice (Gavage Studies). Research Triangle Park, North Carolina: U.S. Department of Health and Human Services, Public Health Service, National Institute of Health. NIH publication No. 86-2545.
- Ott, M.G., J.C. Townsend, W.A. Fishbeck, and R.A. Langner. 1978. Mortality Among Individuals Occupationally Exposed to Benzene. Arch. Environ. Health 33:3-10.
- Rinsky, R.A., R.J. Young, and A.B. Smith. 1981. Leukemia in Benzene Workers. Am. J. Ind. Med. 3:217-245.
- Wong, O., R.W. Morgan, and M.D. Whorton. 1983. Comments on the NIOSH Study of Leukemia in Benzene Workers. Technical Report Submitted to Gulf Canada, Ltd., by Environmental Health Associates.

TOXICOLOGY PROFILE FOR WASTE OIL (RRPH)

GENERAL DATA

"Waste Oil" is a generic term commonly used to describe a range of petroleum compounds from heavy fuels to grease.

As much as 1 to 2 percent of the world's crude oil is refined to produce lubricating oil (24 million tons) (Vazquez-Duhalt 1989). The composition of waste oil is extremely variable and depends upon the original crude oil source, type of processing and refining, blending, additives and use history. Waste oil may therefore range from virgin product accidentally spilled to used machine or automotive oil.

Petroleum oils are produced from the middle to heavy distillate fractions of crude oil. Because of the high boiling points for these fractions, the aromatic hydrocarbons benzene, ethylbenzene, toluene and xylenes, typically found in lighter fuels will not be present in oils in high concentrations. These fractions may be further processed or treated to remove unwanted materials such as nitrogen, sulfur, metals, or polynuclear aromatic hydrocarbons (PAHs). For the most part, oils destined for the consumer market have been laundered to a very low content of PAHs. However, oils in refinery spills may contain several hundred ppm of PAHs. Used motor oil contains Pb, Zn, Cu, Cd, Cr, Ni, and other metals. Lead is the most abundant metal in motor oil and may be present at a concentration high as one percent (Vazquez-Duhalt 1989).

FATE AND TRANSPORT

Oil with characteristics (e.g. vapor pressure, viscosity) closer to fuel oils may volatilize to some extent to the air. However, microbial degradation will, more than likely, be the primary mechanism for the mineralization of spilt material. As much as 90 percent of the material resembling jet fuel may be removed by a combination of evaporation and microbial degradation with a half-life of 1 to 8 weeks; in contrast, heavier fractions that resemble bunker oil (C₁₅ and above) may be degraded only 25 to 30 percent and may be extremely persistent in soils (Song 1988). Given the correct circumstances, waste oils can percolate through the soil and float on the ground water. When spilled onto surface water, waste oils can be toxic to fish, waterfowl and algae, but this is highly dependent upon the characteristics of the oily material and the size of the spill.

Approximately 30 percent of waste motor oil and lubricants produced are released into the environment. Because of the large quantities involved, the persistence of oil residues in the environment, and the potential for ecotoxicity, waste oils are an important environmental concern (Vazquez-Duhalt 1989).

When oil is spilt onto soil, it fills the spaces between the soil particles and hampers oxygen access, thereby promoting anaerobic zones. On the periphery of these oil-soiled zones, aerobic bacteria are promoted. Hence, these outer zones show increased nitrifying, denitrifying, ammonifying, and hydrocarbon-oxidizing microorganisms. The activity of these organisms in the outer zones increases the concentration of easily accessible substrates which stimulates an increase in the numbers of anaerobic nitrogen fixing bacteria (Vazquez-Duhalt 1989). Thus, under certain circumstances, oil addition to soil can function as an amendment and increase the productivity of the soil.

TOXICITY DATA

Human Toxicology

In general, most oily materials derived from petroleum have a low order of toxicity. Inhalation of components of waste oil at concentrations sufficient to achieve a level of intoxication is not likely at normal temperatures and pressures. An attempt to generate a kerosene (diesel) laden atmosphere only resulted in an ambient concentration of 14 ppm (Carpenter et al. 1976). However, under certain occupational settings like tank cleaning, it may be possible to generate mists or aerosols that can lead to symptoms of overexposure. These symptoms may include headache, dizziness, nausea, gastrointestinal symptoms, shortness of breath, weakness, confusion, drowsiness, and possibly death (HSDB 1991). A single report considered chronic repeated exposure to an oil mist for 17 years to be the cause of lipid pneumonia in workers "heavily" exposed (Proctor et al. 1989).

Ingestion of petroleum waste oil (other than fuel oil) either accidentally, intentionally or from contaminated well-water is not expected to have a significant effect except possibly induction of gastrointestinal hypermotility and diarrhea (MacFarland et al. 1982), and nausea and vomiting. Ingestion of the heavier waste oils is not expected to be complicated by aspiration into the lung, which produces a potentially lethal hemorrhagic pneumonitis (Lee and Seymour 1979).

Mammalian Toxicology and Significant Studies

The acute oral and dermal LD₅₀ of petroleum waste oil is expected to be greater than 5 g/kg or practically non toxic. Diesel fuel has an acute oral LD50 is in the range of 9 ml/kg body weight. New or used motor oil has an LD50 of 25 ml/kg, as does heavy fuel #6. Other properties such as eye irritation have ratings of practically non irritating to mildly irritating. Skin irritation scores are similarly low ranging from non-irritating to mildly irritating (Beck et al. 1982). There are no readily available reports on hypersensitivity responses to waste oils, but sensitization is an expected effect because refined products in this distillation range have been reported to produce hypersensitivity reactions (Beck et al. 1982). Dermal absorption of oil can also be expected but the oil itself is unlikely to be the cause of systemic toxicity. Any toxicity is more likely to be attributable to a concomitant absorption of some oil contaminant.

Exposure of CD-1 mice to diesel vapor for eight hours per day on five consecutive days resulted in a decrement of performance on the roto-rod test, square box activity test and hot plate test. However, the corneal reflex and inclined plane test was unaffected. General observations included vasodilation, ataxia, poor grooming and in some cases tremor (Kainz and White 1982).

Exposure of rats to aerosolized diesel fuel at concentrations up to 6 mg/L produced direct toxic effects on the lungs but did not produce any neurotoxicity (Dalbey et al. 1987).

Reproductive Toxicity

Female rats were exposed 6 hours per day to air concentrations of 0, 100, and 400 ppm during days 6 through 15 of gestation. Neither jet fuel or number 2 fuel oil produced any significant detrimental effects on the reproductive parameters of the experimental animals (Beliles and Mecler 1982). Neither Jet Fuel A or diesel fuel at exposure levels of 400 ppm, 6 hours per day, 5 days per week for 8 weeks reduced the fertility of CD-1 male mice (API 1980a,b).

External application of new or used motor oil to the egg shell of a number of bird species caused embryotoxicity and lethality. The used motor oil was more toxic than the new motor oil (Hoffman et al. 1982).

Genotoxicity

Ames testing of several common fuel oils produced mainly negative results. However, the "Modified Ames Assay" introduced by Blackburn et al. (1988) did demonstrate mutagenicity in two straight run gas oils that were previously considered to be negative. Diesel fuel was negative in a mouse lymphoma assay but positive on the rat bone marrow cytogenetics assay when administered by intraperitoneal injection (Conaway et al. 1982). Heating oil #2 did produce a positive Ames test and positive results in two other short term bioassays (Rothman and Emmett 1988).

Used motor oil has been shown to be highly mutagenic to Salmonella bacteria (Peake and Parker 1980). New crankcase motor oil initially tested negative with the standard Ames Assay but after an extraction procedure to remove "interfering chemicals", a dose dependent mutagenic response was observed with both gasoline and diesel crankcase oils. The extracts of the new motor oils, however, are considerably less mutagenic than the Used Crankcase Oil extracts. This effect can be explained by the fact that, during engine operation, the oil accumulates combustion dust and PAH formed in the combustion process or directly from the fuel (Thony et al. 1975). Extracts from the diesel and gasoline type engines were about equally potent (Dutcher et al. 1986).

Carcinogenicity

In classical mouse skin-painting bioassays, all petroleum fractions derived from a crude oil source that boiled between 120 and 700°F showed a low level of tumorigenic activity (Lewis et al. 1982). Home heating oil also showed a low degree of tumorigenicity in a more recent mouse skin-painting assay (Witschi et al. 1987). Topical application of used motor oil from gasoline driven vehicles increased the incidence of local tumors in a dose related fashion. The application of new motor oil to mouse skin did not induce skin tumors (Saffiotti and Shubik 1963). This information plus the demonstrated mutagenic potential of used motor oils and their PAH content, allows a determination that such oils can be considered to be potentially carcinogenic (IARC 1984).

In a case referent study, Seimietycki et al. (1987) reported an increase of several specific cancers associated with exposures to different petroleum products. Leaded gasoline was associated with stomach cancer; aviation gasoline with kidney cancer; diesel fuel with non adenocarcinoma of the lung and prostate cancer; and mineral spirits with squamous cell lung cancer. However not all parameters of concern were properly controlled, excluded, or assessed, making conclusions from this study unreliable.

IARC (1989) has classified gasoline, diesel fuel, and residual oil Category 2B, having limited evidence of carcinogenicity in animals and inadequate evidence in humans. Used motor oil (crankcase oil) is also classified as a category 2B. Light fuel oils, crude oil, and jet fuels have been classified as Category 3, having inadequate evidence of carcinogenicity in either animals or humans.

REGULATIONS AND STANDARDS

Neither the American Conference of Governmental Industrial Hygienists (ACGIH) nor OSHA have recommended or established permissible exposure standards (PELs) for diesel fuels or waste oils. NIOSH has recommended a 10 hour TWA of 100 mg/m³ for kerosene or 14 ppm (MMWR. 37:24). The ACGIH (1991) and OSHA (1985) recommend a TLV of 5 mg/m³ for Oil Mists.

Diesel fuels are not mentioned in HEAST (1990), nor identified for a specific cancer Slope Factor (CSF) or reference dose (RfD). However, individual components such as benzene, other aromatics, and n-hexane that have CSF or RfD values should be evaluated individually.

REFERENCES

- API. 1980a. Mutagenicity Evaluation of Jet Fuel A in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-03. American Petroleum Institute Medical Research Publications, Washington D.C.
- API. 1980b. Mutagenicity Evaluation of Diesel Fuel in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-04. American Petroleum Institute Medical Research Publications, Washington D.C.
- Beck, L.S., D.I. Hepler, and K.L. Hansen. 1982. The Acute Toxicology of Selected Petroleum Hydrocarbons. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (Editors), American Petroleum Institute, Publisher. Washington D.C. May.
- Beliles, R.P. and F.J. Mecler. 1982. Inhalation Teratology of Jet Fuel, Fuel Oil, and Petroleum Naphtha in Rats. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (Editors), American Petroleum Institute, Publisher. Washington D.C. May.
- Blackburn, G.R., R.A. Deitch, and T.A. Roy et al. 1988. Estimation of the Dermal Carcinogenic Potency of Petroleum Fractions Using a Modified Ames Assay. In: Polynuclear Aromatic Hydrocarbons: A Decade of Progress, Proceedings of the Tenth International Symposium. M. Cooke and A.J. Dennis, Editors. Battelle Press, Columbus, Ohio. Pp. 83-97.
- Carpenter, C.P., D.L. Geary, and R.C. Meyers et al. 1976. Petroleum Hydrocarbon Toxicity Studies XI. Animal and Human Response to Vapors of Deodorized Kerosene. Toxicol. Appl. Pharmacol. 36:443-456.
- Clayton, G.D. and F.E. Clayton. 1982. Patty's Industrial Hygiene and Toxicology. 3rd Edition. G.D. Clayton and F.E. Clayton, Editors. John Wiley and Sons, Publishers, New York, New York.
- Conaway, C.C., C.A. Schreiner, and S.T. Cragg. 1982. Mutagenicity Evaluation of Petroleum Hydrocarbons. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (Editors), American Petroleum Institute, Publisher. Washington D.C. May.

- Dalbey, W., M. Henry, and R. Holmberg et al. 1987. Role of Exposure Parameters in the Toxicity of Aerosolized Diesel Fuel in the Rat. *J. Appl. Toxicol.* 7:265-275.
- Dutcher, S.J., A.P. Li, and R.O. McClellan. 1986. Mutagenicity of Used Crankcase Oils from Diesel and Spark Ignition Automobiles. *Environ. Res.* 40:155-163.
- HEAST. 1990. Health Effects Assessment Summary Tables, EPA, OERR 9200 6-303, (90-4).
- Hoffman, D.J., W.C. Eastin, and M.L. Gay. 1982. Embryotoxic and Biochemical Effects of Waste Crankcase Oil on Bird Eggs. *Toxicol. Appl. Pharmacol.* 63:230-241.
- HSDB. 1990. Hazardous Substances Data Bank, National Library of Medicine. Washington D.C.
- IARC. 1984. International Agency for Research on Cancer. Vol 33. Polynuclear Aromatic Hydrocarbons: Part 2. Carbon Black, Mineral Oils and Some Nitroarenes. World Health Organization, Lyon, France.
- IARC. 1989. International Agency for Research on Cancer. Vol 45. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels. World Health Organization, Lyon, France.
- Kainz, R.J. and L.E. White. 1982. Consequences Associated with the Inhalation of Uncombusted Diesel Vapor. In: *Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons*. H.N. MacFarland et al. (Editors), American Petroleum Institute, Publisher. Washington D.C. May.
- Kirk Othmer. 1984. Diesel Fuel. In: *Kirk-Othmer Encyclopedia of Chemical Technology*. F. Mark, M. Grayson, and D. Eckroth et al., Editors. 3rd Edition. John Wiley and Sons, New York.
- Kryzanovskij, N.V. 1971. Occupational Health Conditions Associated with the Cleaning of Oil Tankers and their Effect on Worker's Health. *Gig. Tr. Prof. Zabol.* 15:14-17.
- Lee, T. and W. Seymour. 1979. Pneumonitis Caused by Petrol Siphoning. *The Lancet* 8134:149.
- Lebowitz H., D. Brusick, and D. Matheson et al. 1979. Commonly Used Fuels and Solvents Evaluated in a Battery of Short-Term Bioassays. *Environ. Mutagen.* 1:172-173.
- Lewis, S.C., R.W. King, S.T. Cragg, and D.W. Hillman. 1982. Skin Carcinogenic Potential of Petroleum Hydrocarbons. 2. Carcinogenesis of Crude Oil, Distillate Fractions and Chemical Class Subfractions. In: *Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons*. H.N. MacFarland et al. (Editors), American Petroleum Institute, Publisher. Washington D.C. May.
- NIOSH. 1977. Criteria for a Recommended Standard...Occupational Exposure to Refined Petroleum Solvents. National Institute for Occupational Safety and Health, Cincinnati, Ohio. NIOSH Publication #77-192.
- OHM TADS. 1991. Oil and Hazardous Materials - Technical Assistance Data Systems. Environmental Protection Agency, Emergency Response Division, Washington D.C.

- Peake, E. and K. Parker. 1980. Polynuclear Aromatic Hydrocarbons and the Mutagenicity of Used Crankcase Oil. In: A. Bjorseth and J. Dennis, Editors. Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effects. Battelle Press, Columbus, Ohio. Pp 1025-1039.
- Rothman, N. and E.A. Emmett. 1988. The Carcinogenic Potential of Petroleum-Derived Products. Chapter 7. In: Occupational Medicine: State of the Art Reviews. 3:475-482.
- Saffiotti, U. and P. Shubik. 1963. Studies on Promoting Action in Skin Carcinogenesis. National Cancer Inst. Monogr. 10:489-507.
- Siemiatycki, J., R. Dewar, and L. Nadon et al. 1987. Associations Between Several Sites of Cancer and Twelve Petroleum Derived Liquids. Scan. J. Work Environ. Health. 13:493-504.
- Song, H.G. 1988. Ph.D. Thesis, University of Medicine and Dentistry of New Jersey. Petroleum Hydrocarbons in Soil: Biodegradation and Effects on the Microbial Community.
- Thony, C., J. Thony, and M. Lafontaine et al. 1975. Concentration En Hydrocarbons Polycycliques Aromatiques Concerogenes Deuel Ques Huiles Minerales. Arch. Mal. Prof. Med. Travail Sec. Soc. 36: 281-300.
- Vazquez-Duhalt, R. 1989. Environmental Impact of Used Motor Oil. Sci. Total Environ. 79:1-23.
- Witschi, H.P., L.H. Smith, and E.L. Frome et al. 1987. Skin Tumorigenic Potential of Crude and Refined Coal Liquids and Analogous Petroleum Products. Fund. Appl. Toxicol. 9:297-303.

1,1,2-TRICHLOROETHANE

1,1,2-Trichloroethane (1,1,2-TCA) is rapidly absorbed from oral, inhalation and dermal exposures (Torkelson and Rowe 1981, Arena 1979). In humans, acute oral and inhalation exposures to 1,1,2-TCA result in central nervous system (CNS) depression, equilibrium disturbances, vertigo, headaches, lassitude, hypotension, anesthesia and coma (Arena 1979). Acute oral and inhalation administration to animals produces liver and kidney damage, irritation to the eyes and nose, CNS depression, and death due to respiratory arrest (ACGIH 1986, Torkelson and Rowe 1981). In dogs the hepatotoxic effects include hepatocyte vacuolation, enzyme induction, fatty degeneration and necrosis (NRC 1977, Torkelson and Rowe 1981). The hepatotoxicity and nephrotoxicity of 1,1,2-TCA has been found to be potentiated by pretreatment with certain halogenated organic compounds and solvents. Subchronic oral administration to mice produced alterations in clinical serum levels indicative of adverse liver effects (White et al. 1985, Sanders et al. 1985). Dermal exposures result in irritation and injury to the skin from defatation (Torkelson and Rowe 1981). Evidence suggests that 1,1,2-TCA is embryo toxic to chicken eggs (Elovaara 1979). 1,1,2-TCA was found to be weakly mutagenic in *S. Cerevisiae* (Torkelson and Rowe 1981). Oral administration of 1,1,2-TCA has been associated with the induction of hepatocellular carcinomas and pheochromocytomas in mice but not in rats (NCI 1978, Weisburger 1977)

EPA classified 1,1,2-TCA in group C (Possible Human Carcinogen). This category applies to agents for which there is limited evidence of carcinogenicity in animals. EPA (1996) derived an oral slope factor of $5.7 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$ based on an increased incidence of liver tumors in mice (NCI 1978). EPA (1996) also established an oral reference dose (RfD) of $4.0 \times 10^{-3} \text{ mg/kg/day}$ for 1,1,2-TCA based upon clinical chemistry alterations in mice given 3.9 mg/kg/day in drinking water (White et al 1985, Sanders et al 1985). An uncertainty factor of 1,000 was used to calculate the RfD.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. ACGIH, Inc., Cincinnati, Ohio

ARENA, J.M. 1979. Poisoning: Toxicology, Symptoms, Treatments. 4th ed., Charles C. Thomas, Publisher, Springfield, Illinois. P. 207

ELOVAARA, E., et al. 1979. Effects of methylene chloride, trichloroethane, trichloroethylene, tetrachloroethylene, and toluene on the development of chick embryos. Toxicology. 12:111

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1996. Integrated Risk Information System (IRIS). Health Criteria and Assessment Officer, Cincinnati, Ohio

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of 1,1,2-trichloroethane for possible carcinogenicity. U.S. Dept. Health, Educ, Welfare. Pub. No. NCI-CG-TR-74

NATIONAL RESEARCH COUNCIL (NRC). 1977. Drinking Water and Health, Volume 1, National Academy Press, Washington, D.C. P. 775-776

SANDERS, V.M., WHITE Jr., K.L., SHOPP Jr., G.M., and MUNSON, A.E. 1985. Humoral and cell mediated immune status of mice exposed to 1,1,2-trichloroethane. Drug Chem. Toxicol 8:357-372

TORKELSON, T.R., AND ROWE, U.K. 1981. Halogenated aliphatic hydrocarbons. In Clayton, G.D., and Clayton, P.B., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol. 2B., p. 3510-3512

WHITE Jr., K.L., SANDERS, V.M., BARNES, V.W., SHOPP Jr., G.M., and MUNSON, A.E. 1985. Toxicology of 1,1,2-trichloroethane in the mouse. Drug Chem. Toxicol 8: 333-355

WEISBURBER, E.K. 1977. Carcinogenicity studies on halogenated hydrocarbons. Environ. Health Perspect. 21:7-16

TRICHLOROETHENE

Absorption of trichloroethene (TCE) from the gastrointestinal tract is virtually complete. Absorption following inhalation exposure is proportional to concentration and duration of exposure (EPA 1985). TCE is a CNS depressant following acute and chronic exposures. In humans, single oral doses of 15 to 25 ml (21 to 35 grams) of TCE have resulted in vomiting and abdominal pain, followed by transient unconsciousness (Stephens 1945). High-level exposure can result in death due to respiratory and cardiac failure (EPA 1985). Hepatotoxicity has been reported in human and animal studies following acute exposure to TCE (EPA 1985). Nephrotoxicity has been observed in animals following acute exposure to TCE vapors (ACGIH 1986; Torkelson and Rowe 1981). Subacute inhalation exposures of mice have resulted in transient increased liver weights (Kjellstrand et al. 1983a,b). Industrial use of TCE is often associated with adverse dermatological effects including reddening and skin burns on contact with the liquid form, and dermatitis resulting from vapors. These effects are usually the result of contact with concentrated solvent, however, and no effects have been reported following exposure to TCE in dilute, aqueous solutions (EPA 1985). TCE has caused significant increases in the incidence of hepatocellular carcinomas in mice (NCI 1976) and renal tubular-cell neoplasms in rats exposed by gavage (NTP 1983), and pulmonary adenocarcinomas in mice following inhalation exposure (Fukuda et al. 1983; Maltoni et al. 1986). TCE was mutagenic in *Salmonella typhimurium* and in *E. coli* (strain K-12), utilizing liver microsomes for activation (Greim et al. 1977).

EPA is currently reviewing the carcinogenicity of TCE. The EPA ECAO currently classifies TCE as a Group B2/C--Probable/Possible Human Carcinogen based on inadequate evidence in humans and sufficient evidence of carcinogenicity from animal studies. ECAO (1992) reported an oral cancer potency factor of $1.1 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$ based on two gavage studies conducted in mice in which increased incidence of liver tumors were observed (Maltoni et al. 1986; Fukuda et al. 1983). An inhalation cancer unit risk of $1.7 \times 10^{-6} \text{ (}\mu\text{g/m}^3\text{)}^{-1}$ has been derived for TCE based on an increased incidence of lung tumors in mice exposed via inhalation (ECAO 1992; NCI 1976). The cancer estimates are currently under review by EPA. EPA (1987) developed an oral RfD of $7.35 \times 10^{-3} \text{ mg/kg/day}$ based on a subchronic inhalation study in rats in which elevated liver weights were observed following exposure to 55 ppm, 5 days/week for 14 weeks (Kimmerle and Eben 1973). A safety factor of 1,000 was used to calculate the RfD. However, this RfD is currently under review by EPA.

The National Research Council's Committee on Toxicology has set a one-hour EEGL, for trichloroethene at 200 ppm ($1,000 \text{ mg/m}^3$) (NRC 1988). Formerly known as EEL, the EEGL is defined as a ceiling limit for an unpredicted single exposure lasting one to 24 hours, which is expected to be rare in the lifetime of any person. It is designed to avoid substantial decrements in performance during emergencies and takes into account the statistical likelihood of a non-incapacitative, reversible effect in exposed populations (NRC 1988). A health criterion for acute inhalation exposure to trichloroethene of 100 mg/m^3 can be derived from the EEGL by combining it with a safety factor of 10 to account for the healthy worker effect, which assumes employed persons are healthier than the general population.

REFERENCES

American Conference of Governmental Industrial Hygienists (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th Ed. ACGIH, Cincinnati, Ohio.

- Environmental Protection Agency (EPA). 1985. Health Assessment Document for Trichloroethylene. Environmental Criteria and Assessment Office. Research Triangle Park, North Carolina. EPA/600/8-82/006F.
- Environmental Protection Agency (EPA). 1987. Health Advisory for Trichloroethylene. Office of Drinking Water, Washington D.C. March 31, 1987.
- Environmental Criteria and Assessment Office (ECAO). 1992. Written correspondence from Joan Dollarhide, Chemical Mixtures Assessment Branch, U.S. Environmental Protection Agency. July 16, 1992.
- Fukuda, K., K. Takemoto, and H. Tsuruta. 1983. Inhalation Carcinogenicity of Trichloroethylene in Mice and Rats. *Ind. Health* 21:243-254.
- Greim, H., D. Bimboes, G. Egert, W. Giggelmann, and M. Kramer. 1977. Mutagenicity and Chromosomal Aberrations as an Analytical Tool for *In Vitro* Detection of Mammalian Enzyme-mediated Formation of Reactive Metabolites. *Arch. Toxicol.* 39:159.
- Kimmerle, G. and A. Eben. 1973. Metabolism, Excretion, and Toxicology of Trichloroethylene After Inhalation. 1. Experimental Exposure on Rat. *Arch. Toxicol.* 30:115.
- Kjellstrand, P., B. Holmquist, N. Mandahl, and M. Bjerkemo. 1983a. Effects of Continuous Trichloroethylene Inhalation on Different Strains of Mice. *Acta Pharmacol. Toxicol.* 53:369-374.
- Kjellstrand, P., B. Holmquist, P. Alm, M. Kanje, S. Romare, I. Jonsson, L. Mansson, and M. Bjerkemo. 1983b. Trichloroethylene: Further Studies of the Effects on Body and Organ Weights and Plasma Butyrylcholinesterase Activity in Mice. *Acta Pharmacol. Toxicol.* 53:375-384.
- Maltoni, C., G. Lefemine, and G. Cotti. 1986. Experimental Research on Trichloroethylene. Carcinogenesis *Arch. Res. Industrial Carcinogenesis Series*. C. Maltoni, M.A. Mehlman, Eds. Vol. V. Princeton Scientific Publishing Co., Inc., Princeton, New Jersey. P. 393.
- National Cancer Institute (NCI). 1976. Carcinogenesis Bioassay of Trichloroethylene. CAS No. 79-01-6. Carcinogenesis Technical Report Series No. 2. PB-264 122.
- National Research Council (NRC). 1988. Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants. Volume 8. Prepared by the Committee on Toxicology. National Academy Press, 1988.
- National Toxicology Program (NTP). 1983. Carcinogenesis Studies of Trichloroethylene (Without Epichlorohydrin), CAS No. 79-01-6, in F344/N Rats and B6C3F₁ Mice (Gavage Studies). Draft. August 1983. NTP 81-84, NTP TR 243.
- Stephens, C. 1945. Poisoning by Accidental Drinking of Trichloroethylene. *Br. Med. J.* 2:218.
- Torkelson, T.R. and V.K. Rowe. 1981. Halogenated Aliphatic Hydrocarbons. In G.D. Clayton and P.B. Clayton, Eds. *Patty's Industrial Hygiene and Toxicology*. 3rd Ed. John Wiley and Sons, New York. Vol. 2B, pp. 3553-3559

TETRACHLOROETHENE

Tetrachloroethene is absorbed following inhalation (IARC 1979) and oral (EPA 1985a,b) exposure. Tetrachloroethene vapors and liquid also can be absorbed through the skin (EPA 1985a,b). The principal toxic effects of tetrachloroethene in humans and animals following acute and longer-term exposures include CNS depression and fatty infiltration of the liver and kidney with concomitant changes in serum enzyme activity levels indicative of tissue damage (EPA 1985a,b; Buben and O'Flaherty 1985). Humans exposed to doses of between 136 and 1,018 mg/m³ for 5 weeks develop central nervous system effects, such as lassitude and signs of inebriation (Stewart et al. 1974). The offspring of female rats and mice exposed to high concentrations of tetrachloroethene for 7 hours daily on days 6 through 15 of gestation developed toxic effects, including a decrease in fetal body weight in mice and a small but significant increase in fetal resorption in rats (Schwetz et al. 1975). Mice also exhibited developmental effects, including subcutaneous edema and delayed ossification of skull bones and sternebrae (Schwetz et al. 1975). In a National Cancer Institute bioassay (NCI 1977), an increased incidence of hepatocellular carcinoma was observed in both sexes of B6C3F1 mice administered tetrachloroethylene in corn oil by gavage for 78 weeks. Increased incidence of mononuclear cell leukemia and renal adenomas and carcinomas (combined) have been observed in long term bioassays in which rats were exposed to tetrachloroethene by inhalation (NTP 1986).

Tetrachloroethene is currently under review by the Carcinogen Risk Assessment Verification Endeavor (CRAVE) and estimates of cancer potency were recently withdrawn by EPA (1992b). However, the EPA Environmental Criteria and Assessment Office (ECAO) (1992a) currently classifies tetrachloroethene as a Group B2/C carcinogen (Probable/Possible Human Carcinogen). ECAO (1992a) has reported an oral slope factor of $5.2 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$ based on liver tumors observed in the NCI (1977) gavage bioassay for mice. An inhalation cancer unit risk of $5.8 \times 10^{-7} \text{ (}\mu\text{g/m}^3\text{)}^{-1}$ is based on an NTP (1986) bioassay in rats and mice in which leukemia and liver tumors were observed (ECAO 1992a). Both the cancer slope factor and unit risk are currently under review by EPA. EPA (1993) also derived an oral RfD of $1 \times 10^{-2} \text{ mg/kg/day}$ for tetrachloroethene based on a 6-week gavage study by Buben and O'Flaherty (1985). In this study, liver weight/body weight ratios were significantly increased in mice and rats treated with 71 mg/kg-day tetrachloroethene but not in animals treated with 14 mg/kg-day. The RfD was derived using a NOAEL of 14 mg/kg/day and applying an uncertainty factor of 1,000. EPA (1992b) established a subchronic oral RfD of $1 \times 10^{-1} \text{ mg/kg/day}$, using an uncertainty factor of 100 based on the same study and effect of concern.

The American Conference of Governmental Industrial Hygienists (ACGIH) has set a Short-Term Exposure Level -- Threshold Limit Value of 200 ppm (1,000 mg/m³) for tetrachloroethene (ACGIH 1991). The STEL-TLV is defined as a 15-minute time-weighted average which should not be exceeded at any time during a work day. A health criterion for acute inhalation exposure to tetrachloroethene of 100 mg/m³ can be derived from the STEL-TLV by combining it with a safety factor of 10 to account for the healthy worker effect which assumes that employed persons are healthier than the general population.

REFERENCES

American Conference of Governmental Industrial Hygienist (ACGIH). 1991. Threshold Limit Values and Biological Exposure Indices for 1990-1991. Cincinnati, Ohio.

- Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the Role of Metabolism in the Hepatotoxicity of Trichloroethylene and Perchloroethylene: A Dose-Effect Study. *Toxicol. Appl. Pharmacol.* 78:105-122.
- Environmental Protection Agency (EPA). 1985a. Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Office of Health and Environmental Assessment, Washington D.C. July 1985. EPA 600/8-82-005F.
- Environmental Protection Agency (EPA). 1985b. Drinking Water Criteria Document for Tetrachloroethylene. Office of Drinking Water, Criteria and Standards Division, Washington D.C. June 1985.
- Environmental Criteria and Assessment Office (ECAO). 1992a. Written Correspondence from Joan Dollarhide, Chemical Mixtures Assessment Branch, U.S. Environmental Protection Agency. July 16, 1992.
- Environmental Protection Agency (EPA). 1992b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Remedial Response, Washington D.C. FY-1992.
- Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.
- International Agency for Research on Cancer (IARC). 1979. IARC Monographs on the Evaluation of the Carcinogenic Risks of Chemicals to Humans. Vol. 20: Some Halogenated Hydrocarbons. World Health Organization, Lyon, France.
- National Cancer Institute (NCI). 1977. Bioassay of Tetrachloroethylene for Possible Carcinogenicity. CAS No. 127-18-4. NCI Carcinogenesis Technical Report Series No. 13, Washington D.C. DHEW (NIH) Publication No. 77-813.
- National Toxicology Program (NTP). 1986. Toxicology and Carcinogenesis Studies of Tetrachloroethylene (Perchloroethylene) (CAS No. 127-18-4) in F344/N Rats and B6C3F1 Mice (Inhalation Studies). NTP Technical Report Series No. 311, Research Triangle Park, North Carolina. DHEW (NIH) Publication No. 86-2567.
- Schwetz, B.A., B.K.J. Leong, and P.J. Gehring. 1975. The Effect of Maternally Inhaled Trichloroethylene, Perchloroethylene, Methyl Chloroform, and Methylene Chloride on Embryonal and Fetal Development in Mice and Rats. *Toxicol. Appl. Pharmacol.* 55:207-219.
- Stewart, R.D., C.L. Hake, H.V. Forster, A.J. Lebrun, J.F. Peterson, and A. Wu. 1974. Tetrachloroethylene: Development of a Biologic Standard for the Industrial Worker by Breath Analysis. Medical College of Wisconsin, Milwaukee, Wisconsin. NIOSH-MCOW-ENUM-PCE-74-6.

BARIUM

Barium and compounds are absorbed to a limited extent following oral and inhalation exposures (ATSDR 1992). Respiratory weakness, paralysis, hypertension and abnormalities in heart rhythm have been frequently observed following acute ingestion (ATSDR 1992). In occupationally exposed workers, inhalation of barium sulfate has been associated with a minor lung effect called baritosis, a benign pneumoconiosis (Doig 1976; Goyer 1986). The effects of elevated barium levels in drinking water on blood pressure, hypertension, stroke, heart disease, and altered electrocardiograms have been investigated in several epidemiological studies. No adverse effects were found with barium concentrations as high as 10 mg/L (0.21 mg/kg/day) (Brenniman and Levy 1984, 1985; Brenniman et al. 1979a,b, 1981; Wones et al. 1990). Subchronic and chronic oral exposure of rats to barium (as high as 7.1 mg/kg/day for one month and as low as 0.54 mg/kg/day for 16 months), however, resulted in increased blood pressure and cardiovascular cellular changes (Kopp et al. 1985; Perry et al. 1983, 1985, 1989). One animal study did not result in adverse effects at the highest dose tested of 15 mg/kg/day (McCauley et al. 1985); two other studies did not indicate adverse effects but did not measure blood pressure (Schroeder and Mitchener 1975a,b; Tardiff et al. 1980). Subchronic inhalation of barium carbonate dust (3.6 mg/m^3) by experimental animals has been associated with reduced sperm count, increased fetal mortality, atresia of the ovarian follicles, decreased body weight, and alterations in liver function (Tarasenko et al. 1977).

EPA (1993a) derived an oral reference dose (RfD) based on a weight-of-evidence approach that takes into account recent findings of two human epidemiologic studies (Brenniman and Levy 1984; Wones et al. 1990) as well as various rodent studies (Perry et al. 1983; McCauley et al. 1985; Schroeder and Mitchener 1975a,b; Tardiff et al. 1980). No single study was appropriate to calculate a lifetime RfD for barium (EPA 1993a). A LOAEL was not identified in either of the two epidemiological studies; however, the effect of concern in the animal studies was high blood pressure. Using a NOAEL of 0.21 mg/kg/day (Wones et al. 1990) and an uncertainty factor of 3, an oral RfD of $7 \times 10^{-2} \text{ mg/kg/day}$ was calculated. The chronic RfD was adopted as a subchronic RfD by EPA (1993b). EPA (1993b) has also developed chronic and subchronic inhalation RfCs of 5×10^{-4} and $5 \times 10^{-3} \text{ mg/m}^3$ for barium based on a study by Tarasenko et al. (1977). In this study, rats were exposed to barium carbonate dust at airborne concentrations of up to 5.2 mg/m^3 for 4 to 6 months. Adverse effects noted at this concentration included decreased body weight, alterations in liver function, and increased fetal mortality. Uncertainty factors of 1,000 and 100 were used in developing the chronic and subchronic RfCs, respectively.

REFERENCES

- Agency for Toxic Substances and Disease Control (ATSDR). 1992. Toxicological Profile for Barium and Compounds. U.S. Public Health Center, Center for Disease Control, Research Triangle Park, North Carolina.
- Brenniman G.R. and P.S. Levy. 1984. High Barium Levels in Public Drinking Water and its Association with Elevated Blood Pressure. In: *Advances in Modern Toxicology IX*, E.J. Calabrese, Ed. Princeton Scientific Publication, Princeton, New Jersey. P 231-249.
- Brenniman G.R. and P.S. Levy. 1985. Epidemiological Study of Barium in Illinois Drinking Water Supplies. In: *E.J. Calabrese, R.W. Tuthill, and L. Condie, Eds. Inorganics in Water and Cardiovascular Disease*. Princeton, New Jersey: Princeton Scientific Publishing Co. P. 231-240.

- Brenniman, G.R., W.H. Kojola, and P.S. Levy et al. 1979a. Health Effects of Human Exposure to Barium in Drinking Water. Cincinnati, Ohio: U.S. Environmental Protection Agency, Office of Research and Development, Health Effects Research Laboratory. EPA/600/1-79/003.
- Brenniman, G.R., T. Namekata, and W.H. Kojola et al. 1979b. Cardiovascular Disease Death Rates in Communities with Elevated Levels of Barium in Drinking Water. *Environ. Res.* 20:318-324.
- Brenniman, G.R., W.H. Kojola, and P.S. Levy et al. 1981. High Barium Levels in Public Drinking Water and its Association with Elevated Blood Pressure. *Arch. Environ. Health* 36:28-32.
- Doig, A.T. 1976. Baritosis: A Benign Pneumoconiosis. *Thorax* 31:30-39.
- Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Barium. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-021.
- Environmental Protection Agency (EPA). 1993a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.
- Environmental Protection Agency (EPA). 1993b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. FY-1993.
- Goyer, R.A. 1986. Toxic Effects of Metals. In C.D. Klaasen, M.D. Amdur, and J. Doull, eds. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 3rd Ed. Macmillan Publishing Co., New York. Pp. 623-624.
- Kopp, S.J., H.M. Perry, Jr., and J.M. Feliksik et al. 1985. Cardiovascular Dysfunction and Hypersensitivity to Sodium Pentobarbital Induced by Chronic Barium Chloride Ingestion. *Toxicol. Appl. Pharmacol.* 77:303-314.
- Mc Cauley, P.T., B.H. Douglas, R.D. Laurie, and R.J. Bull. 1985. Investigations into the Effect of Drinking Water Barium on Rats. *Environ. Health. Perspect.* Vol. IX, E.J. Calabrese, Ed. Princeton Scientific Publications, Princeton, New Jersey. p. 197-210.
- Perry, H.M., S.J. Kopp, M.W. Erlanger, and E.G. Perry. 1983. XVII. Cardiovascular Effects of Chronic Barium Ingestion. In D.D. Hemphill, ed. *Proceedings of the University of Missouri's 17th Annual Conference of Trace Substances in Environmental Health*. University of Missouri Press, Columbia, Missouri (As cited in EPA 1984).
- Perry, H.M. Jr., E.F. Perry, and M.W. Erlanger et al. 1985. Barium-induced Hypertension. *Adv. Mod. Environ. Toxicol.* 9:221-229.
- Perry, H.M. Jr., S.J. Kopp, and E.F. Perry et al. 1989. Hypertension and Associated Cardiovascular Abnormalities Induced by Chronic Barium Feeding. *J. Toxicol. Environ. Health* 28:373-388.

- Schroeder, H.A. and M. Mitchener. 1975a. Life-term Effects of Mercury, Methyl Mercury and Nine Other Trace Metals on Mice. J. Nutr. 105:452-458.
- Schroeder, H.A. and M. Mitchener. 1975b. Life-term Studies in Rats: Effects of Aluminum, Barium, Beryllium and Tungsten. J. Nutr. 105:421-427.
- Tarasenko, M., O. Promin, and A. Silayev. 1977. Barium Compounds as Industrial Poisons (An Experimental Study). J. Hyg. Epiderm. Microbial. Immunol. 21:361 (As cited in EPA 1984).
- Tardiff, R.G., M. Robinson, and N.S. Ulmer. 1980. Subchronic Oral Toxicity of BaCl₂ in Rats. J. Environ. Pathol. Toxicol. 4:267-275.
- Wones, R.G., B.L. Stadler, and L.A. Frohman. 1990. Lack of Effect of Drinking Water Barium on Cardiovascular Risk Factor. Environ Health Perspective. 85:1-13.

1,1-DICHLOROETHENE

1,1-Dichloroethene (1,1-DCE) is rapidly absorbed after oral and inhalation exposure (EPA 1984 1987). Studies in animals (mice and rats) indicate that 81 to 99.8 percent of 1,1-DCE is absorbed from the gastrointestinal tract following oral administration (ATSDR 1992). Humans acutely exposed to 1,1-DCE vapors exhibit central nervous system depression. In animals, the liver is the principal target organ of 1,1-DCE toxicity. Acute exposures may result in liver damage ranging from fatty infiltration to necrosis (EPA 1987). Rodents acutely exposed to 1,1-DCE concentrations of 500 to 1,500 ppm experienced irritation of the mucous membranes and pulmonary edema (ATSDR 1992). Workers chronically exposed to 1,1-DCE in combination with other vinyl compounds exhibit liver dysfunction, headaches, vision problems, weakness, fatigue, and neurological sensory disturbances (EPA 1987). Chronic oral administration of 1,1-DCE to experimental animals results in both hepatic and renal toxicity (EPA 1984; Quast et al. 1983). 1,1-DCE appears to produce weak teratogenic effects in laboratory animals (Short et al. 1977). Inhalation exposure of rats, mice, and rabbits has produced fetotoxicity and minor skeletal abnormalities, but usually at maternally toxic doses. Skeletal anomalies in rats and mice and soft tissue anomalies in rats were observed at the low dose (15 ppm) (Short et al. 1977). 1,1-DCE vapors produced renal adenocarcinomas in a single study of mice exposed by inhalation, but the results of other studies were equivocal or negative (EPA 1987; Maltoni et al. 1985). A number of chronic studies in rats and mice have evaluated the carcinogenicity of 1,1-DCE from oral doses of 0.5 to 40 mg/kg/day. These studies have not produced statistically significant changes in the tumor incidence of treated animals (ATSDR 1992).

EPA has classified 1,1-DCE as a Group C agent (Possible Human Carcinogen) and has developed an inhalation unit risk of $5 \times 10^{-5} (\text{ug}/\text{m}^3)^{-1}$ and oral cancer slope factor of $0.6 (\text{mg}/\text{kg}/\text{day})^{-1}$ (EPA 1993). The inhalation unit risk is equivalent to a slope factor of 1.2 ($\text{mg}/\text{kg}/\text{day})^{-1}$. The inhalation slope factor is based on a metabolized dose (15 percent of administered dose) and an increased incidence of renal adenocarcinomas in male mice exposed to 1,1-DCE via inhalation for 52 weeks and observed for a total of 121 weeks (Maltoni et al. 1985). The oral slope factor was derived by estimating an upper-limit value from negative bioassay data and assuming that a carcinogenic response occurs via ingestion, although there is no direct evidence that this is true.

EPA developed an oral reference dose (RfD) of $9 \times 10^{-3} \text{ mg}/\text{kg}/\text{day}$ for both chronic (EPA 1993) and subchronic (EPA 1992) exposure based on the occurrence of hepatic lesions in rats chronically exposed to 1,1-DCE in drinking water (Quast et al. 1983). A safety factor of 1,000 was applied to the lowest-observed-adverse-effect level (LOAEL) of 9 mg/kg/day to derive the chronic and subchronic oral RfDs.

REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological Profile for 1,1-Dichloroethene. U.S. Department of Health and Human Services. Draft for Public Comment. October.
- Environmental Protection Agency (EPA). 1984. Health Effects Assessment for 1,1-Dichloroethylene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-051.

- Environmental Protection Agency (EPA). 1985. Health Assessment Document for Vinylidene Chloride. Final Report. Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. August 1985. EPA 600/8-83/031F.
- Environmental Protection Agency (EPA). 1987. Health Advisory for 1,1-Dichloroethene. Office of Drinking Water, Washington D.C. March 31, 1987.
- Environmental Protection Agency (EPA). 1992. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington D.C. FY-1992.
- Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.
- Maltoni, C., G. Lefemine, G. Cotti, P. Chieco, and V. Patella. 1985. Experimental Research on Vinylidene Chloride Carcinogenesis. In Archives of Research on Industrial Carcinogenesis. Princeton Scientific Publishers, Princeton, New Jersey. 3 vols.
- Quast, J.F., C.G. Humiston, C.E. Wade, J. Ballard, J.E. Beyer, R.W. Schwetz, and J.M. Norris. 1983. A Chronic Toxicity and Oncogenicity Study in Rats and Subchronic Toxicity Study in Dogs on Ingested Vinylidene Chloride. Fund. Appl. Toxicol. 3:55-62.
- Short, R.D., J.L. Minor, and J.M. Winston et al. 1977. Toxicity Studies of Selected Chemicals: Task II. The Development of Vinylidene Chloride Inhaled by Rats and Mice During Gestation. Washington D.C.: USEPA Publication No. EPA-560/6-77/022 (as cited in ATSDR 1992).

cis-1,2-DICHLOROETHENE

EPA (1984) reported that *cis*-1,2-dichloroethene is expected to be absorbed following oral, inhalation, or dermal exposure based on its low molecular weight, lipid solubility, and neutral charge. It is in the same chemical class as 1,1-dichloroethene and is expected to be eliminated via the urine within two to three days of exposure (EPA 1987). *cis*-1,2-Dichloroethene was once used as an anesthetic gas; at high doses general anesthetic and narcotic effects are produced (Irish 1963). Acute exposure to 400 mg/kg of the chemical has caused a significant elevation of liver alkaline phosphatase in rats, which indicates liver toxicity (Jenkins et al. 1972). Subchronic gavage administration to rats resulted in decreased hematocrit and hemoglobin (McCauley et al. n.d). *cis*-1,2-Dichloroethene was not mutagenic in a bacterial assay with or without activation (Greim et al. 1975). It did not induce point mutations, mitotic gene conversions, or mitotic recombinations in yeast (Galli et al. 1982a), nor induce mutations in a host-mediated assay (Galli et al. 1982b).

EPA (1993) calculated an oral RfD for *cis*-1,2-dichloroethene of 1×10^{-2} mg/kg/day based on a study of rats administered *cis*-1,2-dichloroethene by gavage for 90 days (McCauley et al. n.d). Decreased hematocrit and hemoglobin were observed at 32 mg/kg/day. An uncertainty factor of 3,000 was used to calculate the RfD.

REFERENCES

- Environmental Protection Agency (EPA). 1984. Draft Health Effects Criteria Document for the Dichloroethylenes. Criteria and Standards Division, Office of Drinking Water. Washington D.C. December (As cited in EPA 1987).
- Environmental Protection Agency (EPA). 1987. Health Advisory for *cis*-1,2-Dichloroethylene. Office of Drinking Water, Washington D.C. March 31, 1987.
- Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.
- Galli, A., C. Bauer, G. Brenzetti, C. Corsi, R. Del Carratore, R. Nieri, and M. Paolini. 1982a. (a) Studio in vitro. Attivita Genetica Dell' 1,2-Dichloroetilene. Boll. Soc. It. Biol. Sper. 58:860-863 (As cited in EPA 1987).
- Galli, A., C. Bauer, G. Brenzetti, C. Corsi, R. Del Carratore, R. Nieri, and M. Paolini. 1982b. (a) Studio in vivo. Attivita Genetica Dell' 1,2-Dichloroetilene. Boll. Soc. It. Biol. Sper. 58:864-869 (As cited in EPA 1987).
- Greim, H., G. Bonse, Z. Radwan, D. Reichert, and D. Henschler. 1975. Mutagenicity in vitro and Potential Carcinogenicity of Chlorinated Ethylenes as a Function of Metabolic Oxirane Formation. Biochem. Pharmacol. 24:2013-2017 (As cited in EPA 1987).
- Irish, D. 1963. Vinylidene Chloride. In: F.A. Patty (Ed), Industrial Hygiene and Toxicology. 2nd Ed. Vol. II. John Wiley and Sons, Inc., New York. Pp. 1305-1309 (as cited in EPA 1987).
- Jenkins, L., Jr., M. Trabulus, and S. Murphy. 1972. Biochemical Effects of 1,1-Dichloroethylene in Rats: Comparison with Carbon Tetrachloride and 1,2-dichloroethylene. Toxicol. Appl. Pharmacol. 23:501-510 (as cited in EPA 1987).

McCauly, P.T., M. Robinson, L.W. Condie, and M. Parvell. N.D. The Effects of Subacute and Subchronic Oral Exposure to cis-1,2-Dichloroethene in Rats. Health Effects Research Laboratory, U.S. EPA, Cincinnati, Ohio.

DICHLORODIFLUOROMETHANE

The absorption of dichlorodifluoromethane (FC-12) following both oral and inhalation exposure can be inferred from the observed toxicity following these routes of exposure. No quantitative estimates were available in the literature. At high vapor concentrations, FC-12 causes lung irritation (Gosselin et al. 1984). When inhaled, FC-12 has a very low acute toxicity and acts like a weak narcotic (ACGIH 1986). In humans, acute exposure to extremely high FC-12 vapor concentrations was reported to reduce psychomotor performance, increase airway resistance, and cause electrocardiographic changes (Aviado and Micozzi 1981). In animals, FC-12 toxicity is manifested as respiratory and circulatory effects including bronchoconstriction and tachycardia (Aviado and Smith 1975). Other effects include central nervous system depression, enhanced cardiac sensitization of dogs to epinephrine, induced cardiac arrhythmias in anesthetized monkeys, and depression of myocardial contractility in dogs, monkeys and rabbits (Aviado and Micozzi 1981). Longer-term inhalation exposure of guinea pigs continuously for 90 days resulted in microscopic liver injury (Prendergast et al. 1967). Large concentrations (200,000 ppm) produced tremors, mild narcosis and hematological changes in dogs, guinea pigs and monkeys (Sayers et al. 1930). Chronic dietary exposure of rats to 3,000 ppm (150 mg/kg/day) FC-12 resulted in decreased body weight gain (Sherman 1974).

EPA (1993) has derived an oral reference dose (RfD) of 2×10^{-1} mg/kg/day for FC-12 based on reduced body weight observed in a two-year rat feeding study (Sherman 1974). No other adverse effects were observed in the rats or in dogs. The RfD was calculated using a no-observed-adverse-effect level (NOAEL) of 300 ppm (converted to 15 mg/kg/day) and a safety factor of 100. Based on the same study and effects of concern, EPA (1992) derived a subchronic oral RfD of 9×10^{-1} mg/kg/day using an uncertainty factor of 100. EPA (1992) has derived chronic and subchronic inhalation RfCs for FC-12 of 2×10^{-1} mg/m³ and 2 mg/m³, respectively. The RfCs were based on a 6-week inhalation study (Prendergast et al. 1967) showing lesions in the lung and liver and using an uncertainty factor of 10,000 and 1,000, respectively. The chronic inhalation RfC was derived from methodology that is not current with the interim inhalation methodology used by the RfD/RfC work group.

REFERENCES

- American Conference of Industrial Hygienists (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. ACGIH, Cincinnati, Ohio.
- Aviado, D.M. and D.G. Smith. 1975. Toxicology 3:241 (as cited in ACGIH 1986).
- Aviado, D.M. and M.S. Micozzi. 1981. Fluorine-Containing Organic Compounds. In: Patty's Industrial Hygiene and Toxicology. G.D. Clayton and F.E. Clayton, Eds. 3rd Revised Edition. Volume 2B. Toxicology. John Wiley and Sons, New York.
- Environmental Protection Agency (EPA). 1992. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington D.C. FY-1992.
- Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

Gosselin, R.E., R.P. Smith, and H.C. Hodge. 1984. Clinical Toxicology of Commercial Products. 5th Edition. Williams and Wilkins, Baltimore.

Prendergast, J.A., R.A. Jones, L.J. Jenkins, and J. Siegel. 1967. Effects on Experimental Animals of Long-Term Inhalation of Trichloroethylene, Carbon Tetrachloride, 1,1,1-Trichloroethane, Dichlorodifluoromethane and 1,2-Dichloroethylene. Toxicol Appl Pharmacol. 10:270 (as cited in EPA 1992b).

Sayers, R.R., W.P. Yant, J. Chronyak, and H.W. Shoaf. 1930. RI. 3013 U.S. Bureau of Mines (as cited in ACGIH 1986).

Sherman, H. 1974. Long-Term Feeding Studies in Rats and Dogs with Dichlorodifluoromethane (Freon 12 Food Freezant). Haskell Laboratory for Toxicology and Industrial Medicine Report No. 24-74.

MANGANESE

Manganese is considered to be an essential element and among the least toxic of the trace metals (NRC 1989). The oral absorption of dietary manganese ranges from 3 to 10 percent (EPA 1993). However, manganese is absorbed to a greater extent following inhalation exposures. The National Research Council has established a provisional recommended dietary allowance for adults of 2 to 5 mg/day (NRC 1989). The effects following acute exposure to manganese are unknown.

Chronic occupational exposure to manganese dust ($0.02 - 2.6 \text{ mg/m}^3$) has been associated with respiratory symptoms and pneumonitis (Chandra et al. 1981, 1990). Higher levels have been associated with a condition known as manganism, a progressive neurological disease characterized by speech disturbances, tremors, and difficulties in walking. For example, male workers exposed to manganese dioxide, tetroxide and various salts [time-weighted-average (TWA) of total airborne manganese dust ranged from $0.07\text{--}8.61 \text{ mg/m}^3$] experienced an increased incidence of psychomotor disturbances (e.g., reaction time, hand-eye coordination and hand steadiness) (Roels et al. 1987). Other effects observed in humans occupationally exposed to manganese dust include hematological (Chandra et al. 1981; Flinn et al. 1941; Kesic and Hausler 1954), cardiovascular (Saric and Hrustic 1975) and reproductive effects (Cook et al. 1974; Emara et al 1971; Lauwerys et al. 1985; Rodier 1955).

In adults, a safe intake of manganese from dietary sources ranges from 2-10 mg/day (10 mg/day = 0.14 mg/kg/day) (WHO 1973; NRC 1989; Schroeder et al. 1966). Individuals who chronically ingested drinking water from natural wells containing manganese concentrations of 1,600 to 2,300 $\mu\text{g/L}$ (0.06 mg/kg/day), showed a statistically significant increase in minor neurologic effects (neurologic exam scores) (Kondakis et al. 1989). Higher concentrations in drinking water (0.8 mg/kg/day) have resulted in symptoms including lethargy, increased muscle tonus, tremor and mental disturbances (Kawamura et al. 1941).

The apparent differences in manganese toxicity following dietary and drinking water exposures can be attributed to the greater bioavailability of manganese from water (EPA 1993). Chronic oral exposure of rats to manganese chloride can also result in central nervous system dysfunction (Leung et al. 1981; Lai et al. 1982). Chronic inhalation exposure of experimental animals (monkeys, rats, mice, hamsters) has resulted in respiratory effects, however, other studies have demonstrated that these effects may be immunological in origin (ATSDR 1992).

Manganese has not been reported to be teratogenic but it has been observed to cause depressed reproductive performance and reduced fertility in humans and experimental animals (EPA 1984a). Certain manganese compounds have been shown to be mutagenic in a variety of bacterial tests. Manganese chloride and potassium permanganate can cause chromosomal aberrations in mouse mammary carcinomal cells. Manganese was moderately effective in enhancing viral transformation of Syrian hamster embryo cells (EPA 1984a,b).

EPA (1993a) established a weight-of-evidence classification for manganese of D (not classifiable as to human carcinogenicity). EPA (1993a) derived two separate oral reference doses (RfD). The separate RfDs for food and water indicate a potentially higher bioavailability of manganese from drinking water than from the diet. The RfD associated with oral exposure to drinking water is $5 \times 10^{-3} \text{ mg/kg/day}$ based on a no-observed-adverse-effect-level (NOAEL) of $5 \times 10^{-3} \text{ mg/kg/day}$ for humans (Kondakis et al. 1989). EPA (1993a) also derived an RfD of $1.4 \times 10^{-1} \text{ mg/kg/day}$ for manganese in food based on a NOAEL of 0.14 mg/kg/day (10 mg/day) in humans chronically

exposed to dietary levels (WHO 1973; Schroeder et al. 1966; NRC 1989). The effect of concern was on the central nervous system, and an uncertainty factor of one was used to derive both RfDs. The chronic RfD in food was adopted as the subchronic RfD (EPA 1993b). EPA (1993a) derived a chronic inhalation reference concentration (RfC) of 4×10^{-4} mg/m³ based upon an occupational study conducted by Roels et al. (1987) in which respiratory symptoms and psychomotor disturbances were observed. EPA (1993b) adopted the chronic RfC as the subchronic RfC. An uncertainty factor of 900 was used to derive both RfCs.

REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological Profile for Manganese. U.S. Department of Health and Human Services, Center for Disease Control.
- Chandra, S.V., G.S. Shukla, R.S. Striavastava, H. Singh, and V.P. Gupta. 1981. An Exploratory Study of Manganese Exposure to Welders. *Clin. Toxicol.* 18:407-416.
- Cook, D.G., S. Fahn, and K.A. Brait. 1974. Chronic Manganese Intoxication. *Arch. Neurol.* 30:59-64.
- Emara, A.M., S.H. El-Ghawabi, O.I. Madkour, and G.H. El-Sarma. 1971. Chronic Manganese Poisoning in the Dry Battery Industry. *Br. J. Ind. Med.* 28:78-82.
- Environmental Protection Agency (EPA). 1984a. Health Assessment Document for Manganese. Final Report. Environmental Criteria and Assessment Office, Environmental Protection Agency, Cincinnati, Ohio. August 1984. EPA 600/8-83-013F.
- Environmental Protection Agency (EPA). 1984b. Health Effects Assessment for Manganese (and compounds). Environmental Criteria and Assessment Office, Washington D.C. EPA 540/1-86-057.
- Environmental Protection Agency (EPA). 1993a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.
- Environmental Protection Agency (EPA). 1993b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington D.C. FY-1993.
- Flinn, R.H., P.A. Neal, and W.B. Fulton. 1941. Industrial Manganese Poisoning. *J. Ind. Hyg. Toxicol.* 23:374-387.
- Iregren, A. 1990. Psychological Test Performance in Foundry Workers Exposed to Low Levels of Manganese. *Neurotox. Teratol.* 12:673-675.
- Kawamura, R., H. Ikuta, and S. Fukuzumi et al. 1941. Intoxication by Manganese in Well Water. *Kitasato Arch. Exp. Med.* 18:145-149.
- Kesic, B. and V. Hausler. 1954. Hematological Investigation on Workers Exposed to Manganese Dust. *Arch. Ind. Hyg. Occup. Med.* 10:336-343.

- Kondakis, X.G., M. Makris, and M. Leotsinidis et al. 1989. Possible Health Effects of High Manganese Concentration in Drinking Water. *Arch. Environ. Health* 44:175-178.
- Lai, J.C.K., T.K.C. Leung, and L. Lim. 1982. Activities of the Mitochondrial NAD-Linked Isocitric Dehydrogenase in Different Regions of the Rat Brain. Changes in Aging and the Effect of Chronic Manganese Chloride Administration. *Gerontology* 28:81-85.
- Lauwerys, R., H. Roels, and P. Genet et al. 1985. Fertility of Wale Workers Exposed to Mercury Vapor or to Manganese Dust: A Questionnaire Study. *Am. J. Ind. Med.* 7:171-176.
- Leung, T.K.C., J.C.K. Lai, and L. Lim. 1981. The Regional Distribution of Monoamine Oxidase Activities Towards Different Substrates: Effects in Rat Brain of Chronic Administration of Manganese Chloride and of Aging. *J. Neurochem.* 36:2037-2043.
- National Research Council (NRC). 1989. Recommended Dietary Allowances, 10th Ed. Food and Nutrition Board, National Research Council, National Academy Press, Washington D.C. 230-235.
- Rodier, J. 1955. Manganese Poisoning in Moroccan Miners. *Br. J. Ind. Med.* 12:21-35.
- Roels, H., R. Lauwerys, and J.P. Buchet et al. 1987. Epidemiological Survey Among Workers Exposed to Manganese: Effects on Lung, Central Nervous System, and Some Biological Indices. *Am. J. Ind. Med.* 11:307-327.
- Saric, M. and O. Hrusic. 1975. Exposure to Airborne Manganese and Arterial Blood Pressure. *Environ. Res.* 10:314-318.
- Schroeder, H.A., D.D. Balassa, and I.H. Tipton. 1966. Essential Trace Metals in Man: Manganese, a Study in Homeostasis. *J. Chron. Dis.* 19:545-571.
- WHO (World Health Organization). 1973. Trace Elements in Human Nutrition: Manganese. Report of a WHO Expert Committee. Technical Report Service, 532, WHO, Geneva, Switzerland. 34-36.

APPENDIX C
BIOCONCENTRATION FACTOR CALCULATIONS

BIOCONCENTRATION FACTOR CALCULATIONS for ORGANIC CHEMICALS

CALCULATION OF Bv FOR ORGANIC CHEMICALS IN SOIL

COC Organics	log Kow	$1.588 - 0.578 \log Kow$	log Bv	Bv
DRPH	5.30	-1.475	-1.475	0.033
Benzene	2.13	0.357	0.357	2.274
Toluene	2.69	0.033	0.033	1.079
Ethylbenzene	3.15	-0.233	-0.233	0.585
Xylenes (total) ^a	2.77	-0.013	-0.013	0.970
Tetrachloroethene	2.53	0.126	0.126	1.336
Trichloroethane ^b	2.07	0.392	0.392	2.463
Trichloroethene	2.42	0.189	0.189	1.546
Benzyl alcohol	1.01	1.004	1.004	10.098

^a log Kow for ortho-xylene used

^b log Kow for 1,1,2-trichloroethane used

APPENDIX D

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION Point Lay	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
SPECIES brown lemming				
COC				
Inorganics				
Iron	0.00	0.004	0.50	0.000
Lead	60.60	0.045	0.50	1.364
Manganese	0.00	0.250	0.50	0.000
Selenium	17.00	0.025	0.50	0.213
Zinc	96.00	1.500	0.50	72.000
Organics				
DRPH	2886.00	0.03	0.50	48.291
Benzene	0.13	2.27	0.50	0.148
Toluene	0.70	1.08	0.50	0.378
Ethylbenzene	1.66	0.59	0.50	0.486
Xylenes (total)	5.68	0.97	0.50	2.756
Tetrachloroethene	1.60	1.34	0.50	1.068
Trichloroethane	0.13	2.46	0.50	0.160
Trichloroethene	0.11	1.55	0.50	0.085
Benzyl alcohol	0.88	10.10	0.50	4.443

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION	COC	Bioconcentration	Proportion	COC
Point Lay	Concentration	Factor	of vegetation	Concentration
SPECIES	Soil/Sediment		in diet	in food
caribou	(CS)	(Bv)	(%V)	(CF)
COC	mg/kg	unitless	%	mg/kg
Inorganics				
Iron	0.00	0.004	0.01	0.000
Lead	60.60	0.045	0.01	0.027
Manganese	0.00	0.250	0.01	0.000
Selenium	17.00	0.025	0.01	0.004
Zinc	96.00	1.500	0.01	1.440
Organics				
DRPH	2886.00	0.03	0.01	0.966
Benzene	0.13	2.27	0.01	0.003
Toluene	0.70	1.08	0.01	0.008
Ethylbenzene	1.66	0.59	0.01	0.010
Xylenes (total)	5.68	0.97	0.01	0.055
Tetrachloroethene	1.60	1.34	0.01	0.021
Trichloroethane	0.13	2.46	0.01	0.003
Trichloroethene	0.11	1.55	0.01	0.002
Benzyl alcohol	0.88	10.10	0.01	0.089

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION	COC	Bioconcentration	Proportion	COC
Point Lay	Concentration	Factor	of vegetation	Concentration
SPECIES	Soil/Sediment		in diet	in food
Lapland longspur	(CS)	(Bv)	(%V)	(CF)
COC	mg/kg	unitless	%	mg/kg
Inorganics				
Iron	0.00	0.004	0.20	0.000
Lead	60.60	0.045	0.20	0.545
Manganese	0.00	0.250	0.20	0.000
Selenium	17.00	0.025	0.20	0.085
Zinc	96.00	1.500	0.20	28.800
Organics				
DRPH	2886.00	0.03	0.20	19.316
Benzene	0.13	2.27	0.20	0.059
Toluene	0.70	1.08	0.20	0.151
Ethylbenzene	1.66	0.59	0.20	0.194
Xylenes (total)	5.68	0.97	0.20	1.102
Tetrachloroethene	1.60	1.34	0.20	0.427
Trichloroethane	0.13	2.46	0.20	0.064
Trichloroethene	0.11	1.55	0.20	0.034
Benzyl alcohol	0.88	10.10	0.20	1.777

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION Point Lay	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
SPECIES glaucous gull				
COC				
Inorganics				
Iron	0.00	0.004	0.01	0.000
Lead	60.60	0.045	0.01	0.027
Manganese	0.00	0.250	0.01	0.000
Selenium	17.00	0.025	0.01	0.004
Zinc	96.00	1.500	0.01	1.440
Organics				
DRPH	2886.00	0.03	0.01	0.966
Benzene	0.13	2.27	0.01	0.003
Toluene	0.70	1.08	0.01	0.008
Ethylbenzene	1.66	0.59	0.01	0.010
Xylenes (total)	5.68	0.97	0.01	0.055
Tetrachloroethene	1.60	1.34	0.01	0.021
Trichloroethane	0.13	2.46	0.01	0.003
Trichloroethene	0.11	1.55	0.01	0.002
Benzyl alcohol	0.88	10.10	0.01	0.089

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION	COC	Bioconcentration	Proportion	COC
Point Lay	Concentration	Factor	of vegetation	Concentration
SPECIES	Soil/Sediment		in diet	in food
brant	(CS)	(Bv)	(%V)	(CF)
COC	mg/kg	unitless	%	mg/kg
Inorganics				
Iron	0.00	0.004	0.03	0.000
Lead	60.60	0.045	0.03	0.082
Manganese	0.00	0.250	0.03	0.000
Selenium	17.00	0.025	0.03	0.013
Zinc	96.00	1.500	0.03	4.320
Organics				
DRPH	2886.00	0.03	0.03	2.897
Benzene	0.13	2.27	0.03	0.009
Toluene	0.70	1.08	0.03	0.023
Ethylbenzene	1.66	0.59	0.03	0.029
Xylenes (total)	5.68	0.97	0.03	0.165
Tetrachloroethene	1.60	1.34	0.03	0.064
Trichloroethane	0.13	2.46	0.03	0.010
Trichloroethene	0.11	1.55	0.03	0.005
Benzyl alcohol	0.88	10.10	0.03	0.267

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION Point Lay	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
SPECIES pectoral sandpiper				
COC				
Inorganics				
Iron	0.00	0.004	1.00	0.000
Lead	60.60	0.045	1.00	2.727
Manganese	0.00	0.250	1.00	0.000
Selenium	17.00	0.025	1.00	0.425
Zinc	96.00	1.500	1.00	144.000
Organics				
DRPH	2886.00	0.03	1.00	96.582
Benzene	0.13	2.27	1.00	0.296
Toluene	0.70	1.08	1.00	0.756
Ethylbenzene	1.66	0.59	1.00	0.971
Xylenes (total)	5.68	0.97	1.00	5.512
Tetrachloroethene	1.60	1.34	1.00	2.137
Trichloroethane	0.13	2.46	1.00	0.320
Trichloroethene	0.11	1.55	1.00	0.170
Benzyl alcohol	0.88	10.10	1.00	8.886

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION	COC	Bioconcentration	Proportion	COC
Point Lay	Concentration	Factor	of vegetation	Concentration
SPECIES	Soil/Sediment		in diet	in food
spectacled eider	(CS)	(Bv)	(%V)	(CF)
COC	mg/kg	unitless	%	mg/kg
Inorganics				
Iron	0.00	0.004	0.25	0.000
Lead	60.60	0.045	0.25	0.682
Manganese	0.00	0.250	0.25	0.000
Selenium	17.00	0.025	0.25	0.106
Zinc	96.00	1.500	0.25	36.000
Organics				
DRPH	2886.00	0.03	0.25	24.146
Benzene	0.13	2.27	0.25	0.074
Toluene	0.70	1.08	0.25	0.189
Ethylbenzene	1.66	0.59	0.25	0.243
Xylenes (total)	5.68	0.97	0.25	1.378
Tetrachloroethene	1.60	1.34	0.25	0.534
Trichloroethane	0.13	2.46	0.25	0.080
Trichloroethene	0.11	1.55	0.25	0.043
Benzyl alcohol	0.88	10.10	0.25	2.221

APPENDIX E
ESTIMATED EXPOSURE EQUATIONS

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

$$\text{Estimated Exposure} = \{[(CF*FI) + (CS*SI*ROA) + (CW*WI)] * 0.001\} * IS / BW$$

INSTALLATION		COC Conc. Food Items (CF)* (mg/kg)	Food Intake Rate (FI) (g/day)	(A)	% of FI (mg/kg)	COC Conc. Soil/Sed. (CS) (mg/kg)	Soil/Sed. Intake % (SI%) (CF*FI)	Relative Availability (ROA) (SI) (g/day)	Oral (ROA) (SI) (unitless)	(B)	COC Conc. Water (CW) (ug/L)	Water Intake Rate (WI) (L/day)	(C)	(D)	Conver. Units 0.001 (D)*.001 (unitless)	Percent Ingested at Site (IS) (kg)	Body Weight (BW) (kg)	ESTIMATED EXPOSURE (D*IS/BW=EE)
Point Lay	SPECIES																	
arctic fox																		
COC																		
Inorganics																		
Iron			256	0	0.028	0.00		7.168	1	0.00	14800.00	0.42	6216	6216.00	6.22	0.01	4.95	0.013
Lead			256	0	0.028	60.60		7.168	1	434.38	0.00	0.42	0	434.38	0.43	0.01	4.95	0.001
Manganese			256	0	0.028	0.00		7.168	1	0.00	1087.00	0.42	456.54	456.54	0.46	0.01	4.95	0.001
Selenium			256	0	0.028	17.00		7.168	1	121.86	0.00	0.42	0	121.86	0.12	0.01	4.95	0.000
Zinc			256	0	0.028	96.00		7.168	1	688.13	0.00	0.42	0	688.13	0.69	0.01	4.95	0.001
Organics																		
DRPH			256	0	0.028	2886.00		7.168	1	20686.85	461.00	0.42	193.62	20880.47	20.88	0.01	4.95	0.042
Benzene			256	0	0.028	0.13		7.168	1	0.93	0.00	0.42	0	0.93	0.00	0.01	4.95	0.000
Toluene			256	0	0.028	0.70		7.168	1	5.02	0.00	0.42	0	5.02	0.01	0.01	4.95	0.000
Ethylbenzene			256	0	0.028	1.66		7.168	1	11.90	0.00	0.42	0	11.90	0.01	0.01	4.95	0.000
Xylenes (total)			256	0	0.028	5.68		7.168	1	40.71	0.00	0.42	0	40.71	0.04	0.01	4.95	0.000
Tetrachloroethene			256	0	0.028	1.60		7.168	1	11.47	0.00	0.42	0	11.47	0.01	0.01	4.95	0.000
Trichloroethane			256	0	0.028	0.13		7.168	1	0.93	0.00	0.42	0	0.93	0.00	0.01	4.95	0.000
Trichloroethene			256	0	0.028	0.11		7.168	1	0.79	0.00	0.42	0	0.79	0.00	0.01	4.95	0.000
Benzyl alcohol			256	0	0.028	0.88		7.168	1	6.31	0.00	0.42	0	6.31	0.01	0.01	4.95	0.000

* data are not available for the carnivorous diet of the arctic fox

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\frac{[(CF*FI) + (CS*SI*ROA) + (CW*WI)] * .001 * IS}{BW}$														
INSTALLATION Point Lay	COC Conc.	Food Intake Rate (FI)	Soil/Sed. Intake % (SI%)	COC Conc. (CS)	Soil/Sed. Ingestion Rate (SI)	Relative Oral Availability (ROA)	(B)	COC Water Conc. (CW)	Water Intake Rate (WI)	(C)	(D)	Conver. Units 0.001	Percent Ingested at Site (IS)	ESTIMATED EXPOSURE (D*IS/BW=EE)
SPECIES brown lemming	Food Items (CF)	(g/day)	(mg/kg)	(mg/kg)	(g/day)	(unitless)	(unitless)	(ug/L)	(L/day)	(mg/kg-bw/day)	(kg)	(unitless)	(kg)	(mg/kg-bw/day)
COC														
Inorganics														
Iron	0.00	45	0.027	0.00	1.22	1	0.00	14800.00	0.007	103.6	103.60	0.10	0.5	0.055
Lead	1.36	45	0.027	60.60	1.22	1	73.63	0.00	0.007	0	134.99	0.13	0.5	0.055
Manganese	0.00	45	0.027	0.00	1.22	1	0.00	1087.00	0.007	7.609	7.61	0.01	0.5	0.055
Selenium	0.21	45	0.027	17.00	1.22	1	20.66	0.00	0.007	0	30.22	0.03	0.5	0.055
Zinc	72.00	45	0.027	96.00	1.22	1	116.64	0.00	0.007	0	3356.64	3.36	0.5	0.055
Organics														
DRPH	48.29	45	0.027	2886.00	1.22	1	3506.49	461.00	0.007	3.227	5682.81	5.68	0.5	0.055
Benzene	0.15	45	0.027	0.13	1.22	1	0.16	0.00	0.007	0	6.81	0.01	0.5	0.055
Toluene	0.38	45	0.027	0.70	1.22	1	0.85	0.00	0.007	0	17.85	0.02	0.5	0.055
Ethylbenzene	0.49	45	0.027	1.66	1.22	1	2.02	0.00	0.007	0	23.87	0.02	0.5	0.055
Xylenes (total)	2.76	45	0.027	5.68	1.22	1	6.90	0.00	0.007	0	130.92	0.13	0.5	0.055
Tetrachloroethene	1.07	45	0.027	1.60	1.22	1	1.94	0.00	0.007	0	50.02	0.05	0.5	0.055
Trichloroethane	0.16	45	0.027	0.13	1.22	1	0.16	0.00	0.007	0	7.3635	0.01	0.5	0.055
Trichloroethene	0.09	45	0.027	0.11	1.22	1	0.13	0.00	0.007	0	3.9603	0.00	0.5	0.055
Benzyl alcohol	4.44	45	0.027	0.88	1.22	1	1.07	0.00	0.007	0	201.0025	0.20	0.5	0.055
														1.827

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\frac{[(CF*FI) + (CS*SI*ROA) + (CW*WI)] * .001}{IS} / BW$														
INSTALLATION	Point Lay	COC	Food	Soil/Sed.	COC	Soil/Sed.	Relative	COC	Water	Conver.	Percent	Body	ESTIMATED	
SPECIES	caribou	Conc.	Intake	Intake	Conc.	Intake	Availability	Water	Rate	Units	at Site	Weight	EXPOSURE	
		(CF)	(FI)	(CF*FI)	(SI%)	(SI)	(ROA)	(CS*SI*ROA)	(WI)	(A+B+C)	(IS)	(BW)	(D*IS/BW=EE)	
		(mg/kg)	(g/day)	(A)	% of FI	(mg/kg)	(unitless)	(B)	(L/day)	(D)	(unitless)	(kg)	(mg/kg-bw/day)	
Inorganics														
Iron		0.00	2400	0.00	0.02	0.00	1	0.00	88800.00	88800.00	88.80	95.5	0.009	
Lead		0.03	2400	65.45	0.02	60.60	1	2908.80	0.00	2974.25	2.97	95.5	0.000	
Manganese		0.00	2400	0.00	0.02	0.00	1	0.00	6522.00	6522.00	6.52	95.5	0.001	
Selenium		0.00	2400	10.20	0.02	17.00	1	816.00	0.00	826.20	0.83	95.5	0.000	
Zinc		1.44	2400	3456.00	0.02	96.00	1	4608.00	0.00	8064.00	8.06	95.5	0.001	
Organics														
DRPH		0.97	2400	2317.97	0.02	2886.00	1	138528.00	461.00	143611.97	143.61	95.5	0.015	
Benzene		0.00	2400	7.10	0.02	0.13	1	6.24	0.00	13.34	0.01	95.5	0.000	
Toluene		0.01	2400	18.13	0.02	0.70	1	33.60	0.00	51.73	0.05	95.5	0.000	
Ethylbenzene		0.01	2400	23.31	0.02	1.66	1	79.68	0.00	102.99	0.10	95.5	0.000	
Xylenes (total)		0.06	2400	132.28	0.02	5.68	1	272.64	0.00	404.92	0.40	95.5	0.000	
Tetrachloroethene		0.02	2400	51.29	0.02	1.60	1	76.80	0.00	128.09	0.13	95.5	0.000	
Trichloroethane		0.00	2400	7.69	0.02	0.13	1	6.24	0.00	13.9259	0.01	95.5	0.000	
Trichloroethene		0.00	2400	4.08	0.02	0.11	1	5.28	0.00	9.3617	0.01	95.5	0.000	
Benzyl alcohol		0.09	2400	213.26	0.02	0.88	1	42.24	0.00	255.5022	0.26	95.5	0.000	

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\frac{((CF*FI) + (CS*SI*ROA) + (CW*WI)) * .001 * IS}{BW}$														
INSTALLATION Point Lay	COC Conc.	Food Intake Rate (FI)	Soil/Sed. Intake % (SI%)	COC Conc. (CS)	Soil/Sed. Rate (SI)	Relative Oral Availability (ROA)	COC Conc. (CW)	Water Intake Rate (WI)	(CW*WI) (A+B+C)	Conver. Units 0.001	Percent Ingested at Site (IS)	Body Weight (BW)	ESTIMATED EXPOSURE (D*IS/BW=EE)	
SPECIES Lapland longspur	Food Items (CF)	(g/day)	(g/day)	(mg/kg)	(g/day)	(unitless)	(ug/L)	(L/day)	(C)	(D)	(D)*.001	(kg)	(mg/kg-bw/day)	
COC	(mg/kg)	(A)	% of FI	(mg/kg)	(g/day)	(unitless)	(B)	(L/day)	(C)	(D)	(D)*.001	(kg)	(mg/kg-bw/day)	
Inorganics														
Iron	0.00	6.6	0.00	0.00	0.13	1	0.00	0.005	74.00	74.00	0.07	0.2	0.027	0.548
Lead	0.55	6.6	3.60	60.60	0.13	1	8.00	0.005	0	11.60	0.01	0.2	0.027	0.086
Manganese	0.00	6.6	0.00	0.00	0.13	1	0.00	0.005	5.44	5.44	0.01	0.2	0.027	0.040
Selenium	0.09	6.6	0.56	17.00	0.13	1	2.24	0.005	0	2.81	0.00	0.2	0.027	0.021
Zinc	28.80	6.6	190.08	96.00	0.13	1	12.67	0.005	0	202.75	0.20	0.2	0.027	1.502
Organics														
DRPH	19.32	6.6	127.49	2886.00	0.13	1	380.95	0.005	2.31	510.75	0.51	0.2	0.027	3.783
Benzene	0.06	6.6	0.39	0.13	0.13	1	0.02	0.005	0	0.41	0.00	0.2	0.027	0.003
Toluene	0.15	6.6	1.00	0.70	0.13	1	0.09	0.005	0	1.09	0.00	0.2	0.027	0.008
Ethylbenzene	0.19	6.6	1.28	1.66	0.13	1	0.22	0.005	0	1.50	0.00	0.2	0.027	0.011
Xylenes (total)	1.10	6.6	7.28	5.68	0.13	1	0.75	0.005	0	8.03	0.01	0.2	0.027	0.059
Tetrachloroethene	0.43	6.6	2.82	1.60	0.13	1	0.21	0.005	0	3.03	0.00	0.2	0.027	0.022
Trichloroethane	0.06	6.6	0.42	0.13	0.13	1	0.02	0.005	0	0.4399	0.00	0.2	0.027	0.003
Trichloroethene	0.03	6.6	0.22	0.11	0.13	1	0.01	0.005	0	0.2390	0.00	0.2	0.027	0.002
Benzyl alcohol	1.78	6.6	11.73	0.88	0.13	1	0.12	0.005	0	11.8456	0.01	0.2	0.027	0.086

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\frac{((CF*FI) + (CS*SI*ROA) + (CW*WI)) * .001 * IS}{BW}$																
INSTALLATION Point Lay	COC Conc.	Food Intake	Soil/Sed. Intake	COC Conc.	Soil/Sed. Ingestion	Relative Oral	COC Conc.	Water Rate	Conver. Units	Percent Ingested at Site	Body Weight	ESTIMATED EXPOSURE				
	Food Items (CF)	Rate (FI)	% (SI%)	Soil / Sed. (CS)	Rate (SI)	Availability (ROA)	(CW*SI*ROA) (CW)	(WI)	(A+B+C)	(IS)	(BW)	(D*IS/BW*EE)				
COC	(mg/kg)	(g/day)	(A)	% of FI	(mg/kg)	(g/day)	(unitless)	(B)	(ug/L)	(L/day)	(C)	(D)	(D)* .001	(unitless)	(kg)	(mg/kg-bw/day)
Inorganics																
Iron	0.00	73.9	0.00	0.076	0.00	5.62	1	0.00	14800.00	0.08	1184	1184.00	1.18	0.01	1.45	0.008
Lead	0.03	73.9	2.02	0.076	60.60	5.62	1	340.35	0.00	0.08	0	342.37	0.34	0.01	1.45	0.002
Manganese	0.00	73.9	0.00	0.076	0.00	5.62	1	0.00	1087.00	0.08	86.96	86.96	0.09	0.01	1.45	0.001
Selenium	0.00	73.9	0.31	0.076	17.00	5.62	1	95.48	0.00	0.08	0	95.79	0.10	0.01	1.45	0.001
Zinc	1.44	73.9	106.42	0.076	96.00	5.62	1	539.17	0.00	0.08	0	645.59	0.65	0.01	1.45	0.004
Organics																
DRPH	0.97	73.9	71.37	0.076	2886.00	5.62	1	16208.93	461.00	0.08	36.88	16317.18	16.32	0.01	1.45	0.113
Benzene	0.00	73.9	0.22	0.076	0.13	5.62	1	0.73	0.00	0.08	0	0.95	0.00	0.01	1.45	0.000
Toluene	0.01	73.9	0.56	0.076	0.70	5.62	1	3.93	0.00	0.08	0	4.49	0.00	0.01	1.45	0.000
Ethylbenzene	0.01	73.9	0.72	0.076	1.66	5.62	1	9.32	0.00	0.08	0	10.04	0.01	0.01	1.45	0.000
Xylenes (total)	0.06	73.9	4.07	0.076	5.68	5.62	1	31.90	0.00	0.08	0	35.97	0.04	0.01	1.45	0.000
Tetrachloroethene	0.02	73.9	1.58	0.076	1.60	5.62	1	8.99	0.00	0.08	0	10.57	0.01	0.01	1.45	0.000
Trichloroethane	0.00	73.9	0.24	0.076	0.13	5.62	1	0.73	0.00	0.08	0	0.9688	0.00	0.01	1.45	0.000
Trichloroethene	0.00	73.9	0.13	0.076	0.11	5.62	1	0.62	0.00	0.08	0	0.7435	0.00	0.01	1.45	0.000
Benzyl alcohol	0.09	73.9	6.57	0.076	0.88	5.62	1	4.94	0.00	0.08	0	11.5091	0.01	0.01	1.45	0.000

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\frac{((CF*FI) + (CS*SI*ROA) + (CW*WI)) * .001 * IS}{BW}$																	
INSTALLATION		COC	Food	Soil/Sed.	COC	Soil/Sed.	Relative	COC	Water	Percent	Body	ESTIMATED					
Point Lay	Conc.	Intake	Rate	Intake	Conc.	Ingestion	Oral	Conc.	Intake	Conver.	Weight	EXPOSURE					
SPECIES	Food Items	(FI)	(CF*FI)	%	Soil /Sed.	Rate	Availability	(CW)	Rate	Units	(BW)	(D*IS/BW=EE)					
brant	(CF)			(SI%)	(CS)	(SI)	(ROA)	(CW*SI*ROA)	(WI)	(A+B+C)	(IS)						
COC	(mg/kg)	(g/day)	(A)	% of FI	(mg/kg)	(g/day)	(unitless)	(B)	(L/day)	(D)	(unitless)	(mg/kg-bw/day)					
Inorganics	Iron	0.00	69.2	0.00	0.082	0.00	5.67	1	0.00	14800.00	0.07	1036	1036.00	1.04	0.03	1.31	0.024
	Lead	0.08	69.2	5.66	0.082	60.60	5.67	1	343.87	0.00	0.07	0	349.53	0.35	0.03	1.31	0.008
	Manganese	0.00	69.2	0.00	0.082	0.00	5.67	1	0.00	1087.00	0.07	76.09	76.09	0.08	0.03	1.31	0.002
	Selenium	0.01	69.2	0.88	0.082	17.00	5.67	1	96.46	0.00	0.07	0	97.35	0.10	0.03	1.31	0.002
	Zinc	4.32	69.2	298.94	0.082	96.00	5.67	1	544.74	0.00	0.07	0	843.69	0.84	0.03	1.31	0.019
Organics	DRPH	2.90	69.2	200.50	0.082	2886.00	5.67	1	16376.32	461.00	0.07	32.27	16609.09	16.61	0.03	1.31	0.380
	Benzene	0.01	69.2	0.61	0.082	0.13	5.67	1	0.74	0.00	0.07	0	1.35	0.00	0.03	1.31	0.000
	Toluene	0.02	69.2	1.57	0.082	0.70	5.67	1	3.97	0.00	0.07	0	5.54	0.01	0.03	1.31	0.000
	Ethylbenzene	0.03	69.2	2.02	0.082	1.66	5.67	1	9.42	0.00	0.07	0	11.44	0.01	0.03	1.31	0.000
	Xylenes (total)	0.17	69.2	11.44	0.082	5.68	5.67	1	32.23	0.00	0.07	0	43.67	0.04	0.03	1.31	0.001
	Tetrachloroethene	0.06	69.2	4.44	0.082	1.60	5.67	1	9.08	0.00	0.07	0	13.52	0.01	0.03	1.31	0.000
	Trichloroethane	0.01	69.2	0.66	0.082	0.13	5.67	1	0.74	0.00	0.07	0	1.4025	0.00	0.03	1.31	0.000
	Trichloroethene	0.01	69.2	0.35	0.082	0.11	5.67	1	0.62	0.00	0.07	0	0.9773	0.00	0.03	1.31	0.000
	Benzyl alcohol	0.27	69.2	18.45	0.082	0.88	5.67	1	4.99	0.00	0.07	0	23.4407	0.02	0.03	1.31	0.001

$$\text{Estimated Exposure} = \{ \pi(CF*FI) + (CS*SI*ROA) + (CW^{**}WI) \} * .001 \}^{*} IS) / BW$$
EST_EXP3.XLS PECT SANDP 8/24/95

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\{[(CF*FI) + (CS*SI*ROA) + (CW*WI)] * 0.001\} * IS\} / BW$														
INSTALLATION	COC	Food	Soil/Sed.	COC	Soil/Sed.	Relative	COC	Water	Percent	Body	ESTIMATED			
Point Lay	Conc.	Intake	Intake	Conc.	Intake	Oral	Conc.	Intake	Ingested	Weight	EXPOSURE			
SPECIES	Food Items	Rate	%	Soil / Sed.	Rate	Availability	Water	Rate	at Site	(BW)	(D*IS/BW=EE)			
spectacled eider	(CF)	(FI)	(CF*FI)	(CS)	(SI)	(ROA)	(CS*SI*ROA)	(WI)	(IS)	(B)				
COC	(mg/kg)	(g/day)	(A)	(mg/kg)	(g/day)	(unitless)	(B)	(L/day)	(D)	(kg)	(mg/kg-bw/day)			
Inorganics														
Iron	0.00	71.6	0.00	0.082	0.00	5.87	1	0.00	1036	1.04	0.25			
Lead	0.68	71.6	48.81	0.082	60.60	5.87	1	355.79	0	404.61	0.25			
Manganese	0.00	71.6	0.00	0.082	0.00	5.87	1	0.00	76.09	0.08	0.25			
Selenium	0.11	71.6	7.61	0.082	17.00	5.87	1	99.81	0	107.42	0.25			
Zinc	36.00	71.6	2577.60	0.082	96.00	5.87	1	563.64	0	3141.24	0.25			
Organics														
DRPH	24.15	71.6	1728.82	0.082	2886.00	5.87	1	16944.28	32.27	18705.37	0.25			
Benzene	0.07	71.6	5.29	0.082	0.13	5.87	1	0.76	0	6.06	0.25			
Toluene	0.19	71.6	13.52	0.082	0.70	5.87	1	4.11	0	17.63	0.25			
Ethylbenzene	0.24	71.6	17.39	0.082	1.66	5.87	1	9.75	0	27.13	0.25			
Xylenes (total)	1.38	71.6	98.66	0.082	5.68	5.87	1	33.35	0	132.01	0.25			
Tetrachloroethene	0.53	71.6	38.25	0.082	1.60	5.87	1	9.39	0	47.64	0.25			
Trichloroethane	0.08	71.6	5.73	0.082	0.13	5.87	1	0.76	0	6.50	0.25			
Trichloroethene	0.04	71.6	3.04	0.082	0.11	5.87	1	0.65	0	3.69	0.25			
Benzyl alcohol	2.22	71.6	159.06	0.082	0.88	5.87	1	5.17	0	164.22	0.25			

APPENDIX F

SCALING FACTOR CALCULATIONS

SCALING FACTOR CALCULATIONS

Scaling factor (SF) = (representative species average body weight/ test species average body weight)^{1/3}
 based on the mass to surface area ratios of the test species and the representative species
 (Mantel and Schneiderman 1975)

Representative Species	Average Body Weight (kg)	Test Species	Average Body Weight (kg)	Scaling Factor (SF)
brown lemming	0.055	mouse	0.025	1.30
	0.055	rat	0.25	0.60
arctic fox	4.95	mouse	0.025	5.82
	4.95	rat	0.25	2.70
caribou	95.5	sheep	60	1.17
	95.5	cattle	500	0.58
	95.5	rat	0.25	7.24
	95.5	mouse	0.025	15.59
Lapland longspur	0.027	chicken	0.8	0.32
	0.027	mallard	1.08	0.29
	0.027	Japanese quail	0.10	0.65
	0.027	ringed dove	0.155	0.56
	0.027	ring-necked pheasant	1.14	0.29
brant	1.305	chicken	0.8	1.18
	1.305	mallard	1.08	1.07
	1.305	Japanese quail	0.10	2.35
	1.305	ringed dove	0.155	2.03
	1.305	ring-necked pheasant	1.14	1.05
glaucous gull	1.445	chicken	0.8	1.22
	1.445	mallard	1.08	1.10
	1.445	Japanese quail	0.10	2.43
	1.445	ringed dove	0.155	2.10
	1.445	ring-necked pheasant	1.14	1.08
pectoral sandpiper	0.079	chicken	0.8	0.46
	0.079	mallard	1.08	0.42
	0.079	Japanese quail	0.10	0.92
	0.079	ringed dove	0.155	0.80
	0.079	ring-necked pheasant	1.14	0.41
spectacled eider	1.375	chicken	0.8	1.20
	1.375	mallard	1.08	1.08
	1.375	Japanese quail	0.10	2.39
	1.375	ringed dove	0.155	2.07
	1.375	ring-necked pheasant	1.14	1.06

APPENDIX G
RI ANALYTICAL DATA

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①

TABLE G-1. SUMMARY OF SAMPLING AND ANALYSES CONDUCTED

ANALYSES	HVOC ^a	BTEX ^b	VOC 8260	SVOC	Metals	TPH-Diesel ^b Range 3510/3550	TPH
ANALYTICAL METHOD	SW8010M	SW8020	SW8260	SW8270	SW3050 (Soil) 3005 (Water)/6010	Diesel 8100 M	Ga
POINT LAY (LIZ-2)							
Background (BKGD)	5 Soil 2 Water	5 Soil 2 Water	5 Soil 2 Water	5 Soil 2 Water	5 Soil 2 Water (Total) 2 Water (Dissolved)	5 Soil 2 Water	
Deactivated Landfill (LF01)	10 Soil 7 Water	10 Soil 7 Water	4 Soil 3 Water	1 Soil 1 Water	1 Soil 1 Water (Total) 1 Water (Dissolved)	13 Soil 9 Water	
Garage (SS06)	13 Soil 2 Water	19 Soil 3 Water	4 Soil 1 Water	4 Soil 1 Water	2 Soil 1 Water (Total) 1 Water (Dissolved)	21 Soil 3 Water	
Drainage Pathways from POL Tanks (SS07)	3 Water	4 Soil 3 Water	4 Soil 4 Water	1 Water	NA	7 Soil 6 Water	
Crushed Drum Area (SS08)	9 Soil 2 Water	12 Soil 2 Water	4 Soil 1 Water	3 Soil 1 Water	2 Soil 1 Water (Total) 1 Water (Dissolved)	14 Soil 2 Water	
Total Field Analyses	37 Soil 16 Water	50 Soil 17 Water	21 Soil 11 Water	13 Soil 6 Water	10 Soil 5 Water (Total) 5 Water (Dissolved)	60 Soil 22 Water	
QA/QC SAMPLES							
Trip Blanks	2 Water	2 Water	3 Water	NA	NA	NA	
Equipment Blanks	2 Water	2 Water	3 Water	2 Water	2 Water (Total) 1 Water (Dissolved)	2 Water	
Ambient Condition Blanks	NA	NA	1 Water	NA	NA	NA	
Field Replicates	4 Soil	5 Soil	2 Soil	1 Soil	1 Soil	6 Soil	
Field Duplicates	1 Water	2 Water	1 Water	1 Water	1 Water (Total) 1 Water (Dissolved)	2 Water	
Total Site Analyses	41 Soil 21 Water	55 Soil 23 Water	23 Soil 19 Water	14 Soil 9 Water	11 Soil 8 Water (Total) 7 Water (Dissolved)	66 Soil 26 Water	

NA Not analyzed.

* These analyses were completed on a quick turnaround basis.

a The number of soil sample includes sediment samples collected from surface water features.

b These analyses were completed on a 24-hour turnaround at a temporary fixed laboratory at Barrow, Alaska.

c Investigation derived wastes from Point Lay were combined with the investigation derived wastes from Point Barrow.

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ANALYSES CONDUCTED FOR POINT LAY REMEDIAL INVESTIGATIONS^a

	TPH-Diesel ^b Range 3510/3550	TPH - Gasoline ^b Range	TPH Residual Range*	PCB*	Pesticides*	TDS	TSS	TOC	TOTAL SAMPL
0	Diesel 8100 M	Gas 5030/8015M	Diesel 8100M	SW8080/8080M	SW8080/8080M	E160.1	E160.2	SW9060	
d)	5 Soil 2 Water	5 Soil 2 Water	5 Soil 2 Water	5 Soil 2 Water	5 Soil 2 Water	2 Water	2 Water	3 Soil 2 Water	5 Soil 2 Water
d)	13 Soil 9 Water	13 Soil 9 Water	10 Soil 7 Water	10 Soil 7 Water	1 Soil 1 Water	1 Water	1 Water	1 Soil 1 Water	13 Soil 9 Water
d)	21 Soil 3 Water	21 Soil 3 Water	13 Soil 3 Water	13 Soil	2 Soil	NA	NA	NA	21 Soil 3 Water
	7 Soil 6 Water	7 Soil 6 Water	4 Soil 3 Water	NA	1 Soil	1 Water	1 Water	1 Water	7 Soil 6 Water
d)	14 Soil 2 Water	14 Soil 2 Water	9 Soil 2 Water	9 Soil 2 Water	1 Soil	1 Water	1 Water	1 Water	14 Soil 2 Water
d)	60 Soil 22 Water	60 Soil 22 Water	41 Soil 17 Water	37 Soil 11 Water	10 Soil 3 Water	5 Water	5 Water	4 Soil 5 Water	60 Soil 22 Water
d)	NA	2 Water	NA	NA	NA	NA	NA	NA	3 Water
d)	2 Water	2 Water	2 Water	2 Water	2 Water	NA	NA	NA	3 Water
	NA	NA	NA	NA	NA	NA	NA	NA	1 Water
	6 Soil	6 Soil	4 Soil	4 Soil	2 Soil	NA	NA	1 Soil	6 Soil
d)	2 Water	2 Water	2 Water	1 Water	1 Water	1 Water	1 Water	1 Water	2 Water
d)	66 Soil 26 Water	66 Soil 28 Water	45 Soil 21 Water	41 Soil 14 Water	12 Soil 6 Water	6 Water	6 Water	5 Soil 6 Water	66 Soil 31 Water

er features.
 oratory at Barrow, Alaska.
 derived wastes from Point Barrow, Point Lonely, and Wainwright. These were collectively sampled during the Point Barrow investigation

3

OS	TSS	TOC	TOTAL SAMPLES
0.1	E160.2	SW9060	
ater	2 Water	3 Soil 2 Water	5 Soil 2 Water
ater	1 Water	1 Soil 1 Water	13 Soil 9 Water
A	NA	NA	21 Soil 3 Water
ater	1 Water	1 Water	7 Soil 6 Water
ater	1 Water	1 Water	14 Soil 2 Water
ater	5 Water	4 Soil 5 Water	60 Soil 22 Water
A	NA	NA	3 Water
A	NA	NA	3 Water
A	NA	NA	1 Water
A	NA	1 Soil	6 Soil
ater	1 Water	1 Water	2 Water
ater	6 Water	5 Soil 6 Water	66 Soil 31 Water

mpled during the Point Barrow investigation.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY

Installation: Point Lay Site: Background (BKGD)				Matrix: Soil/Sediment Units: mg/kg										Field Blanks			Lab Blanks	
Parameters	Detect Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples													
					S01	S02	S03	S04	SD01	AB01	EB01	TB01						
Laboratory Sample ID Numbers					433 4327-6	435 4327-7	437 4327-8	439 4327-9	431 4327-5	4356-5	443/448 4328-2	441 4328-1	#384-82493 #5-82493 #5-82593 #182-82493 #384-82793 4328 4327					
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L	mg/kg		
DRPH	5-10	50-100	500 ^a	<50 ^b <100 ^b	<80 ^b	<100 ^b	<100 ^b	<70 ^b	<50 ^b	NA	<1,000 ^b	NA	<1,000	<50	<1,000	<50		
GRPH	0.3-0.4	3-4	100	<3 ^b <4 ^b	<3 ^b	<4 ^b	<4 ^b	<3 ^b	<3 ^b	NA	<50 ^b	<50 ^b	<100J	<2J	<100J	<2J		
RRPH (Approx.)	10	100	2,000 ^a	<100	<100	<100	<100	<100	<100	NA	<2,000	NA	<2,000	<100	<2,000	<100		
BTEX (8020/ 8020 Mod.)			10 Total BTEX	<0.13 <0.20	<0.13	<0.20	<0.20	<0.18	<0.13									
Benzene	0.003-0.004	0.03-0.04	0.5	<0.02 <0.04	<0.02	<0.04	<0.04	<0.03	<0.03	<1 ^c	<1	<1	<1	<0.02	<1	<0.02		
Toluene	0.003-0.004	0.03-0.04		<0.02 <0.04	<0.02	<0.04	<0.04	<0.03	<0.03	<1 ^c	<1	<1	<1	<0.02	<1	<0.02		
Ethylbenzene	0.003-0.004	0.03-0.04		<0.03 <0.04	<0.03	<0.04	<0.04	<0.04	<0.03	<1 ^c	<1	<1	<1	<0.02	<1	<0.02		
Xylenes (Total)	0.004-0.008	0.04-0.08		<0.04 <0.08	<0.04	<0.08	<0.08	<0.08	<0.04	<2 ^c	<2	<2	<2	<0.04	<2	<0.04		
HVOC 8010	0.003-0.004	0.03-0.04		<0.03 <0.04	<0.03	<0.04	<0.04	<0.03	<0.03	NA	<1	<1	<1	<0.02J	<1	<0.02J		
VOC 8260	0.020	0.030-0.150		<0.03 <0.150	<0.150	<0.070	<0.040	<0.030	<0.050	<1-3.1	<1	<1-12	<1	<0.020	<1	<0.020		
SVOC 8270	0.200	6.9-15.0		<6.90 <15	<10	<15.0	<12.0	<6.90	<13.0	NA	<36	NA	<10	<0.200	<10	<0.200		
Pesticides	0.002-0.05	0.02-0.5		<0.02J <0.5J	<0.02J <0.5J	<0.02J <0.5J	<0.02J <0.5J	<0.02J <0.5J	<0.02J <0.5J	NA	<0.2J <50J	NA	NA	<0.01	NA	<0.01		
PCBs	0.01	0.1	10	<0.1	<0.1	<0.2	<0.2	<0.1	<0.1	NA	<2	NA	<10	<0.5	<10	<0.5		
TOC				57,000-69,300	69,300	NA	NA	57,000	68,100	NA	NA	NA	<5,000	NA	<5,000	NA		

CT&E Data.

F&B Data.

Not analyzed.

Result is an estimate.

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

BTEX determined by 8260 method analysis.

☐ CT&E Data.
☒ F&B Data.
☐ Not analyzed.

Result is an estimate.

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

BTEX determined by 8260 method analysis.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Background (BKGD)		Matrix: Soil/Sediment Units: mg/kg		METALS ANALYSES										Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Point Lay Bkgd. Range	Environmental Samples					Field Blank				
						S01	S02	S03	S04	SD01			EB01		
Laboratory Sample ID Numbers						4327-6	4327-7	4327-8	4327-9	4327-5			4328-2	4328	4327
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/L	μg/L	μg/L
Aluminum	0.35	2		1,500-25,000	9,700-23,000	23,000	9,700	17,000	19,000	19,000	19,000	19,000	<100	<100	<100
Antimony	N/A	66-115		<7.8-<230	<66-<115	<115J	<114	<79	<66	<85J			<100	<100	<100
Arsenic	0.11	66-115		<4.9-8.5	<66-<115	<115	<114	<79	<66	<85			<100	<100	<100
Barium	0.024	1		27-390	170-390	390	170	260	290	340			<50	<50	<50
Beryllium	N/A	1-5.7		<2.6-6.4	<4.0-4.2	<5.7	<5.7	<4.0	4.2	<4.3			<50	<50	<50
Cadmium	0.33	3.3-5.7		<3.0-<36	<3.3-<5.7	<5.7	<5.7	<4.0	<3.3	<4.3			<50	<50	<50
Calcium	0.69	4		360-59,000	1,800-3,000	2,500	2,200	3,000	1,800	2,000			<200	<200	<200
Chromium	0.066	1		<4.3-47	16-37	37	16	28	31	33			<50	<50	<50
Cobalt	N/A	1-11		<5.1-12	<7.9-7.3	<11	<11	<7.9	7.3	<8.6			<100	<100	<100
Copper	0.045	1		<2.7-45	15-25	20	15	19	19	25			<50	<50	<50
Iron	0.50	2		5,400-35,000	13,900-35,000	35,000	13,900	21,000	30,100	33,000			<100	<100	<100
Lead	0.13	2-11		<5.1-22	<11-20	20	<11	12	14	17			<100	<100	<100
Magnesium	0.96	4		360-7,400	2,500-4,400	4,300	2,500	4,400	4,000	3,800			<200	<200	<200
Manganese	0.025	1		25-290	73-110	97J	73	92	110	100J			<50	<50	<50
Molybdenum	N/A	3.3-5.7		<2.5-<11	<3.3-<5.7	<5.7	<5.7	<4.0	<3.3	<4.3			<50	<50	<50
Nickel	0.11	1		4.2-46	19-27	26	19	26	25	27			<50	<50	<50
Potassium	23	100		<300-2,200	660-1,500	1,500	660	1,300	1,100	1,200			<5,000	<5,000	<5,000

☐ CT&E Data.
☐ N/A Not available.
☐ J Result is an estimate.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Background (BKGD)		Matrix: Soil/Sediment Units: mg/kg		METALS ANALYSES										Field Blank		Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Point Lay Bkgd. Range	Environmental Samples					SD01			EB01		
Laboratory Sample ID Numbers						S01	S02	S03	S04		SD01	SD01	SD01	EB01		
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	4327-6	4327-7	4327-8	4327-9		4327-5	4327-5	4327-5	4328-2		4328 4327
Selenium	1.2	66-115		<7.8-<170	<66-<115	mg/kg	mg/kg	mg/kg	mg/kg		mg/kg	mg/kg	mg/kg	µg/L		µg/L
Silver	0.53	33-57		<3-<110	<33-<57	<115	<114	<79	<66		<85	<85	<85	<100		<100
Sodium	0.55	5		<160-680	110-174	<57R	<57	<40	<33		<43R	<43R	<43R	<50J		<50
Thallium	0.011	0.31-0.57		<0.2-<1.2	<0.31-<0.57	170	120	140	110		174	174	174	<250		<250
Vanadium	0.036	1		6.3-59	28-56	<0.57	<0.55	<0.36	<0.31		<0.40	<0.40	<0.40	<5		<5
Zinc	0.16	1		9.2-95	26-48	56	28	44	52		54	54	54	<50		<50
						46	26	48	44		48	48	48	<50		<50

☐ J R

CT&E Data.
Result is an estimate.
Result has been rejected.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Background (BKGD)		Matrix: Surface Water Units: µg/L		Environmental Samples				Field Blanks			Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	SW01	SW02		AB01	EB01	TB01		
Laboratory Sample ID Numbers					448/457 4328-5 4329-1	461/462 4328-8 4329-4		4356-5	443/446 4328-2	441 4328-1	#5-82593 #3&4-82493 4356 4328 4329	
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L		µg/L	µg/L	µg/L	µg/L	
DRPH	100	1,000		<1,000 ^b	<1,000 ^b	<1,000 ^b		NA	<1,000 ^b	NA	<1,000	
GRPH	5	50		<50J ^b	<50J ^b	<50J ^b		NA	<50J ^b	<50J ^b	<100J	
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000		NA	<2,000	NA	<2,000	
BTEX (8020/8020 Mod.)												
Benzene	0.1	1	5	<1	<1	<1		<1 ^c	<1	<1	<1	
Toluene	0.1	1	1,000	<1	<1	<1		<1 ^c	<1	<1	<1	
Ethylbenzene	0.1	1	700	<1	<1	<1		<1 ^c	<1	<1	<1	
Xylenes (Total)	0.2	2	10,000	<2	<2	<2		<2 ^c	<2	<2	<2	
HVOC 8010	0.1	1		<1	<1	<1		NA	<1	<1	<1	
VOC 8260	1	1		<1	<1	<1		<1-3.1	<1	<1-12	<1	
SVOC 8270	10	20-31		<20-<31	<31	<20		NA	<36	NA	<10	
Pesticides	0.2-5	2-50		<0.2J-<50J	<0.2J-<50J	<0.2J-<50J		NA	<0.2J-<50J	NA	NA	
PCBs	0.2	2	0.5	<2	<2	<2		NA	<2	NA	<10	

☐ CT&E Data.
☒ F&B Data.
☒ Not analyzed.
☒ Result is an estimate.
 DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
 BTEX determined by 8260 method analysis.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Background (BKGD)		Matrix: Surface Water Units: µg/L									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples			Field Blanks			Lab Blanks
					SW01	SW02		AB01	EB01	TB01	
Laboratory Sample ID Numbers					448/457 4328-5 4329-1	461/462 4328-8 4329-4		4356-5	443/446 4328-2	441 4328-1	4356 4328 4329
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L		µg/L	µg/L	µg/L	µg/L
TOC	5,000	5,000		31,700-40,000	40,000	31,700		NA	NA	NA	<5,000
TSS	100	200		6,000-77,000	6,000	77,000		NA	NA	NA	<100
TDS	10,000	10,000		149,000-151,000	149,000	151,000		NA	NA	NA	<10,000

☐ NA
CT&E Data.
Not analyzed.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Background (BKGD)		Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)				Field Blank		Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Point Lay Bkgd. Range	Environmental Samples			Field Blank	
						SW01	SW02		EB01	
Laboratory Sample ID Numbers						4329-1 4328-5	4329-4 4328-8			4329 4328
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
Aluminum	17.4	100		<100-350 (<100-340)	130-350 (<100-340)	350 (340)	130 (<100)		<100	<100
Antimony	N/A	100	6	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)		<100	<100
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)		<100	<100
Barium	1.2	50	2,000	<50-93 (<50-91)	51-56 (<50)	51 (<50)	56 (<50)		<50	<50
Beryllium	N/A	50	4	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)		<50	<50
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)		<50	<50
Calcium	34.5	200		4,500-88,000 (4,100-86,000)	5,500-9,000 (5,300-8,600)	5,500 (5,300)	9,000 (8,600)		<200	<200
Chromium	3.29	50	100	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)		<50	<50
Cobalt	N/A	100		<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)		<100	<100
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)		<50	<50
Iron	25	100		180-2,800 (<100-1,600)	2,000-2,800 (950-1,600)	2,000 (1,600)	2,800 (950)		<100	<100

☐ CT&E Data.
☐ N/A Not available.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Background (BKGD)			Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)							
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Point Lay Bkgd. Range	Environmental Samples				Field Blank		Lab Blanks
						SW01	SW02				EB01	
Laboratory Sample ID Numbers						4329-1 4328-5	4329-4 4328-8				4328-2	4329 4328
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L				µg/L	µg/L
Lead	6.6	100	15	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)				<100	<100
Magnesium	47.8	200		<5,000-53,000 (2,600-54,000)	<5,000-5,500 (<4,900-5,500)	5,000 (4,900)	5,500 (5,500)				<200	<200
Manganese	1.24	50		<50-510 (<50-120)	120-510 (66-120)	120 (120)	510 (66)				<50	<50
Molybdenum	N/A	50		<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)				<50	<50
Nickel	5.5	50	100	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)				<50	<50
Potassium	1,154	5,000		<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)				<5,000	<5,000
Selenium	62.4	100	50	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)				<100	<100
Silver	2.6	50	50	<50 (<50)	<50 (<50)	<50J (<50)J	<50 (<50)				<50J	<50
Sodium	27.7	250		8,400-410,000 (8,200-450,000)	17,000-18,000 (17,000-19,000)	17,000 (17,000)	18,000 (19,000)				<250	<250
Thallium	0.57	5	2	<5 (<5)	<5 (<5)	<5 (<5)	<5 (<5)				<5	<5

☐ CT&E Data.
☐ N/A
☐ J Not available.
Result is an estimate.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Background (BKGD)				Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)					
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Point Lay Bkgd. Range	Environmental Samples			Field Blank		Lab Blanks
						SW01	SW02			EB01	
Laboratory Sample ID Numbers						4329-1 4328-5	4329-4 4328-8			4328-2	4329 4328
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L			µg/L	µg/L
Vanadium	1.8	50		<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)			<50	<50
Zinc	8.2	50		<50-160 (<50)	<50 (<50)	<50 (<50)	160 (<50)			<50	<50

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY

Installation: Point Lay Site: Deactivated Landfill (LF01)				Matrix: Sediment Units: mg/kg		Environmental Samples										Field Blanks			Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	SD01	SD02	SD03	SD04 & SD08 (Replicates)	SD05	SD06	AB01	EB02	TB02	Lab Blanks						
Laboratory Sample ID Numbers					478	480	482	484 4354-6	486	488	4356-5	557/572 4356-2	569 4356-1	#5-82593 #1&2-82593 4354 4356						
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	mg/kg					
DRPH	5-19	50-190	500 ^a	<50 ^b <100 ^b	<160 ^b	<190 ^b	<50 ^b	<60 ^b	<60 ^b	<80 ^b	NA	<1,000 ^b	NA	<1,000	<50					
GRPH	0.1-3.2	1-32	100	<3.2 ^b <4.4 ^b	<32 ^b	<4.4 ^b	<1.0 ^b	<1.0 ^b	<1.0 ^b	<2.0 ^b	NA	<100 ^b	<100 ^b	<100J	<1J					
RRPH (Approx.)	10-40	100-400	2,000 ^a	<100	<300	<400	<100	<120	<120	<200	NA	<2,000	NA	<2,000	<100					
BTEX (8020/ 8020 Mod.)			10 Total BTEX	<0.13 <0.20	<3.17	<0.61	<0.10	<0.15	<0.15	<0.20										
Benzene	0.002- 0.007	0.02-0.07	0.5	<0.02 <0.04	<0.07	<0.07	<0.02	<0.03	<0.03	<0.04	<1 ^c	<1	<1	<1	<0.02					
Toluene	0.002- 0.006	0.02-0.6		<0.02 <0.04	<0.6	<0.07	<0.02	<0.03	<0.03	<0.04	<1 ^c	<1	<1	<1	<0.02					
Ethyl- benzene	0.002-0.12	0.02-1.2		<0.03 <0.04	<1.2	<0.07	<0.02	<0.03	<0.03	<0.04	<1 ^c	<1	<1	<1	<0.02					
Xylenes (Total)	0.004-0.13	0.04-1.3		<0.04 <0.06	<1.3	<0.4	<0.04	<0.06	<0.06	<0.08	<2 ^c	<2	<2	<2	<0.04					
HVOC 8010																				
Tetrachloro- ethene	0.002- 0.007	0.02-0.07		<0.03J <0.04J	<0.07J	<0.07J	<0.02J	<0.03J	<0.03J	<0.04J	NA	<1	<1	<1	<0.02J					
VOC 8260																				
Tetrachloro- ethene	0.020	0.025-0.050		<0.030 <0.150	NA	NA	NA	0.306J	0.622	NA	<1	<1	<1	<1	<0.020					

CT&E Data.

F&B Data.

NA

Not analyzed.

Result is an estimate.

Result has been rejected.

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

BTEX determined by 8260 method analysis.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay		Matrix: Sediment													
Site: Deactivated Landfill (LF01)		Units: mg/kg													
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bgkd. Levels	Environmental Samples						Field Blanks			Lab Blanks	
					SD01	SD02	SD03	SD04 & SD08 (Replicates)	SD05	SD06	AB01	EB02	TB02		
Laboratory Sample ID Numbers					478	480	482	484 4354-6	486	488	4356-5	557/572 4356-2	569 4356-1	#5-82793 4356	#5-82593 4354 4356
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
1,2,4-Trimethylbenzene	0.020	0.025-0.050		<0.030-<0.150	NA	NA	NA	0.055J	NA	NA	<1	<1	<1	<1	<0.020
SVOC 8270					NA	NA	NA	1.38	NA	NA	NA	<25	NA	<10	<1.00
Benzyl alcohol	0.200	0.400		<6.9-<15.0	NA	NA	NA	<0.02-<0.5	NA	NA	NA	<0.2J-<10J	NA	NA	<0.2J-<0.5J
Pesticides	0.002-0.05	0.02-0.5		<0.02J-<0.5J	NA	NA	NA	<0.02-<0.5	NA	NA	NA	<2	NA	<2J	<0.1
PCBs	0.01-0.04	0.1-0.4	10	<0.1	<0.3	<0.4	<0.1	<0.1	<0.1	<0.2	NA	NA	NA	NA	NA
TOC				57,000-69,300	NA	NA	NA	15,800	NA	NA	NA	NA	NA	NA	NA

☐ CT&E Data.
☒ F&B Data.
☐ Not analyzed.
 Result is an estimate.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay		Matrix: Sediment											
Site: Deactivated Landfill (LF01)		Units: mg/kg											
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			Lab Blanks	
					SD07	SD09	SD10	SD11	AB01	EB02	TB02		
Laboratory Sample ID Numbers					490	494	496	498	4356-5	557/572 4356-2	569 4356-1	#5-82793 #3&4-82593 4356	#5-82593 #1&2-82593
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	mg/kg
DRPH	6-40	60-400	500 ^a	<50 ^b <100 ^b	<60 ^b	<100 ^b	<60 ^b	<400 ^b	NA	<1,000 ^b	NA	<1,000	<50
GRPH	0.1-0.8	1-8	100	<3 ^b <4 ^b	<1 ^b	<2 ^b	<1 ^b	<8 ^b	NA	<100 ^b	<100 ^b	<50	<1J
RRPH (Approx.)	12-80	120-800	2,000 ^a	<100	<120	<200	<120	<800	NA	<2,000	NA	<2,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.13- <0.20	<0.15	<0.25	<0.15	<1.0					
Benzene	0.003-0.02	0.03-0.2	0.5	<0.02- <0.04	<0.03	<0.05	<0.03	<0.2	<1 ^c	<1	<1	<1	<0.02
Toluene	0.003-0.02	0.03-0.2		<0.02- <0.04	<0.03	<0.05	<0.03	<0.2	<1 ^c	<1	<1	<1	<0.02
Ethylbenzene	0.003-0.02	0.03-0.2		<0.03- <0.04	<0.03	<0.05	<0.03	<0.2	<1 ^c	<1	<1	<1	<0.02
Xylenes (Total)	0.006-0.04	0.06-0.4		<0.04- <0.08	<0.06	<0.1	<0.06	<0.4	<2 ^c	<2	<2	<2	<0.04
HVOC 8010	0.003-0.02	0.03-0.2		<0.03- <0.04	<0.03J	<0.05J	<0.03J	<0.2J	NA	<1	<1	<1	<0.02J
PCBs	0.01-0.08	0.1-0.8	10	<0.1	<0.1	<0.2	<0.1	<0.8	NA	<2	NA	<2J	<0.1

☐ CT&E Data.
☒ F&B Data.
☒ Not analyzed.
☒ Result is an estimate.
☒ The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
☒ DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
☒ BTEX determined by 8260 method analysis.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)			Matrix: Sediment Units: mg/kg										
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks			Lab Blanks		
					2SD12	2SD13	2SD14	AB01	2EB03	2TB03			
Laboratory Sample ID Numbers					4692-11	4692-12	4692-15	4356-5	4692-17	4692-16	4356 4692	4356 4692	
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	mg/kg	
DRPH	4.00	4.00	500 ^a	<50 ^b <100 ^b	<4.00	624 ^c	39.9 ^d	NA	NA	NA	NA	<4.00	
GRPH	0.400	0.400	100	<3 ^b <4 ^b	1.73	<2.75	11.9	NA	NA	NA	NA	<0.400	
VOC 8260													
Ethylbenzene	0.020	0.020-0.240		<0.03-<0.150	0.054	<0.130	<0.240	<1	<1	<1	<1	<0.020	
p-Isopropyltoluene	0.020	0.020-0.240		<0.03-<0.150	0.067	<0.130	<0.240	<1	<1	<1	<1	<0.020	
Naphthalene	0.020	0.020-0.240		<0.03-<0.150	0.129	<0.130	0.409	<1	<1	<1	<1	<0.020	
Tetrachloroethene	0.020	0.020-0.240		<0.03-<0.150	0.727	<0.130	1.92	<1	<1	<1	<1	<0.020	
Toluene	0.020	0.020-0.240		<0.03-<0.150	0.042	<0.130	<0.240	<1	<1	<1	<1	<0.020	
1,2,4-Trimethylbenzene	0.020	0.020-0.240		<0.03-<0.150	0.213	<0.130	0.552	<1	<1	<1	<1	<0.020	
1,3,5-Trimethylbenzene	0.020	0.020-0.240		<0.03-<0.150	0.128	<0.130	0.342	<1	<1	<1	<1	<0.020	
Xylenes (Total)	0.040	0.040-0.480		<0.06-<0.300	0.312	<0.260	0.724	<2	<2	<2	<2	<0.040	

□ GT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

a The action level for DRPH is based on conversations with ADEC, a final action level has not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c The laboratory reported that the EPH pattern in this sample was not consistent with an unweathered middle distillate fuel.

d The laboratory reported that the EPH pattern in this sample was not consistent with a middle distillate fuel.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)			Matrix: Sediment Units: mg/kg		METALS ANALYSES								
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples					Field Blank		Lab Blanks	
					SD04	SD08							EB02
Laboratory Sample ID Numbers					4354-6	4356-13					4356-2	4354 4356	
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg					µg/L	µg/L	
Aluminum	0.35	2		1,500-25,000	2,400	2,200					<100	<100	
Antimony	N/A	56-82		<7.8-<230	<82J	<56					<100	<100	
Arsenic	0.11	56-82		<4.9-8.5	<82	<56					<100	<100	
Barium	0.024	1		27-390	260	190					<50	<50	
Beryllium	N/A	28-41		<2.6-6.4	<41	<28					<50	<50	
Cadmium	0.33	28-41		<3.0-<36	<41	<28					<50	<50	
Calcium	0.69	4		360-59,000	6,300	2,600					410	<200	
Chromium	0.066	1		<4.3-47	13	5.1					<50	<50	
Cobalt	N/A	5.6-8.2		<5.1-12	<8.2	<5.6					<100	<100	
Copper	0.045	1		<2.7-45	55	16					<50	<50	
Iron	0.50	2		5,400-35,000	70,000	19,000					<100	<100	
Lead	0.13	2-5.6		<5.1-22	18	<5.6					<100	<100	
Magnesium	0.96	4		360-7,400	1,900	1,500					<200	<200	
Manganese	0.025	1		25-290	220J	180					<50	<50	
Molybdenum	N/A	2.8-41		<2.5-<11	<41	<2.8					<50	<50	
Nickel	0.11	1		4.2-46	13	13					<50	<50	
Potassium	23	100-410		<300-2,200	<410	280					<5,000	<5,000	

☐ CT&E Data.
☐ N/A Not available.
☐ J Result is an estimate.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)			Matrix: Sediment Units: mg/kg		METALS ANALYSES							
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples				Field Blank			Lab Blanks
					SD04	SD08				EB02		
Laboratory Sample ID Numbers					4354-6	4356-13				4356-2		4354 4356
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg				µg/L		µg/L
Selenium	1.2	56-82		<7.8-<170	<82	<56				<100		<100
Silver	0.53	2.8-41		<3-<110	<41R	<2.8				<50		<50
Sodium	0.55	5		<160-680	120	75				370		<250
Thallium	0.011	0.29-0.42		<0.2-<1.2	<0.42	<0.29				<5		<5
Vanadium	0.036	1		6.3-59	11	11				<50		<50
Zinc	0.16	1		9.2-95	380	125				<50		<50

☐ R CT&E Data.
Result has been rejected.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)				Matrix: Surface Water Units: µg/L											Lab Blanks	
Parameters	Detect Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			TB02			
					SW01	SW02	SW03	SW04 & SW08 (Duplicates)		SW05	AB01	EB02				
Laboratory Sample ID Numbers					559/592	560/597	561/598	562/601 4356-1 4356-10	566/613 4356-11 4356-4	563/604	4356-5	557/572 4356-2	569 4356-1	#5-82783 #3&4-82593 4356 4358		
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L		
DRPH	100	1,000		<1,000 ^b	<1,000 ^b	<1,000 ^b	<1,000 ^b	<1,000 ^b	<1,000 ^b	<1,000 ^b	NA	<1,000 ^b	NA	<1,000		
GRPH	10	100		<50 ^b	<100 ^b	<100 ^b	<100 ^b	<100 ^b	<100 ^b	<100 ^b	NA	<100 ^b	<100 ^b	<100J		
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	<2,000	<2,000	<2,000	<2,000	NA	<2,000	NA	<2,000		
BTEX (8020/8020 Mod.)																
Benzene	0.1	1	5	<1	<1	<1	<1	23	20	<1	<1 ^c	<1	<1	<1		
Toluene	0.1	1	1,000	<1	<1	<1	<1	9	7	<1	<1 ^c	<1	<1	<1		
Ethylbenzene	0.1	1	700	<1	<1	<1	<1	10J	12J	<1	<1 ^c	<1	<1	<1		
Xylenes (Total)	0.2	2	10,000	<2	<2	<2	<2	20J	20J	<2	<2 ^c	<2	<2	<2		
HVOC 8010																
Tetrachloroethene	0.1	1	5	<1	<1	<1	<1	93	80	<1	NA	<1	<1	<1		
VOC 8280																
Benzene	1	1	5	<1	NA	NA	NA	18	18	NA	<1	<1	<1	<1		

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)				Matrix: Surface Water Units: µg/L													
Parameters	Detect Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples						Field Blanks			Lab Blanks			
					SW01	SW02	SW03	SW04 & SW08 (Duplicates)		SW05	AB01	EB02	TB02				
Laboratory Sample ID Numbers					559/592	560/597	561/598	562/601 4358-1 4356-10	568/613 4358-11 4356-4	563/604	4356-5	557/572 4356-2	569 4356-1	#5-82793 4356 4358			
ANALYSES	µg/L	µg/L	µg/L	µg/L													
Dichlorodifluoro- methane	1	1		<1	NA	NA	NA	33	34	NA	<1	<1	<1	<1			
cis-1,2- Dichloroethene	1	1	70	<1	NA	NA	NA	6.5	6.9	NA	<1	<1	<1	<1			
Ethylbenzene	1	1	700	<1	NA	NA	NA	4.0	4.1	NA	<1	<1	<1	<1			
p-Isopropyltoluene	1	1		<1	NA	NA	NA	1.7	1.7	NA	<1	<1	<1	<1			
Naphthalene	1	1		<1	NA	NA	NA	<1J	3.4J	NA	<1	<1	<1	<1			
Tetrachloroethene	1	1	5	<1	NA	NA	NA	84	82	NA	<1	<1	<1	<1			
Toluene	1	1	1,000	<1	NA	NA	NA	7.3	7.2	NA	<1	<1	<1	<1			
Trichloroethene	1	1	5	<1	NA	NA	NA	3.3	3.3	NA	<1	<1	<1	<1			
Trichloro- fluoromethane	1	1		<1	NA	NA	NA	3.0	2.9	NA	<1	<1	<1	<1			
1,2,4- Trimethylbenzene	1	1		<1	NA	NA	NA	<1	10	NA	<1	<1	<1	<1			
1,3,5- Trimethylbenzene	1	1		<1	NA	NA	NA	<1	6.7	NA	<1	<1	<1	<1			
Xylenes (Total)	2	2	10,000	<2	NA	NA	NA	21.7	21.7	NA	<2	<2	<2	<2			
SVOC 8270	10	11-17		<20-<31	NA	NA	NA	<17	<11	NA	NA	<25	NA	<10			
Pesticides	0.02-1	0.2-10		<0.2J-<50J	NA	NA	NA	<0.2J-<10J	<0.2J-<10J	NA	NA	<0.2J-<10J	NA	NA			
PCBs	0.2	2	0.5	<2	<2	<2	<2	<2	<2	<2	NA	<2	NA	<2J			
TOC	5,000	5,000		31,700-40,000	NA	NA	NA	17,800	20,400	NA	NA	NA	NA	<5,000			

☐ CT&E Data.
☒ F&B Data.
☒ Not analyzed.
☒ Result is an estimate.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)		Matrix: Surface Water Units: µg/L												
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples						Field Blanks			Lab Blanks
					SW01	SW02	SW03	SW04 & SW08 (Duplicates)		SW05	AB01	EB02	TB02	
Laboratory Sample ID Numbers					559/592	560/597	561/598	562/601 4356-1 4356-10	566/613 4356-11 4356-4	563/604	4356-5	557/572 4356-2	569 4356-1	4356 4358
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
TSS	100	200		6,000-77,000	NA	NA	NA	96,000	84,000	NA	NA	NA	NA	<200
TDS	10,000	10,000		149,000-151,000	NA	NA	NA	808,000	870,000J	NA	NA	NA	NA	<10,000

☐ CT&E Data.
☐ NA
☐ J
 Not analyzed.
 Result is an estimate.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)		Matrix: Surface Water Units: µg/L									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks			Lab Blanks
					SW06	SW07		AB01	EB02	TB02	
Laboratory Sample ID Numbers					564/609	565/612		4356-5	557/572 4356-2	569 4356-1	#5-82793 #3&4-82593 4356
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L		µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000 ^b	<1,000 ^b	<1,000 ^b		NA	<1,000 ^b	NA	<1,000
GRPH	10	100		<50 ^b	<100 ^b	<100 ^b		NA	<100 ^b	<100 ^b	<100J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000		NA	<2,000	NA	<2,000
BTX (8020/8020 Mod.)											
Benzene	0.1	1	5	<1	<1	<1		<1 ^c	<1	<1	<1
Toluene	0.1	1	1,000	<1	<1	<1		<1 ^c	<1	<1	<1
Ethylbenzene	0.1	1	700	<1	<1	<1		<1 ^c	<1	<1	<1
Xylenes (Total)	0.2	2	10,000	<2	<2	<2		<2 ^c	<2	<2	<2
HVOC 8010	0.1	1		<1	<1	<1		NA	<1	<1	<1
PCBs	0.2	2	0.5	<2	<2	<2		NA	<2	NA	<2J

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTX determined by 8260 method analysis.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)				Matrix: Surface Water Units: µg/L								
Parameters	Detect. Limits	Quant. Limits	Action Levels	Btgd. Levels	Environmental Samples			Field Blanks			Lab Blanks	
					2SW09	2SW10				AB01	2EB03	2TB03
Laboratory Sample ID Numbers					4692-9	4692-10			4356-5	4692-17	4692-16	4356 4692
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L			µg/L	µg/L	µg/L	µg/L
DRPH	100	100		<1,000 ^a	181 ^{ad}	240 ^{ad}			NA	NA	NA	<100
GRPH	20	20		<50 ^{a,b}	223 ^a	<20			NA	NA	NA	<20
VOC 8260												
Benzene	1	1	5	<1	13	1.1			<1	<1	<1	<1
Chloromethane	1	1		<1	3.5B	8.3B			<1	3.1U	3.1U	1.09
cis-1,2-Dichloroethene	1	1	70	<1	5.5B	<1			<1	4.6	1.5	<1
Dichlorodifluoromethane	1	1		<1	58	<1			<1	<1	<1	<1
Ethylbenzene	1	1	700	<1	3.6	<1			<1	<1	<1	<1
p-Isopropyltoluene	1	1		<1	1.3	<1			<1	<1	<1	<1
Methylene Chloride	1	1	5	<1	2.6B	<1			3.1	3.4	2.8	<1
Tetrachloroethene	1	1	5	<1	109	<1			<1	<1	<1	<1
Toluene	1	1	1,000	<1	8.9	<1			<1	<1	<1	<1
Trichloroethene	1	1	5	<1	2.9B	<1			<1	1.7	<1	<1
Trichlorofluoromethane	1	1		<1	3.8	<1			<1	<1	<1	<1

CT&E Data.

F&B Data.

NA Not analyzed.

B The analyte was detected in the associated blank.

J Result is an estimate.

U Compound is not present above the concentration listed.

a Total petroleum hydrocarbons in these water samples exceed the 15 µg/L stated for fresh water in ADEC's Water Quality Criteria 18AAC70 (ADEC 1989).

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

d The laboratory reported that the EPH pattern in this sample was not consistent with a middle distillate fuel.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)		Matrix: Surface Water Units: µg/L										
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			Lab Blanks
					2SW09	2SW10			AB01	2EB03	2TB03	
Laboratory Sample ID Numbers					4692-9	4692-10			4356-5	4692-17	4692-16	4356 4692
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L			µg/L	µg/L	µg/L	µg/L
1,2,4-Trimethylbenzene	1	1		<1	9.3	<1			<1	<1	<1	<1
1,3,5-Trimethylbenzene	1	1		<1	5.1	<1			<1	<1	<1	<1
Xylene	2	2		<2	2.07	<2			<2	<2	<2	<2

☐ CT&E Data.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)				Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples						Field Blank			Lab Blanks	
					SW04 & SW08 (Duplicates)										EB02
Laboratory Sample ID Numbers					4358-1 4356-10	4358-4	µg/L						4356-2		4358 4356
ANALYSES	µg/L	µg/L	µg/L	µg/L									µg/L		µg/L
Aluminum	17.4	100		<100-350 (<100-340)	180 (<100)	<100 (<100)							<100 (<100)		<100
Antimony	N/A	100	6	<100 (<100)	<100 (<100)	<100 (<100)							<100 (<100)		<100
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)	<100 (<100)							<100 (<100)		<100
Barium	1.2	50	2,000	<50-93 (<50-91)	210 (320)	200 (210)							<50 (<50)		<50
Beryllium	N/A	50	4	<50 (<50)	<50 (<50)	<50 (<50)							<50 (<50)		<50
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)	<50 (<50)							<50 (<50)		<50
Calcium	34.5	200		4,500-88,000 (4,100-86,000)	81,000 (81,000)	83,000 (83,000)							410 (<200)		<200
Chromium	3.29	50	100	<50 (<50)	<50 (<50)	<50 (<50)							<50 (<50)		<50
Cobalt	N/A	100		<100 (<100)	<100 (<100)	<100 (<100)							<100 (<100)		<100
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)	<50 (<50)							<50 (<50)		<50
Iron	25	100		180-2,800 (<100-1,600)	32,000 (22,000)	33,000 (23,000)							<100 (<100)		<100

☐ CT&E Data.
N/A Not available.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)		Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)					Field Blank		Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples						
					SW04 & SW08 (Duplicates)					EB02	
Laboratory Sample ID Numbers					4358-1 4356-10	4358-4				4356-2	4358 4356
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L				µg/L	µg/L
Lead	6.6	100	15	<100 (<100)	<100 (<100)	<100 (<100)				<100 (<100)	<100
Magnesium	47.8	200		<5,000-53,000 (2,600-54,000)	25,000 (25,000)	25,000 (25,000)				<200 (<200)	<200
Manganese	1.24	50		<50-510 (<50-210)	940 (930)	960 (960)				<50 (<50)	<50
Molybdenum	N/A	50		<50 (<50)	<50 (<50)	<50 (<50)				<50 (<50)	<50
Nickel	5.5	50	100	<50 (<50)	<50 (<50)	<50 (<50)				<50 (<50)	<50
Potassium	1,154	5,000		<5,000 (<5,000)	8,000 (8,200)	8,100 (8,300)				<5,000 (<5,000)	<5,000
Selenium	62.4	100	50	<100 (<100)	<100 (<100)	<100 (<100)				<100 (<100)	<100
Silver	2.6	50	50	<50 (<50)	<50J (<50)J	<50J (<50)J				<50 (<50)	<50
Sodium	27.7	250		8,400-410,000 (8,200-450,000)	47,000 (49,000)	47,000 (47,000)				370 (400)	<250
Thallium	0.57	5	2	<5 (<5)	<5 (<5)	<5 (<5)				<5 (<5)	<5
Vanadium	1.8	50		<50 (<50)	<50 (<50)	<50 (<50)				<50 (<50)	<50

☐ CT&E Data.
☐ N/A Not available
☐ J Result is an estimate.

TABLE G-3. DEACTIVATED LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Deactivated Landfill (LF01)		Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)							Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples				Field Blank			
					SW04 & SW08 (Duplicates)					EB02		
Laboratory Sample ID Numbers					4358-1 4356-10	4358-4				4356-2	4358 4356	
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L				µg/L	µg/L	
Zinc	8.2	50		<50-160 (<50)	<50 (60)	<50 (<50)				<50 (<50)	<50 (<50)	<50

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY

Installation: Point Lay Site: Garage (SS06)		Matrix: Soil Units: mg/kg		Environmental Samples										Field Blanks		Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	S01 & S09 (Replicates)	S02-1	S03	S04	S05-1.5	S06-2.5	AB01	EB02	TB02				
Laboratory Sample ID Numbers					636	652	640 4354-4	642	644	648	4356-5	557/572 4356-2	569 4356-1	#5-82593 #384-82593 4356	#5-82593 #182-82593 4354		
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/L	mg/L	mg/L	mg/L		
DRPH	6	60	500 ^a	<50 ^b - <100 ^b	23,000 ^b	18,000 ^b	<60 ^b	12,800 ^b	80 ^b	12,000 ^b	NA	<1,000 ^b	NA	<1,000	<50		
GRPH	3.4-3.7	34-37	100	<3 ^b - <4 ^b	25 ^b	44 ^b	<37 ^b	397 ^b	<34 ^b	316 ^b	NA	<100 ^b	<100 ^b	<100 ^b	<100 ^b		
RRPH (Approx.)	18-18	160-180	2,000 ^a	<100	40,000	40,000	<180	1,900	<160	<160	NA	<2,000	NA	<2,000	<100		
BTEX (8020/ 8020 Mod.)			10 Total BTEX	<0.13 - <0.20	<0.10	11.2J	<0.15	60	<4.0J	16J							
Benzene	0.002-0.05	0.02-0.5	0.5	<0.02 - <0.04	<0.02	<0.03	<0.03	<0.03	<0.5J	<0.4J	<1 ^c	<1 ^c	<1 ^c	<1	<0.02		
Toluene	0.002-0.05	0.02-0.5		<0.02 - <0.04	<0.02	0.2	<0.03	4	<0.5	<0.4	<1 ^c	<1 ^c	<1 ^c	<1	<0.02		
Ethyl- benzene	0.002-0.2	0.02-2.0		<0.03 - <0.04	<0.02	3	<0.03	14	<2.0J	<0.4J	<1 ^c	<1 ^c	<1 ^c	<1	<0.02		
Xylenes (Total)	0.004-0.1	0.04-1.0		<0.04 - <0.08	<0.04	8J	<0.06	42J	<1J	16J	<2 ^c	<2 ^c	<2 ^c	<2	<0.04		
HVOC 8010																	
Tetrachlo- roethene	0.002-0.004	0.02-0.04		<0.03 - <0.04	1.3J	0.1J	1J	<0.03J	<0.03J	<0.04J	NA	<1 ^c	<1 ^c	<1	<0.02J		
Trichloro- ethane	0.002-0.004	0.02-0.04		<0.03 - <0.04	1.8J	<0.03J	<0.03J	0.5J	<0.03J	<0.04J	NA	<1 ^c	<1 ^c	<1	<0.02J		
Trichloro- ethene	0.002-0.004	0.02-0.04		<0.03 - <0.04	1J	<0.03J	<0.03J	<0.03J	<0.03J	<0.04J	NA	<1 ^c	<1 ^c	<1	<0.02J		

☐ CT&E Data.
☒ F&B Data.
☒ Not analyzed.

☐ NA
☐ J
☐ R
☐ a
☐ b
☐ c

Result is an estimate.

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

BTEX determined by 8260 method analysis.

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)				Matrix: Soil Units: mg/kg		Environmental Samples										Field Blanks			Lab Blanks	
Parameters	Detect Limits	Quant. Limits	Action Levels	Bkgd. Levels	S01 & S09 (Replicates)		S02-1	S03	S04	S05-1.5	S06-2.5	AB01	EB02	TB02	Lab Blanks					
Laboratory Sample ID Numbers					636	652	638	640 4354-4	642	644	646	4356-5	557/572 4356-2	569 4356-1	4356	4354				
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	mg/kg				
VOC 8260																				
cis-1,2- Dichloro- ethene	0.020	0.020		<0.030-<0.150	NA	NA	NA	0.022J	NA	NA	NA	<1	<1	<1	<1	<0.020				
p-isopro- pytoluene	0.020	0.020		<0.030-<0.150	NA	NA	NA	0.042J	NA	NA	NA	<1	<1	<1	<1	<0.020				
Ethyl- benzene	0.020	0.020		<0.030-<0.150	NA	NA	NA	0.023J	NA	NA	NA	<1	<1	<1	<1	<0.020				
Methylene Chloride	0.020	0.020	90	<0.030-<0.150	NA	NA	NA	0.023BJ	NA	NA	NA	3.1	2.3	12	<1	<0.020				
Naph- thalene	0.020	0.020		<0.030-<0.150	NA	NA	NA	0.158J	NA	NA	NA	<1	<1	<1	<1	<0.020				
Tetrachloro- ethene	0.020	0.020		<0.030-<0.150	NA	NA	NA	0.359J	NA	NA	NA	<1	<1	<1	<1	<0.20				
Toluene	0.020	0.020		<0.030-<0.150	NA	NA	NA	0.094J	NA	NA	NA	<1	<1	<1	<1	<0.020				
1,2,4-Tri- methyl- benzene	0.020	0.020		<0.030-<0.150	NA	NA	NA	0.315J	NA	NA	NA	<1	<1	<1	<1	<0.020				
1,3,5-Tri- methyl- benzene	0.020	0.020		<0.030-<0.150	NA	NA	NA	0.497J	NA	NA	NA	<1	<1	<1	<1	<0.020				
Xylenes (Total)	0.040	0.040		<0.060-<0.300	NA	NA	NA	0.849J	NA	NA	NA	<2	<2	<2	<2	<0.040				
SVOC 8270	0.200	2.20		<6.9-<15.0	NA	NA	NA	<2.20	NA	NA	NA	NA	<25	NA	<10	<0.200				

☐ CT&E Data.
☐ Not analyzed.
 NA The analyte was detected in the associated blank.
 B Result is an estimate.
☐ J

AK-RISK\LAY\APP-G\4109661203\G-4.TBL

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04 MARCH 1996

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)		Matrix: Soil Units: mg/kg		Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks			Lab Blanks		
Parameters	Detect Limits	Quant Limits	S07 & S10 (Replicates)			S08	S11	AB01	EB02	TB02				
Laboratory Sample ID Numbers						648	654	650 4354-5	656	4356-5	557/572 4356-2	569 4356-1	#3&4-82593 #5-82793 4356	#5-82593 #1&2-82593 4354
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg										
DRPH	6-7	60-70	500 ^a	<50 ^b <100 ^b	<70 ^b	<60 ^b	<7,800 ^b			NA	<1,000 ^b	NA	<1,000	<50
GRPH	0.5-4	5-40	100	<3 ^b <4 ^b	<40 ^b	<52 ^b	150 ^b			NA	<100 ^b	<100 ^b	<100J	<1J
RRPH (Approx.)	10	100	2,000 ^a	<100	<150	<100	<100	12,000	<100	NA	<2,000	NA	<2,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.13 <0.20	<2.4J	<2.4J	<0.15		5.0J					
Benzene	0.002-0.06	0.02-0.6	0.5	<0.02 <0.04	<0.6J	<0.6J	<0.03	<0.02	<1 ^c	<1	<1	<1	<1	<0.02
Toluene	0.002-0.06	0.02-0.6		<0.02 <0.04	<0.6	<0.6	<0.03	<0.02	<1 ^c	<1	<1	<1	<1	<0.02
Ethylbenzene	0.002-0.06	0.02-0.6		<0.03 <0.04	<0.6J	<0.6J	<0.03	1.1	<1 ^c	<1	<1	<1	<1	<0.02
Xylenes (Total)	0.004-0.06	0.04-0.6		<0.04 <0.08	<0.6J	<0.6J	<0.06		<2 ^c	<2	<2	<2	<2	<0.04
HVOC 8010														
Trichloroethane	0.002-0.003	0.02-0.03		<0.03 <0.04	<0.03J	<0.03J	<0.03J	0.7J	NA	<1	<1	<1	<1	<0.02J
VOC 8260	0.020	0.100		<0.030 <0.150	NA	NA	<0.100	NA	<1	<1	<1	<1-12	<1	<0.020
SVOC 8270	0.200	2.20		<6.9 <15.0	NA	NA	<2.20	NA	NA	NA	<25	NA	<10	<0.010
Pesticides	0.002-0.05	0.02-0.5		<0.02 <0.5	NA	NA	<0.02J <0.5J	NA	NA	NA	<0.2J <10J	NA	NA	<0.02J <0.5J
PCBs	0.01-0.02	0.1-0.2	10	<0.1	<0.1	<0.1	<0.2	<0.1	<0.1	NA	<2	NA	<2J	<0.1

CT&E Data.

F&B Data.

Not analyzed.

Result is an estimate.

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

BTEX determined by 8260 method analysis.

☐ CT&E Data.
☒ F&B Data.
☐ Not analyzed.
☐ Result is an estimate.
☐ The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
☐ DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
☐ BTEX determined by 8260 method analysis.

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)														Matrix: Soil Units: mg/kg									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bgkd. Levels	Environmental Samples						Field Blanks			Lab Blanks									
					2S12-1	2S13	2S14-4.5	2S15-4	2S16-6 & 2S16-6 (Replicates)	AB01	2EB03	2TB03											
Laboratory Sample ID Numbers					4693-14	4693-15	4693-16	4693-17	4693-18	4693-19		4692-17	4692-18	4356 4692	4693								
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	mg/kg								
DRPH	4.00	4.00	500 ^a	<50 ^b <100 ^b	2,900J ^d	18.5 ^e	220 ^f	14.4 ^e	9,030 ^g	15,200 ^h	NA	NA	NA	NA	<4.00								
GRPH	0.400	0.400	100	<3J ^b <4J ^b	172	1.41	1.28	0.607	733	937	NA	NA	NA	NA	<0.400								
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.13 <0.20	NA	<0.125	0.031	<0.10	8,628	7,322													
Benzene	0.020	0.020	0.5	<0.02 <0.04	NA	<0.025	<0.020	<0.020	0.178	0.156	<1 ^c	<1 ^c	<1 ^c	<1	<0.020								
Toluene	0.020	0.020		<0.02 <0.04	NA	<0.025	<0.020	<0.020	1.56	0.856	<1 ^c	<1 ^c	<1 ^c	<1	<0.020								
Ethylbenzene	0.020	0.020		<0.02 <0.04	NA	<0.025	<0.020	<0.020	2.15	2.57	<1 ^c	<1 ^c	<1 ^c	<1	<0.020								
Xylenes (Total)	0.040	0.040		<0.04 <0.08	NA	<0.050	0.031	<0.040	4.74	3.74	<2 ^c	<2 ^c	<2 ^c	<2	<0.040								
VOC 8260	0.020	2.00		<0.030 <0.150	<2.00J	NA	NA	NA	NA	NA	<1	3.1U	3.1U	1.09	<0.020								
SVOC 8270	0.200	2.20-2.79	8,000	<6.90 <15.0	<2.20-2.79U	NA	NA	NA	NA	NA	NA	NA	NA	NA	<0.200-2.31								

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

U Compound is not present above the concentration listed.

a The action level for DRPH is based on conversations with ADEC; a final action level has not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTEX determined by 8260 method analysis.

d The laboratory reported that 2,500 mg/kg of the EPH pattern in this sample was not consistent with a middle distillate fuel.

e The laboratory reported that the EPH pattern in this sample was not consistent with a middle distillate fuel.

f The laboratory reported that 55.8 mg/kg of the EPH pattern in this sample was not consistent with a middle distillate fuel.

g The laboratory reported that 4,630 mg/kg of the EPH pattern in this sample was not consistent with a middle distillate fuel.

h The laboratory reported that 6,530 mg/kg of the EPH pattern in this sample was not consistent with a middle distillate fuel.

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)		Matrix: Sediment Units: mg/kg		Bkgd. Levels	Environmental Samples				Field Blanks			Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels		SD01	SD02	SD03	SD04	AB01	EB02	TB02		
Laboratory Sample ID Numbers					470	472	474	476	4356-5	557/572 4356-2	569 4356-1	#3&4-82593 #5-82793	#5-82593 #1&2-82593
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	mg/kg
DRPH	6	60	500 ^a	<50 ^b <100 ^b	10,400 ^b	<60 ^b	<60 ^b	1,570 ^b	NA	<1,000 ^b	NA	<1,000	<50
GRPH	0.1	1	100	<3 ^b <4 ^b	90 ^b	<1 ^b	<1 ^b	59 ^b	NA	<100 ^b	<100 ^b	<100J	<1J
RRPH (Approx.)	12	120	2,000 ^a	<100	<120	<120	<120	620	NA	<2,000	NA	<2,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.13 <0.20	10,76J	<0.15	<0.15	8.7J					
Benzene	0.003	0.03	0.5	<0.02 <0.04	<0.03	<0.03	<0.03	0.3J	<1 ^c	<1	<1	<1	<0.02
Toluene	0.003	0.03		<0.02 <0.04	0.18	<0.03	<0.03	0.9	<1 ^c	<1	<1	<1	<0.02
Ethylbenzene	0.003	0.03		<0.03 <0.04	2.5	<0.03	<0.03	1.5	<1 ^c	<1	<1	<1	<0.02
Xylenes (Total)	0.006	0.06		<0.04 <0.08	8.1J	<0.06	<0.06	6J	<2 ^c	<2	<2	<2	<0.04
HVOC 8010	0.003-0.005	0.03-0.05		<0.03 <0.04	<0.03J	<0.03J	<0.03J	<0.05J	NA	<1	<1	<1	<0.02J
PCBs	0.01-0.03	0.1-0.3	10	<0.1	<0.1	<0.1	<0.1	<0.3	NA	<2	NA	<2J	<0.1

☐ CT&E Data.
☒ F&B Data.
☒ NA
☐ J
☐ a
☐ b
☐ c

Not analyzed.
 Result is an estimate.

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
 DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
 BTEX determined by 8260 method analysis.

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)		Matrix: Sediment Units: mg/kg									Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks				
Laboratory Sample ID Numbers					2SD05	2SD06	2SD07	AB01	2EB03	2TB03		
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	4693-11	4693-12	4693-13	4356-1	4692-17	4692-16	4356	4693
DRPH	4.00	4.00	500 ^a	<50 ^b <100 ^c	10,300 ^d	4.61 ^d	5.19J ^d	µg/L	µg/L	µg/L	µg/L	mg/kg
GRPH	0.400	0.400	100	<3J ^e <4J ^e	16.3	<0.500	3.64	NA	NA	NA	NA	<4.00
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.13 <0.20	<2.5	<0.125	NA	<1 ^c	<1 ^c	<1 ^c	<1	<0.020
Benzene	0.020	0.025-0.500	0.5	<0.02 <0.04	<0.500	<0.025	NA	<1 ^c	<1 ^c	<1 ^c	<1	<0.020
Toluene	0.020	0.025-0.500		<0.02 <0.04	<0.500	<0.025	NA	<1 ^c	<1 ^c	<1 ^c	<1	<0.020
Ethylbenzene	0.020	0.025-0.500		<0.03 <0.04	<0.500	<0.025	NA	<1 ^c	<1 ^c	<1 ^c	<1	<0.020
Xylenes (Total)	0.040	0.050-1.000		<0.04 <0.08	<1	<0.050	NA	<2 ^c	<2 ^c	<2 ^c	<2	<0.040
VOC 8260												
Benzene	0.020	0.020	0.5	<0.030 <0.150	NA	NA	0.020J	<1	<1	<1	<1	<0.020
Ethylbenzene	0.020	0.020		<0.030 <0.150	NA	NA	0.085J	<1	<1	<1	<1	<0.020
Isopropylbenzene	0.020	0.020		<0.030 <0.150	NA	NA	0.022J	<1	<1	<1	<1	<0.020
p-Isopropyltoluene	0.020	0.020		<0.030 <0.150	NA	NA	0.021J	<1	<1	<1	<1	<0.020
Naphthalene	0.020	0.020		<0.030 <0.150	NA	NA	0.092J	<1	NA	NA	<1	<0.020

□ CT&E Data.

■ F&B Data.

■ Not analyzed.

J Result is an estimate.

U Compound is not present above the concentration listed.

a The action level for DRPH are based on conversations with ADEC; A final action level HAS not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTEX determined by 8260 method analysis.

d The laboratory reported that the EPH pattern in this sample was not consistent with a middle distillate fuel.

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)		Matrix: Sediment Units: mg/kg		Environmental Samples			Field Blanks			Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	2SD05	2SD06	2SD07	AB01	2EB03	2TB03	Lab Blanks
Laboratory Sample ID Numbers					4693-11	4693-12	4693-13	4356-1	4692-17	4692-16	4356 4692
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L
n-Propylbenzene	0.020	0.020		<0.030-<0.150	NA	NA	0.068J	<1	<1	<1	<1
Toluene	0.020	0.020		<0.030-<0.150	NA	NA	0.199J	<1	<1	<1	<0.020
1,2,4-Trimethylbenzene	0.020	0.020		<0.030-<0.150	NA	NA	0.287J	<1	<1	<1	<0.020
1,3,5-Trimethylbenzene	0.020	0.020		<0.030-<0.150	NA	NA	0.203J	<1	<1	<1	<0.020
Xylenes (Total)	0.040	0.040		<0.060-<0.300	NA	NA	0.451J	<2	<2	<2	<0.040
SVOC 8270	0.200	0.228		<6.9-<15.0	NA	NA	<0.228-2.88U	NA	NA	NA	NA <0.200-2.31

☐ CT&E Data.

☐ Not analyzed.

☐ Result is an estimate.

☐ Compound is not present above the concentration listed.

☐ NA J U

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

METALS ANALYSES												
Installation: Point Lay Site: Garage (SS06)		Matrix: Soil Units: mg/kg		Bkgd. Range from 7 DEW Line Installations	Environmental Samples				Field Blank		Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels		S03	S08				EB02		
Laboratory Sample ID Numbers					4354-4	4354-5				4356-2	4356 4354	
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg				µg/L	µg/L	
Aluminum	0.35	2		1,500-25,000	2,300	2,100				<100	≤100	
Antimony	N/A	50-63		<7.8-<230	<63J	<50				<100	<100	
Arsenic	0.11	50-63		<4.9-8.5	<63	<50				<100	<100	
Barium	0.024	1		27-390	290	280				<50	<50	
Beryllium	N/A	25-32		<2.6-6.4	<32	<25				<50	<50	
Cadmium	0.33	25-32		<3.0-<36	<32	<25				<50	<50	
Calcium	0.69	4		360-59,000	2,000	1,500				410	<200	
Chromium	0.066	1		<4.3-47	21	54				<50	<50	
Cobalt	N/A	5.0-6.3		<5.1-12	<6.3	<5.0				<100	<100	
Copper	0.045	25-32		<2.7-45	<32	<25				<50	<50	
Iron	0.50	2		5,400-35,000	14,000	20,000				<100	<100	
Lead	0.13	2		<5.1-22	92	195				<100	<100	
Magnesium	0.96	4		360-7,400	1,300	1,200				<200	<200	
Manganese	0.025	1		25-290	160J	270				<50	<50	
Molybdenum	N/A	25-32		<2.5-<11	<32	<25				<50	<50	
Nickel	0.11	1		4.2-46	16	12				<50	<50	
Potassium	23	100		<300-2,200	430	410				<5,000	<5,000	

☐ CT&E Data.
☐ NA Not analyzed.
☐ J Result is an estimate.

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)				Matrix: Soil Units: mg/kg		METALS ANALYSES						
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples				Field Blank		Lab Blanks	
					S03	S08				EB02		
Laboratory Sample ID Numbers					4354-4	4354-5				4356-2	4356 4354	
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg				µg/L	µg/L	
Selenium	1.2	50-63		<7.8-<170	<63	<50				<100	<100	
Silver	0.53	25-32		<3-<110	<32R	<25				<50	<50	
Sodium	0.55	5		<160-680	250	81				370	<250	
Thallium	0.011	0.27-0.31		<0.2-<1.2	<0.31	<0.27				<5	<5	
Vanadium	0.036	1		6.3-59	13	12				<50	<50	
Zinc	0.16	1		9.2-95	85	59				<50	<50	

☐ CT&E Data.
☐ Not analyzed.
☐ Result has been rejected.

☐ NA
☐ R

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)		Matrix: Surface Water Units: µg/L										Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			TB02	
					SW01 & SW03 (Duplicates)	SW02	SW04	AB01	EB02	SW04	AB01	EB02	
Laboratory Sample ID Numbers					501 619	500/626	499/616	4356-5	557/572 4356-2	567/622 4356-12 4354-10 4358-5	4356-5	569 4356-1	#5-82793 #3&4-82593 4354 4356 4358
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000 ^b	<1,000 ^b	<1,000 ^b	<1,000 ^b	NA	<1,000 ^b		NA	NA	<1,000
GRPH	10	100		<50 ^b	<100 ^b	<100 ^b	<100 ^b	NA	<100 ^b		NA	<100 ^b	<100J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	<2,000	NA	<2,000		NA	NA	<2,000
BTEX (8020/8020 Mod.)													
Benzene	0.1	1	5	<1	<1	<1	<1	<1 ^c	<1		<1 ^c	<1	<1
Toluene	0.1	1	1,000	<1	<1	<1	<1	<1 ^c	<1		<1 ^c	<1	<1
Ethylbenzene	0.1	1	700	<1	<1	<1	<1	<1 ^c	<1		<1 ^c	<1	<1
Xylenes (Total)	0.2	2	10,000	<2	<2	<2	<2	<2 ^c	<2		<2 ^c	<2	<2
HVOC 8010	0.1	1		<1	<1	<1	NA	NA	<1		NA	<1	<1
VOC 8260													
Naphthalene	1	1		<1	NA	2.1	NA	<1	<1		NA	<1	<1
1,3,5- Trimethylbenzene	1	1		<1	NA	1.2	NA	<1	<1		NA	<1	<1
SVOC 8270	10	10		<20-<31	NA	<10	NA	NA	<25		NA	NA	<10

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTEX determined by 8260 method analysis.

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)		Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)						Field Blank		Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	SW02	Environmental Sample				EB02		
Laboratory Sample ID Numbers					4356-12 4354-10 4358-5					4356-2		4354 4356 4358
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L					µg/L		µg/L
Aluminum	17.4	100		<100-350 (<100-340)	<100 (<100)					<100 (<100)		<100 (<100)
Antimony	N/A	100	6	<100 (<100)	<100 (<100)					<100 (<100)		<100 (<100)
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)					<100 (<100)		<100 (<100)
Barium	1.2	50	2,000	<50-93 (<50-91)	360 (340)					<50 (<50)		<50 (<50)
Beryllium	N/A	50	4	<50 (<50)	<50 (<50)					<50 (<50)		<50 (<50)
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)					<50 (<50)		<50 (<50)
Calcium	34.5	200		4,500-88,000 (4,100-86,000)	52,000 (51,000)					410 (<200)		<200 (<200)
Chromium	3.29	50	100	<50 (<50)	<50 (<50)					<50 (<50)		<50 (<50)
Cobalt	N/A	100		<100 (<100)	<100 (<100)					<100 (<100)		<100 (<100)
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)					<50 (<50)		<50 (<50)
Iron	25	100		180-2,800 (<100-1,600)	5,600 (880)					<100 (<100)		<100 (<100)

☐ CT&E Data.
N/A Not available.

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)			Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)							Field Blank		Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	SW02							EB02		
Laboratory Sample ID Numbers					4356-12 4354-10 4358-5							4356-2		4354 4356 4358
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L							µg/L		µg/L
Lead	6.6	100	15	<100 (<100)	<100 (<100)							<100 (<100)		<100 (<100)
Magnesium	47.8	200		<5,000-53,000 (2,600-54,000)	26,000 (26,000)							<200 (<200)		<200 (<200)
Manganese	1.24	50		<50-510 (<50-120)	1,700 (1,700)							<50 (<50)		<50 (<50)
Molybdenum	N/A	50		<50 (<50)	<50 (<50)							<50 (<50)		<50 (<50)
Nickel	5.5	50	100	<50 (<50)	<50 (<50)							<50 (<50)		<50 (<50)
Potassium	1,154	5,000		<5,000 (<5,000)	<5,000 (<5,000)							<5,000 (<5,000)		<5,000 (<5,000)
Selenium	62.4	100	50	<100 (<100)	<100 (<100)							<100 (<100)		<100 (<100)
Silver	2.6	50	50	<50 (<50)	<50J (<50)J							<50 (<50)		<50 (<50)
Sodium	27.7	250		8,400-410,000 (8,200-450,000)	45,000 (44,000)							370 (400)		<250 (<250)
Thallium	0.57	5	2	<5 (<5)	<5 (<5)							<5 (<5)		<5 (<5)

☐ CT&E Data.
☐ N/A
☐ J
 Not available.
 Result is an estimate.

TABLE G-4. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Garage (SS06)			Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)							
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Sample					Field Blank		Lab Blanks
					SW02						EB02	
Laboratory Sample ID Numbers					4356-12 4354-10 4358-5						4356-2	4354 4356 4358
ANALYSES	µg/L	µg/L	µg/L	µg/L							µg/L	µg/L
Vanadium	1.8	50		<50 (<50)	<50 (<50)						<50 (<50)	<50 (<50)
Zinc	8.2	50		<50-160 (<50)	<61 (<50)						<50 (<50)	<50 (<50)

TABLE G-5. DRAINAGE PATHWAY FROM POL TANKS ANALYTICAL DATA SUMMARY

Installation: Point Lay Site: Drainage Pathway from POL Tanks (S507)											
Matrix: Sediment Units: mg/kg											
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks		Lab Blanks
					SD01	SD02	SD03	SD04	AB01	EB02	TB02
Laboratory Sample ID Numbers					628	630 4354-9	632	634	4356-5	557/572 4356-2	569 4356-1
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg	µg/L
DRPH	6-9	60-90	500 ^a	<50 ^b <100 ^b	<60 ^b	<60 ^b	<80 ^b	<70 ^b	NA	<1,000 ^b	NA
GRPH	0.3-0.4	3-4	100	<3.7 ^a <4.3 ^b	<4.3 ^b	<4.3 ^b	<3.3 ^b	3.3 ^b	NA	<100 ^b	<100J ^b
RRPH (Approx.)	12	120	2,000 ^a	<100	<120	<120	<120	<120	NA	<2,000	NA
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.13 <0.20	<0.20	<0.15	<0.15	0.71J			
Benzene	0.003-0.004	0.03-0.04	0.5	<0.02 <0.04	<0.04	<0.03	<0.03	0.07J	<1 ^c	<1	<1
Toluene	0.003-0.004	0.03-0.04		<0.02 <0.04	<0.04	<0.03	<0.03	0.04	<1 ^c	<1	<1
Ethylbenzene	0.003-0.004	0.03-0.04		<0.03 <0.04	<0.04	<0.03	<0.03	0.2	<1 ^c	<1	<1
Xylenes (Total)	0.006-0.008	0.06-0.08		<0.04 <0.08	<0.05	<0.06	<0.06	0.4J	<2 ^c	<2	<2
VOC 8260	0.020	0.025		<0.030 <0.150	NA	<0.025	NA	NA	<1	<1	<1-12
Pesticides	0.2-5	2-50		<0.02J <0.5J	<2 <50	NA	NA	NA	NA	<0.2J <50J	NA
											<0.2J <0.5J

CT&E Data.

F&B Data.

Not analyzed.

Result is an estimate.

The action levels for DRPH and GRPH are based on conversations with ADEC; final action levels have not yet been determined.

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

Total BTEX determined by 8260 method analysis.

☐ CT&E Data.
☒ F&B Data.
☐ Not analyzed.
☐ Result is an estimate.
☐ The action levels for DRPH and GRPH are based on conversations with ADEC; final action levels have not yet been determined.
☐ DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
☐ Total BTEX determined by 8260 method analysis.

TABLE G-5. DRAINAGE PATHWAY FROM POL TANKS ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Drainage Pathway from POL Tanks (SS07) Matrix: Surface Water Units: µg/L												
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			Lab Blanks
					SW01	SW02	SW03		AB01	EB02	TB02	
Laboratory Sample ID Numbers					558/582	584/586 4356-7	588/589		4356-5	557/572 4356-2	569 4356-1	#5-82793 #384-82593 4356
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L		µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000 ^b	<1,000 ^b	<1,000 ^b	<1,000 ^b		NA	<1,000 ^b	NA	<1,000
GRPH	10	100		<50 ^b	<100 ^b	<100 ^b	<100 ^b		NA	<100 ^b	<100 ^b	<100J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	<2,000		NA	<2,000	NA	<2,000
BTX (8020/8020 Mod.)												
Benzene	0.1	1	5	<1	<1	<1	<1		<1 ^c	<1	<1	<1
Toluene	0.1	1	1,000	<1	<1	<1	<1		<1 ^c	<1	<1	<1
Ethylbenzene	0.1	1	700	<1	5J	<1	<1		<1 ^c	<1	<1	<1
Xylenes (Total)	0.2	2	10,000	<2	12J	<2	<2		<2 ^c	<2	<2	<2
HVOC 8010												
Tetrachloroethene	0.1	1	5	<1	2	<1	<1		NA	<1	<1	<1
Trichloroethene	0.1	1	5	<1	4	<1	133		NA	<1	<1	<1
VOC 8260												
cis-1,2-Dichloroethene	1	1	70	<1	NA	7.5	NA		<1	<1	<1	<1

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTX determined by 8260 method analysis.

TABLE G-5. DRAINAGE PATHWAY FROM POL TANKS ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Drainage Pathway from POL Tanks (SS07)												
Matrix: Surface Water Units: µg/L												
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			Lab Blanks
					SW01	SW02	SW03			AB01	EB02	
Laboratory Sample ID Numbers												
ANALYSES	µg/L	µg/L	µg/L	µg/L		558/582	584/586 4356-7	588/589		4356-5	557/572 4356-2	569 4356-1
trans-1,2-Dichloroethene	1	1	100	<1		NA	2.1	NA		<1	<1	<1
1,2,3-Trichlorobenzene	1	1		<1		NA	1.3	NA		<1	<1	<1
Trichloroethene	1	1	5	<1		NA	3.9	NA		<1	<1	<1
SVOC 8270	10	19		<20-<31		NA	<19	NA		NA	<25	NA
TOC	5,000	5,000		31,700-40,000		NA	15,500	NA		NA	NA	NA
TSS	100	200		6,000-77,000		NA	28,000	NA		NA	NA	<200
TDS	10,000	10,000		149,000-151,000		NA	1,976,000	NA		NA	NA	<10,000

☐ NA
CT&E Data.
Not analyzed.

TABLE G-5. DRAINAGE PATHWAY FROM POL TANKS ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Drainage Pathway from POL Tanks (SS07)													Matrix: Surface Water Units: µg/L		
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples						Field Blanks			Lab Blanks	
					2SW04	2SW05	2SW06				AB01	2EB03	2TB03		
Laboratory Sample ID Numbers					4692-1	4692-4	4692-5					4356-5	4692-17	4692-16	4356 4692
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L					µg/L	µg/L	µg/L	µg/L
DRPH	100	100-150		<1,000 ^b	961 ^{ad}	<150	260 ^{ad}					NA	NA	NA	<100
GRPH	20	20		<50 ^{JP}	189 ^a	<20	<20					NA	NA	NA	<20
VOC 8260															
Benzene	1	1	5	<1	1.7	<1	<1					<1	<1	<1	<1
Chloromethane	1	1		<1	3.2B	2.6B	3.6B					<1	3.1U	3.1U	1.09
1,1-Dichloroethene	1	1	7	<1	2.3	<1	<1					<1	<1	<1	<1
cis-1,2-Dichloroethene	1	1	70	<1	178	<1	<1					<1	4.6	1.5	<1
trans-1,2-Dichloroethene	1	1	100	<1	3.6	<1	<1					<1	<1	<1	<1
Trichloroethene	1	1	5	<1	6.8	<1	<1					<1	1.7	<1	<1

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

B The analyte was detected in the associated blank.

J Result is an estimate.

U Compound is not present above the concentration listed.

a Total petroleum hydrocarbons in these water samples exceed the 15 µg/L stated for fresh water in ADEC's Water Quality Criteria 18AAC70 (ADEC 1989).

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

d The laboratory reported that the EPH pattern in this sample was not consistent with a middle distillate fuel.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY

Installation: Point Lay		Matrix: Soil		Field Blanks										Lab Blanks	
Site: Crushed Drum Area (SS08)		Units: mg/kg		Environmental Samples										Field Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	S01 & S07 (Replicates)	S02-2	S03-2	S04	S05-2	S06	AB01	EB01	TB01	Lab Blanks	
Laboratory Sample ID Numbers					417	429	419	421	423	425	4356-5	443/446 4328-2	441 4328-1	#5-82593 #384-82493 4356 4328	#5-82483 #182-82493 #384-82793 4327
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/L	mg/L	mg/L	μg/L	mg/kg
DRPH	5	50	500 ^a	<50 ^b <100 ^b	7,200 ^b	7,200 ^b	<50 ^b	<50 ^b	<50 ^b	2,800 ^b	NA	<1,000 ^b	NA	<1,000	<50
GRPH	0.2	2	100	<3 ^b <4 ^b	550 ^b	470 ^b	<2 ^b	<2 ^b	<2 ^b	330 ^b	NA	<50 ^b	<50 ^b	<100J	<2J
RRPH (Approx.)	10	100	2,000 ^a	<100	<100	<100	<100	<100	<100	<100	NA	<2,000	NA	<2,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.13 <0.20	33.08R	49.7R	<0.1	<0.1	<0.1	37.2J					
Benzene	0.002	0.02	0.5	<0.02 <0.04	0.26R	<0.02	<0.02	<0.02	<0.02	1.4	<1 ^c	<1	<1	<1	<0.02
Toluene	0.002	0.02		<0.02 <0.04	1.6R	1.7R	<0.02	<0.02	<0.02	2.8	<1 ^c	<1	<1	<1	<0.02
Ethylbenzene	0.002	0.02		<0.03 <0.04	12R	14R	<0.02	<0.02	<0.02	11J	<1 ^c	<1	<1	<1	<0.02
Xylenes (Total)	0.004	0.04		<0.04 <0.08	18R	34R	<0.04	<0.04	<0.04	22J	<2 ^c	<2	<2	<2	<0.04
HVOC 8010	0.002	0.02		<0.03 <0.04	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	NA	<1	<1	<1	<0.02J
VOC 8260															
n-Butylbenzene	0.020	0.250		<0.030 <0.150	NA	NA	NA	NA	NA	4.95	<1	<1	<1	<1	<0.020
sec-Butylbenzene	0.020	0.250		<0.030 <0.150	NA	NA	NA	NA	NA	1.99	<1	<1	<1	<1	<0.020
Ethylbenzene	0.020	0.250		<0.030 <0.150	NA	NA	NA	NA	NA	1.55	<1	<1	<1	<1	<0.020
Isopropylbenzene	0.020	0.250		<0.030 <0.150	NA	NA	NA	NA	NA	1.18	<1	<1	<1	<1	<0.020

☐ CT&E Data.
☒ F&B Data.
☒ Not analyzed.

☐ J
☐ R
☐ a
☐ b
☐ c

Result is an estimate.

Result has been rejected.

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

BTEX determined by 8260 method analysis.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)																Matrix: Soil Units: mg/kg	
Parameters	Detect Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples							Field Blanks			Lab Blanks		
					S01 & S07 (Replicates)		S02-2	S03-2	S04	S05-2	S06	AB01	EB01	TB01			
Laboratory Sample ID Numbers					417	429	419	421	423	425	427 4327-2	443/448 4328-2	441 4328-1	#5-82493 #182-82493 #384-82793 4358 4327			
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	mg/kg	
p-Isopropyl- toluene	0.020	0.250		<0.030-<0.150	NA	NA	NA	NA	NA	NA	1.94	<1	<1	<1	<1	<0.020	
Naphthalene	0.020	0.250		<0.030-<0.150	NA	NA	NA	NA	NA	NA	4.94	<1	<1	<1	<1	<0.020	
n-Propyl- benzene	0.020	0.250		<0.030-<0.150	NA	NA	NA	NA	NA	NA	3.09	<1	<1	<1	<1	<0.020	
Toluene	0.020	0.250		<0.030-<0.150	NA	NA	NA	NA	NA	NA	1.60	<1	<1	<1	<1	<0.020	
1,2,4- Trimethyl- benzene	0.020	0.250		<0.030-<0.150	NA	NA	NA	NA	NA	NA	16.1	<1	<1	<1	<1	<0.020	
1,3,5- Trimethyl- benzene	0.020	0.250		<0.030-<0.150	NA	NA	NA	NA	NA	NA	6.82	<1	<1	<1	<1	<0.020	
Xylenes (Total)	0.040	0.500		<0.060-<0.300	NA	NA	NA	NA	NA	NA	9.91	<2	<2	<2	<2	<0.040	
SVOC 8270																	
Naphthalene	0.200	0.230		<6.90-<15.0	NA	NA	NA	NA	NA	NA	2.05	NA	<36	NA	<10	<0.200	
2-Methyl- naphthalene	0.200	0.230		<6.90-<15.0	NA	NA	NA	NA	NA	NA	1.28	NA	<36	NA	<10	<0.200	
Pesticides	0.002-0.05	0.02-0.5		<0.02J-<0.5J	<0.02J-<0.5J	<0.2J-<0.5J	NA	NA	NA	NA	NA	NA	<0.2J-<0.5J	NA	<0.02J-<0.5J	NA	
PCBs	0.01	0.1	10	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	NA	<2	NA	<0.1	<0.1-<0.5	

☐ CT&E Data.
☒ F&B Data.
☒ Not analyzed.
 Result is an estimate.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)		Matrix: Soil Units: mg/kg											
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			Lab Blanks
					2S08	2S09	2S10-1	2S11 & 2S15 (Replicates)	2S12	AB01	2EB03	2TB03	
Laboratory Sample ID Numbers					4693-1	4693-4	4693-5	4693-8	4693-9	4356-5	4692-17	4692-18	4356 4692
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L
DRPH	4.00	4.00	500 ³	<30 ³ < 100 ³	3.050	6,290	17,500	<4.00	4.19 ^d	NA	NA	NA	NA
GRPH	0.400	0.400-0.500	100	<3 ³ < 4 ³	22.6	2,430	2,330	<0.500	1.97	NA	NA	NA	NA
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.13 < 0.20	NA	NA	122.2	<0.125	NA	<0.125	<1 ^c	<1 ^c	<1
Benzene	0.020	0.025-4.00	0.5	<0.02 < 0.04	NA	NA	<4.00	<0.025	NA	<1 ^c	<1 ^c	<1 ^c	<1
Toluene	0.020	0.025-4.00		<0.02 < 0.04	NA	NA	12.4	<0.025	NA	<1 ^c	<1 ^c	<1 ^c	<1
Ethylbenzene	0.020	0.025-4.00		<0.03 < 0.04	NA	NA	18.2	<0.025	NA	<1 ^c	<1 ^c	<1 ^c	<1
Xylenes (Total)	0.040	0.050-8.00		<0.04 < 0.08	NA	NA	91.6	<0.050	NA	<2 ^c	<2 ^c	<2 ^c	<2
VOC 8260													
Benzene	0.020	0.020-2.50	0.5	<0.030 < 0.150	<0.100J	<2.50	NA	NA	0.021	<1	<1	<1	<1
n-Butylbenzene	0.020	0.020-2.50		<0.030 < 0.150	<0.100J	11.6	NA	NA	<0.020	<1	<1	<1	<1
sec-Butylbenzene	0.020	0.020-2.50		<0.030 < 0.150	<0.100J	5.03	NA	NA	<0.020	<1	<1	<1	<1
Ethylbenzene	0.020	0.020-2.50		<0.030 < 0.150	<0.100J	5.35	NA	NA	0.057	<1	<1	<1	<1
Isopropylbenzene	0.020	0.020-2.50		<0.030 < 0.150	<0.100J	3.23	NA	NA	<0.020	<1	<1	<1	<1
p-Isopropyltoluene	0.020	0.020-2.50		<0.030 < 0.150	<0.100J	6.04	NA	NA	<0.020	<1	<1	<1	<1
Naphthalene	0.020	0.020-2.50		<0.030 < 0.150	<0.100J	26.2	NA	NA	0.100	<1	<1	<1	<1

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

Result is an estimate.

The action level for DRPH is based on conversations with ADEC; a final action level has not yet been determined.

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

BTEX determined by 8260 method analysis.

The laboratory reported that the EPH pattern in this sample was not consistent with a middle distillate fuel.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)														Matrix: Soil Units: mg/kg	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples						Field Blanks			Lab Blanks	
					2S08	2S09	2S10-1	2S11 & 2S15 (Replicates)	2S12	AB01	2EB03	2TB03			
Laboratory Sample ID Numbers					4693-1	4693-4	4693-5	4693-8	4693-9	4356-5	4692-17	4692-18	4356 4692	4693	
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	mg/kg	
n-Propylbenzene	0.020	0.020-2.50		<0.030-<0.150	<0.100J	7.88	NA	NA	0.023	<1	<1	<1	<1	<0.020	
Toluene	0.020	0.020-2.50		<0.030-<0.150	0.308J	5.72	NA	NA	0.240	<1	<1	<1	<1	<0.020	
1,2,4- Trimethylbenzene	0.020	0.020-2.50		<0.030-<0.150	0.264J	48.0	NA	NA	0.142	<1	<1	<1	<1	<0.020	
1,3,5- Trimethylbenzene	0.020	0.020-2.50		<0.030-<0.150	0.153J	19.6	NA	NA	0.054	<1	<1	<1	<1	<0.020	
Xylenes (Total)	0.040	0.040-5.00		<0.080-<0.300	0.490J	38.1	NA	NA	0.500	<2	<2	<2	<2	<0.040	
SVOC 8270															
Acenaphthene	0.200	0.220-2.28		<6.90-<15.0	NA	1.85J	NA	NA	NA	NA	NA	NA	<10	<0.200	
di-n-Butylphthalate	0.200	0.220-2.28	8,000	<6.90-<15.0	NA	8.48B	NA	NA	1.95B	NA	NA	NA	<10	2.31	
Dibenzofuran	0.200	0.220-2.28		<6.90-<15.0	NA	1.83J	NA	NA	<0.220	NA	NA	NA	<10	<0.200	
Fluorene	0.200	0.220-2.28		<6.90-<15.0	NA	1.95J	NA	NA	<0.220	NA	NA	NA	<10	<0.200	
2-Methyl- naphthalene	0.200	0.220-2.28		<6.90-<15.0	NA	11.7	NA	NA	<0.220	NA	NA	NA	<10	<0.200	
Naphthalene	0.200	0.220-2.28		<6.90-<15.0	NA	7.66	NA	NA	<0.220	NA	NA	NA	<10	<0.200	
Phenanthrene	0.200	0.220-2.28		<6.90-<15.0	NA	2.44	NA	NA	<0.220	NA	NA	NA	<10	<0.200	

☐ CT&E Data.
☐ Not analyzed.
☐ The analyte was detected in the associated blank.
☐ Result is an estimate.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)		Matrix: Sediment Units: mg/kg		Environmental Samples			Field Blanks			Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	SD01	SD02	SD03	AB01	EB01	TB01	
Laboratory Sample ID Numbers					463 4327-1	465	467	4356-5	443/446 4328-2	441 4328-1	#5-82593 #1&2-82493 #3&4-82793 4328 4356 4327
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	mg/kg
DRPH	20	200	500 ^a	<50 ^b <100 ^b	1,100 ^b	<700 ^b	<200 ^b	NA	<1,000 ^b	NA	<50
GRPH	0.2	2	100	<3 ^b <4 ^b	46 ^b	<2 ^b	<2 ^b	NA	<50 ^b	<50 ^b	<2J
RRPH (Approx.)	40-150	400-1,500	2,000 ^a	<100	120	<1,500	<400	NA	<2,000	NA	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.13- <0.20	10.83J	<1.44	<0.4				
Benzene	0.002-0.029	0.02-0.29	0.5	<0.02- <0.04	0.23J	<0.29	<0.08	<1 ^c	<1	<1	<0.02
Toluene	0.002-0.029	0.02-0.29		<0.02- <0.04	1	<0.29	<0.08	<1 ^c	<1	<1	<0.02
Ethylbenzene	0.002-0.029	0.02-0.29		<0.03- <0.04	1.9	<0.29	<0.08	<1 ^c	<1	<1	<0.02
Xylenes (Total)	0.004-0.058	0.04-0.58		<0.04- <0.06	7.7J	<0.57	<0.16	<2 ^c	<2	<2	<0.04
HVOC 8010	0.002-0.029	0.02-0.29		<0.03- <0.04	<0.02J	<0.29	<0.08	NA	<1	<1	<0.02J
VOC 8260											
n-Butylbenzene	0.020	0.250		<0.030- <0.150	3.66J	NA	NA	<1	<1	<1	<0.020
sec-Butylbenzene	0.020	0.250		<0.030- <0.150	1.51J	NA	NA	<1	<1	<1	<0.020
Ethylbenzene	0.020	0.250		<0.030- <0.150	2.30J	NA	NA	<1	<1	<1	<0.020
Isopropylbenzene	0.020	0.250		<0.030- <0.150	1.34J	NA	NA	<1	<1	<1	<0.020

☐ CT&E Data.
☒ F&B Data.
☒ NA
☐ J
☐ a
☐ b
☐ c

Result is an estimate.
 The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
 DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
 BTEX determined by 8260 method analysis.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)		Matrix: Sediment Units: mg/kg									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks			Lab Blanks
					SD01	SD02	SD03	AB01	EB01	TB01	
Laboratory Sample ID Numbers					463 4327-1	465	467	4356-5	443/446 4328-2	441 4328-1	#5-82593 4328 #1&2-82493 4327
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	mg/kg
p-Isopropyltoluene	0.020	0.250		<0.030-<0.150	1.45J	NA	NA	<1	<1	<1	<0.020
Naphthalene	0.020	0.250		<0.030-<0.150	8.10J	NA	NA	<1	<1	<1	<0.020
n-Propylbenzene	0.020	0.250		<0.030-<0.150	2.81J	NA	NA	<1	<1	<1	<0.020
Toluene	0.020	0.250		<0.030-<0.150	2.11J	NA	NA	<1	<1	<1	<0.020
1,2,4-Trimethylbenzene	0.020	0.250		<0.030-<0.150	13.9J	NA	NA	<1	<1	<1	<0.020
1,3,5-Trimethylbenzene	0.020	0.250		<0.030-<0.150	5.31J	NA	NA	<1	<1	<1	<0.020
Xylenes (Total)	0.040	0.500		<0.060-<0.300	14.87J	NA	NA	<2	<2	<2	<0.040
SVOC 8270											
Benzyl Alcohol	0.200	0.240		<6.90-<15.0	0.680	NA	NA	NA	<36	NA	<10
Naphthalene	0.200	0.240		<6.90-<15.0	2.02	NA	NA	NA	<36	NA	<10
2-Methylnaphthalene	0.200	0.240		<6.90-<15.0	2.43	NA	NA	NA	<36	NA	<10
Phenanthrene	0.200	0.240		<6.90-<15.0	0.661	NA	NA	NA	<36	NA	<10
Fluoranthene	0.200	0.240		<6.90-<15.0	0.261	NA	NA	NA	<36	NA	<10
PCBs	0.01-0.15	0.1-1.5	10	<0.1	<0.1	<1.5	<0.4	NA	<2	NA	<10

☐ CT&E Data.
☒ F&B Data.
☐ NA
 Not analyzed.
 Result is an estimate.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)				Matrix: Soil/Sediment Units: mg/kg		METALS ANALYSES									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples						Field Blank		Lab Blanks		
					S06	SD01								EB01	
Laboratory Sample ID Numbers					4327-2	4327-1						4328-2	4328 4327		
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg						µg/L	µg/L		
Aluminum	0.35	2		1,500-23,000	3,400	1,700						<100	<100		
Antimony	N/A	56		<7.8-<230	<56J	<56J						<100	<100		
Arsenic	0.11	56		<4.9-7.0	<56	<56						<100	<100		
Barium	0.024	1		27-390	360	330						<50	<50		
Beryllium	N/A	2.8		<2.6-6.4	<2.8	<2.8						<50	<50		
Cadmium	0.33	2.8		<3.0-<27	<2.8	<2.8						<50	<50		
Calcium	0.69	4		360-59,000	1,200	1,200						<200	<200		
Chromium	0.066	1		<4.3-47	6.7	4.7						<50	<50		
Cobalt	N/A	5.6		<5.1-12	<5.6	<5.6						<100	<100		
Copper	0.045	1		<2.7-45	8.6	7.1						<50	<50		
Iron	0.50	2		5,400-35,000	23,000	21,000						<100	<100		
Lead	0.13	5.6		<5.1-22	<5.6	<5.6						<100	<100		
Magnesium	0.96	4		360-7,400	1,600	920						<200	<200		
Manganese	0.025	1		25-290	220J	200J						<50	<50		
Molybdenum	N/A	2.8		<2.5-<11	<2.8	<2.8						<50	<50		
Nickel	0.11	1		4.2-46	15	13						<50	<50		
Potassium	23	100		<300-2,200	590	420						<5,000	<5,000		

☐ CT&E Data.
☐ N/A Not available.
☐ J Result is an estimate.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)			Matrix: Soil/Sediment Units: mg/kg		METALS ANALYSES								
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples						Field Blank		Lab Blanks
					S06	SD01							
Laboratory Sample ID Numbers					4327-2	4327-1					4328-2	4328 4327	
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg					µg/L	µg/L	
Selenium	1.2	• 2-56		<7.8-<170	17J	<56					<100	<100	
Silver	0.53	28		<3-<110	<28R	<28R					<50J	<50	
Sodium	0.55	5		<160-680	94	87					<250	<250	
Thallium	0.011	0.28-0.29		<0.2-<0.82	<0.28	<0.29					<5	<5	
Vanadium	0.036	1		6.3-59	19	13					<50	<50	
Zinc	0.16	1		9.2-95	37	44					<50	<50	

☐ J R
CT&E Data.
Result is an estimate.
Result has been rejected.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)		Matrix: Surface Water Units: µg/L									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks			Lab Blanks
					SW01	SW02		AB01	EB02	TB02	
Laboratory Sample ID Numbers					573/576 4356-6	577/578 4356-7		4356-5	557/572 4356-2	569 4356-1	#5-82793 #3&4-82593 4356
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L		µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000 ^b	<1,000 ^b	<1,000 ^b		NA	<1,000 ^b	NA	<1,000
GRPH	10	100		<500 ^b	<100 ^b	<100 ^b		NA	<100 ^b	<100 ^b	<100J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000		NA	<2,000	NA	<2,000
BTEX (8020/8020 Mod.)											
Benzene	0.1	1	5	<1	15	<1		<1 ^c	<1	<1	<1
Toluene	0.1	1	1,000	<1	43	<1		<1 ^c	<1	<1	<1
Ethylbenzene	0.1	1	700	<1	12J	<1		<1 ^c	<1	<1	<1
Xylenes (Total)	0.2	2	10,000	<2	82J	<2		<2 ^c	<2	<2	<2
HVOC 8010	0.1	1		<1	<1	<1		NA	<1	<1	<1
VOC 8260											
Benzene	1	1	5	<1	7.0	NA		<1	<1	<1	<1
Ethylbenzene	1	1	700	<1	6.5	NA		<1	<1	<1	<1

☐ CT&E Data.
☒ F&B Data.
☒ Not analyzed.
 NA Result is an estimate.
 J DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
 b BTEX determined by 8260 method analysis.
 c

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)		Matrix: Surface Water Units: µg/L									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks			Lab Blanks
					SW01	SW02		AB01	EB02	TB02	
Laboratory Sample ID Numbers					573/576 4356-6	577/578 4356-7		4356-5	557/572 4356-2	569 4356-1	#5-82793 4356
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L		µg/L	µg/L	µg/L	µg/L
Isopropylbenzene	1	1		<1	1.2	NA		<1	<1	<1	<1
Naphthalene	1	1		<1	15	NA		<1	<1	<1	<1
n-Propylbenzene	1	1		<1	1.3	NA		<1	<1	<1	<1
Toluene	1	1	1,000	<1	30	NA		<1	<1	<1	<1
1,2,4-Trimethylbenzene	1	1		<1	14	NA		<1	<1	<1	<1
1,3,5-Trimethylbenzene	1	1		<1	11	NA		<1	<1	<1	<1
Xylenes (Total)	2	2	10,000	<2	66	NA		<2	<2	<2	<1
SVOC 8270	10	17		<20-<31	<17	NA		NA	<25	NA	<10
PCBs	0.2	2	0.5	<2	<2	<2		NA	<2	NA	<2J
TOC	5,000	5,000		31,700-40,000	19,600	NA		NA	NA	NA	NA
TSS	100	200		6,000-77,000	23,500	NA		NA	NA	NA	<200
TDS	10,000	10,000		149,000-151,000	596,000J	NA		NA	NA	NA	<10,000

☐ CT&E Data.
☒ F&B Data.
☒ Not analyzed.
☒ Result is an estimate.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)			Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)										
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Sample							Field Blank		Lab Blank	
					SW01										EB02
Laboratory Sample ID Numbers					4356-6	µg/L							4356-2		4356
ANALYSES	µg/L	µg/L	µg/L	µg/L									µg/L		µg/L
Aluminum	17.4	100		<100-350 (<100-340)	120 (<100)								<100 (<100)		<100
Antimony	N/A	100	6	<100 (<100)	<100 (<100)								<100 (<100)		<100
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)								<100 (<100)		<100
Barium	1.2	50	2,000	<50-93 (<50-91)	330 (270)								<50 (<50)		<50
Beryllium	N/A	50	4	<50 (<50)	<100 (<50)								<50 (<50)		<50
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)								<50 (<50)		<50
Calcium	34.5	200		4,500-88,000 (4,100-86,000)	57,000 (57,000)								410 (<200)		<200
Chromium	3.29	50	100	<50 (<50)	<50 (<50)								<50 (<50)		<50
Cobalt	N/A	100		<100 (<100)	<100 (<100)								<100 (<100)		<100
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)								<50 (<50)		<50
Iron	25	100		180-2,800 (<100-1,600)	6,300 (<100)								<100 (<100)		<100
Lead	6.6	100	15	<100 (<100)	<100 (<100)								<100 (<100)		<100

☐ CT&E Data.
N/A Not available.

TABLE G-6. CRUSHED DRUM AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Point Lay Site: Crushed Drum Area (SS08)				Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Sample						Field Blank		Lab Blank		
					SW01									EB02	
Laboratory Sample ID Numbers					4356-6							4356-2		4356	
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L							µg/L		µg/L	
Magnesium	47.8	200		2,900-53,000 (2,600-54,000)	29,000 (28,000)							<200 (<200)		<200	
Manganese	1.24	50		<50-510 (<50-120)	610 (540)							<50 (<50)		<50	
Molybdenum	N/A	50		<50 (<50)	<50 (<50)							<50 (<50)		<50	
Nickel	5.5	50	100	<50 (<50)	<50 (<50)							<50 (<50)		<50	
Potassium	1,154	5,000		<5,000-5,000 (<5,000-5,000)	5,400 (6,100)							<5,000 (<5,000)		<5,000	
Selenium	62.4	100	50	<100 (<100)	<100 (<100)							<100 (<100)		<100	
Silver	2.6	50	50	<50 (<50)	<50 (<50)J							<50 (<50)		<50	
Sodium	27.7	250		8,400-410,000 (8,200-450,000)	28,000 (29,000)							370 (400)		<250	
Thallium	0.57	5	2	<5 (<5)	<5 (<5)							<5 (<5)		<5	
Vanadium	1.8	50		<50 (<50)	<50 (<50)							<50 (<50)		<50	
Zinc	8.2	50		<50-160 (<50)	<50 (<50)							<50 (<50)		<50	

☐ CT&E Data.
☐ N/A
☐ J Not available.
 Result is an estimate.