



## **FINAL REPORT**

# **HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT OF COMINCO'S POLARIS MINE ON LITTLE CORNWALLIS ISLAND: DERIVATION OF SOIL QUALITY REMEDIATION OBJECTIVES**

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## EXECUTIVE SUMMARY

The Cominco Ltd. Polaris Mine Operation located on Little Cornwallis Island (LCI) in Nunavut Territory is scheduled for decommissioning in 2002. The objective of the ecological and human health risk assessment was to evaluate the chemicals identified at the site that may be of concern to human health and/or the environment and to use this information to derive site-specific soil quality remediation objectives (SQRO). In deriving site-specific SQROs, the risk assessment:

- Estimated a daily exposure to the chemical of potential concern (*i.e.*, metals) that would result in no measurable health effects on human individuals or resident wildlife populations; and in turn,
- Estimated a metal soil concentration that, when ingested/inhaled/contacted, would not be expected to result in any adverse effects for either humans or wildlife populations.

To ensure that the risk assessment incorporated accurate information related to the potential wildlife at risk, as well as the expected type, and level, of Inuit activity on the mine site once Cominco shut its operation down, members of the Inuit communities of Resolute Bay and Grise Fiord were consulted.

### Human Health Risk Assessment

The main objectives of the human health risk assessment were to identify:

- 1) chemicals of potential concern;
- 2) sensitive human receptors potentially at risk;
- 3) important exposure pathways;
- 4) potential risks associated with terrestrial areas on the site for the identified future land uses (*i.e.*, once the mine has been decommissioned);
- 5) estimate site-specific SQROs that can be used to guide the risk management of the chemicals of concern identified at the actively mined area.

In order to accurately depict the type of land use in the risk assessment, a questionnaire was prepared for distribution to the potentially-impacted Inuit communities of Resolute Bay and Grise Fiord. The questionnaire was designed to capture the expected land use of the LCI site following the decommissioning of the mine. Some of the key land-use issues that arose from the consultation process included:

- When visiting LCI, most individuals are not expected to spend more than 3 days on the island;
- Respondents rarely eat wild game or fish harvested from LCI;
- Individuals were not expected to visit LCI more than three times per season;

- Community members of all age groups could visit LCI;
- Most people will miss the Cominco facilities once they have been removed (*i.e.*, opportunity to collect food, gas, *etc.*);
- LCI is generally viewed as a transitory stop while en route to more suitable hunting and fishing areas (*e.g.*, Bathurst Island)

Screening of the contaminant concentrations in soils on the actively mined area of Little Cornwallis Island identified three metals of potential human health concern. These included:

- Cadmium
- Lead
- Zinc

At various sampling locations, soil concentrations of these metals exceeded soil guidelines designed to be protective of human health.

Results of the risk assessment identified exceedance of the targeted/acceptable risk level for both lead and zinc.

#### Ecological Risk Assessment

An ecological risk assessment (ERA) was conducted to determine the potential risks to ecological receptors as a result of on-site contamination of soils with heavy metals. Given the nature of the chemical contamination, the ERA focused on potential risks to terrestrial wildlife species.

The objectives of the ERA were:

- 1) to determine whether the on-site soil contamination at the Polaris Mine site would likely pose risks to indigenous terrestrial wildlife species.
- 2) to identify areas within the site which may potentially require remediation based on potential ecological risks and to subsequently determine soil quality remediation objectives to guide the reclamation plans at the Polaris Mine site.

Screening of the soil concentrations at the Polaris Mine site identified four metals of potential ecological concern, including:

- Cadmium
- Copper
- Lead
- Zinc

The results of the ecological risk assessment identified elevated risks for the following wildlife receptors exposed to lead:

- Lemming
- Arctic hare
- Caribou
- Arctic fox

It was concluded that predicted ecological risks and potential deleterious effects would be confined to the individual organism level. This means that the metal contamination of the surface soils surrounding Cominco's Polaris mine was not expected to impact the survivability or sustainability of any of the evaluated terrestrial wildlife populations (*i.e.*, no measurable effects on the ecological populations).

#### Soil Quality Remediation Objectives

To ensure the overall environmental well-being of Little Cornwallis Island, soil quality remediation objectives were derived. The SQROs were based on the most sensitive receptor spending time on-site, which in the case of lead and zinc were found to be Inuit children.

The surface soil concentrations considered safe for the most sensitive receptors were estimated to be:

- Lead                      2,000 mg/kg
- Zinc                        10,000 mg/kg

Lead and zinc concentrations in surficial soil that are equal to or below these recommended objectives were not expected to cause adverse health effects to Inuit children visiting the mine site under the conditions identified through the consultation process.

## **HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT OF COMINCO'S POLARIS MINE ON LITTLE CORNWALLIS ISLAND: DERIVATION OF SOIL QUALITY REMEDIATION OBJECTIVES**

### **1.0 INTRODUCTION**

The Cominco Ltd. Polaris Mine Operation located on Little Cornwallis Island (LCI) in Nunavut Territory is scheduled for decommissioning in 2002. Heavy metals such as lead and zinc were released into the environment as a result of dusts produced during mining and milling operations. The objective of this project was to conduct an assessment of these chemicals identified at the site that may be of concern to human health and/or the environment and to use this information to derive site-specific soil quality remediation objectives (SQRO). In deriving site-specific SQROs, the risk assessment:

- Estimated a daily exposure to the chemical of potential concern (*i.e.*, metals) that would result in no measurable health effects on human individuals or resident wildlife populations; and in turn,
- Estimated a metal soil concentration that, when ingested/inhaled/contacted, would not be expected to result in any adverse effects for either humans or wildlife populations.

This report provides the methods and results of the human health and ecological risk assessment for the terrestrial portion of this site and the derivation of site-specific soil quality remediation objectives.

### **1.1 Objectives of the Study**

Based on information obtained through a review of site investigation reports by Gartner Lee Limited, B.C. Research and discussions among project team members, the following objectives were set:

- 1) Conduct a screening level human health and ecological risk assessment of the site in its future land use option (*i.e.*, following closure of the Cominco mine);
- 2) Determine if further data are required for additional risk assessment activities;
- 3) Using the risk assessment results as the basis, derive site-specific soil quality remediation objectives (SQROs)
- 4) Recommend further technical studies and a communication strategy for the Inuit communities which are potentially affected by this site (*e.g.*, Resolute Bay, Grise Fiord), if necessary.

As Cominco prepares for the decommissioning of its Polaris Mine, careful consideration was given to the remediation of all areas of potential environmental concern. Evaluation of remediation strategies resulted in the derivation of site-specific soil quality remediation objectives that adequately protect the area's environmental health. SQROs were determined

within the permitted framework as governed by the Canadian Environmental Quality Guidelines (CCME, 1999).

The framework outlined in the National Contaminated Sites Soil Protocol provides the opportunity to move from generic soil guidelines to site-specific remediation objectives, which in turn “allows the proponent to ensure that the assumptions used in the soil protocol apply to the site-specific conditions” (CCME, 1999).

The unique nature of the Polaris Mine site suggests that the application of generic soil quality guidelines is not appropriate for remediative purposes. The scarcity of vegetation in the area results in a limited number of wildlife. The “barren and rugged” terrain of LCI (Graham, 1982) precludes the regular use of the area by both wildlife and human receptors. Further, the atypical characteristics and unusual scenarios indicative of the high Arctic location of the Mine site necessitated the development of site-specific SQROs.

## **2.0 PREVIOUS WORK AND OVERALL DESCRIPTION OF THE MINE SITE**

### **2.1 Relevant Studies**

In an attempt to accurately evaluate potential environmental health risks associated with the Cominco Mine Site (*aka* mine), Cantox Environmental Inc. (Cantox) incorporated all relevant site-specific information into the current risk assessment. This information was gathered from the following reports:

- BC Research 1975. Environmental Study of Polaris Mine, Little Cornwallis Island. Submitted to Cominco Ltd., Project No. 1642. Prepared by Division of Applied Biology, BC Research.
- Bryant Environmental Consultants Ltd. 1997. Environmental Evaluation Report: The Eclipse Report (Draft). Vol I and II. Yellowknife, Northwest Territories.
- GLL (Gartner Lee Ltd.) 1999. Cominco Ltd. – Polaris Operations (Draft). Environmental Assessment Report. November 1999. GL 99-902.
- GLL (Gartner Lee Ltd.) 2000. Cominco Ltd. – Polaris Operations (Draft). 2000 Contaminant Assessment Report. December 2000. GL 20-930.
- Graham, K.A. 1982. Eastern Arctic Study, Case Study Series: The Development of the Polaris Mine. Joint project of the Institute of Local Government and the Centre for Resource Studies. Queen’s University, Kingston, Ontario.
- LaVigne, M. 1980. Potential Socio-Economic Impacts of the Polaris Mine Project. Prepared by Outcrop Ltd. (Yellowknife) in association with DPA Consulting Ltd. (Vancouver).

Gartner Lee Limited conducted Environmental Site Assessments (ESA) of Cominco Ltd.’s Polaris Mine on Little Cornwallis Island in 1999 and 2000. The objective of the ESAs was to identify and assess the significance of potential environmental issues at the Polaris mine site and receiving environment in order to provide direction for future remedial planning (GLL, 1999). For the current risk assessment, the data and field information obtained for the ESAs served as



the primary sources of information relating to the extent of the mining area's metal contamination.

In an effort to determine the mine's impact on historical land use activities such as hunting and trapping, LaVigne (1980) examined the prime hunting areas of the Resolute Inuit. Based on interviews with 20 Resolute Inuit, the study concluded that "the mine location [would] have little impact on the hunting/trapping harvests of the Resolute Inuit, since Little Cornwallis Island [was considered to be] a low priority hunting area" (LaVigne, 1980).

Following interviews with four Inuit hunters from Resolute, Bryant Environmental Consultants (1997) noted that although hunting on LCI did continue after the mine began operating, the reduced number of caribou (as a result of severe weather) led to less frequent hunting trips to LCI.

Surveys of the region identified 36 archaeologically significant sites on LCI north and east of the mine site. None of the sites that were deemed significant were located within Cominco's surface leases at the Polaris Mine.

To ensure that the risk assessment incorporated accurate information related to the potential wildlife at risk, as well as the expected type, and level, of Inuit activity on the mine site once Cominco shut its operation down, members of the Inuit communities of Resolute Bay and Grise Fiord were consulted. Results of this consultation are presented in section 3.2.2.

Results from Bryant Environmental's mammal survey on LCI (1997), along with BC Research's environmental baseline study (1975), identified some key issues relating to observations of the "presence, abundance, and population fluctuations" (GLL 2000) of the primary species hunted by regional Inuit (*e.g.*, caribou and muskox):

- Vegetation surveys of the mine site prior to mine construction indicated that the mine is located primarily on "bare" habitat which has relatively low biological sensitivity and low vulnerability to mechanical disturbance. Suitable vegetation for caribou and muskox feeding are found north and east of the current mine site.
- Caribou and muskox populations have fluctuated over the years. Severe weather in 1973/1974 decimated the caribou and the Inuit imposed a hunting moratorium until the mid 1980's. Population studies have shown the caribou resident on LCI to range from 11 in 1975 to 7 in 1987. A study in 1997 found no caribou, but some signs of recent usage. Caribou numbers throughout the Arctic islands have again been reduced considerably in the last few years due to severe weather conditions in the early winter months of two successive years (GLL 2000).
- The muskox population seems to be declining on LCI. In 1974, the estimated population was 20, while only three were reported in 1975. No muskox were reported in the 1997 survey. Like caribou, muskox are susceptible to extreme weather conditions.

- Lemming and Arctic fox are reasonably abundant but populations cycle. Arctic hare are present in small numbers but are found largely in the vegetated areas immediately north and east of the mine. Wolves have been seen in the area but none were present during the surveys in 1975 and 1997.
- Polar bears are culturally and economically significant to the Inuit and are common in the LCI area. Polar bear sightings at the mine have been as high as 250 in a winter season. The mine reports less sightings since fencing the landfill site in 1999.

## **2.2 Little Cornwallis Island – Environmental Setting**

### **2.2.1 Site Description**

The terrain on LCI consists of gently rolling low-relief hills and plains rising out of the ocean (GLL, 1999). Cominco Ltd.'s Polaris Mine is located at the southern tip of Little Cornwallis Island in Canada's High Arctic (75°N, 97°W), approximately 100 km northwest of the community of Resolute. The mine site is bordered by Garrow and Cominco Bays to the east, Crozier Strait to the southwest and North Bay to the north (see figure 1). Polaris is an underground zinc-lead mining operation that occupies approximately 952 hectares of land under surface leases from the Government of Canada (GLL, 1999).

The mine site was first explored in the 1960's, but full production did not begin until late 1981 (GLL, 1999). In 1998, production was about 1,040,000 tonnes of ore at an approximate grade of 4.0% lead and 14.0% zinc, producing 48,000 tonnes of lead concentrate and 226,000 tonnes of zinc concentrate (GLL, 1999). Mining and milling operations at the mine site produced dusts from the ore and concentrates which resulted in the release of lead and zinc into the receiving environment.

Several facilities have been constructed on the mine site including: huts, trailer accommodation, an accommodation building, concrete storage building, underground crusher and conveyor system, a barge containing the process plant and related facilities, above ground fuel storage tanks, fuel conditioning building, generator building and the Cemented Rock Fill plant (GLL, 1999).

The Polaris Mine site is located approximately 950 km north of the Arctic Circle in the Northern Arctic Ecozone, an area that incorporates the coldest and driest landscapes in Canada (Gartner Lee Limited, 1999). Due to the extremely cold temperatures, precipitation levels are low, resulting in low levels of absolute humidity and available moisture.

Cominco's mining activities have largely remained isolated to the bare, coarse-textured areas of the southwest corner of LCI. BC Research's (1975) evaluation of the mine's potential environmental impact identified the proposed active mining areas to be of moderate to low environmental sensitivity. The majority of the mine facilities are located within the barren, southwest land area (see figure 2 and 3 in GLL 2000).

### **2.2.2        *Soils***

Soils in the Polaris Mine area are described as polar desert in character, with poorly developed horizons (GLL, 1999). Barren, gravel type surface material predominates the Polaris Mine site. Soil pHs have been reported as alkaline (pH 8), confirming the calcareous nature of the parent materials. Finer textured materials are found in natural depressions where vegetated meadow type zones have formed in poorly drained sand to clay materials (GLL, 1999).

Measured soil concentrations of the metals of potential concern are outlined in Section 3.2.1.4.

### **2.2.3        *Local Ecology***

The vegetation throughout the mine site area is classified as “arctic tundra” consisting of a variety of moss and lichen species but few vascular plants. Plants identified in baseline studies included:

- Grasses
- Lichen
- Algae
- Mosses
- Willow
- Saxifrage
- Arctic poppies
- Dwarf draba.

Wildlife observed on LCI (also for environmental baseline studies, BC Research 1975) included:

- Herbivores: Peary Caribou, Muskox, Lemmings, and Arctic Hares;
- Carnivores: Arctic Fox; Arctic Wolf, and Polar Bear;
- Migratory birds: Red-throated Loons, Rock Ptarmigan, Eider Duck, Parasitic and Long-tailed Jaegers, Glaucus Gull, Arctic Tern, and Snowy Owl.

The vegetation of LCI is classified as “Arctic Tundra”. Due to the harsh climate, high winds and shallow soils, vegetation forms are typically dwarfed, low-lying and grow in clusters or as a dense mat. There is considerable variation in the moss and lichen flora, but few species of vascular plants. Vegetative cover tends to be greater on wetter sites in coastal lowlands, along streams and rivers and in sheltered valleys.

The area of the active mine site is considered to have low biological sensitivity due to the scarcity of vegetation and the associated low potential for wildlife use. The BC Research study (1975) found that vegetated areas, represented predominantly by the lichen-moss-algae type, were located no closer than 500 metres from what is now the active mine site area.

Three major lakes are located within the mine site area: Frustration, Lois and Garrow Lakes. Frustration Lake is used as the potable and process water source for the mine site and Garrow Lake, a non-mixing, saline (meromictic) lake, is used for the disposal of tailings (GLL, 1999).

### **3.0 HUMAN HEALTH RISK ASSESSMENT**

This section of the report presents the objectives, methods and results of the screening level human health risk assessment of chemicals identified at the Polaris mine site.

#### **3.1 Objectives of the Human Health Risk Assessment**

The main objectives of the human health risk assessment were to identify:

- chemicals of potential concern;
- sensitive human receptors potentially at risk;
- important exposure pathways;
- potential risks associated with terrestrial areas on the site for the identified future land uses (*i.e.*, once the mine has been decommissioned);
- estimate site-specific SQROs that can be used to guide the risk management of the chemicals of concern identified at the actively mined area.

#### **3.2 Human Health Risk Assessment Methodology**

For the human health risk assessment, potential risks to human receptors were estimated based on existing chemical information available for the site (*i.e.*, 1999 and 2000 sampling data). The methods used to predict the possible adverse effects to humans from exposure to chemicals originating from the site were based on the fundamental dose-response principle of toxicology. That is, the response of a receptor to chemicals is determined by the toxic potency of the chemical and the rate or degree of exposure (*i.e.* the dose) that occurs to that chemical. In addition, the potential response to a chemical increases in proportion to the rate of exposure which occurs beyond the chemical-specific exposure threshold. Below this exposure threshold, no measurable adverse effects from the chemical would be expected to occur. The rate of exposure is dependent on such factors as receptor characteristics, physical/chemical properties of the chemicals and the chemical concentrations in the environment.

As the Polaris site is located in the high Arctic, a unique approach to risk assessment, typically unnecessary for more southerly sites, was applied to account for special concerns related to an Arctic ecosystem and the way of life of the Inuit people from affected communities. This unique approach is described in subsequent sections of this report.

Risk assessment procedures used in the current assessment were those endorsed by such regulatory agencies as Health Canada, Environment Canada and the US Environmental Protection Agency (EPA). Briefly, the four basic steps of human health risk assessment are:

- 1) Problem Formulation: identification of the land use scenarios, human receptors, exposure pathways and chemicals of concern;

- 2) Exposure Assessment: quantification of the estimated rate of exposure to chemicals, from all assessed exposure pathways and routes;
- 3) Toxicity Assessment: the identification and assessment of potential hazards and the recommendation of upper limits of exposure (*i.e.*, maximum exposures that could occur without measurable adverse health effects) for the chemicals of concern; and,
- 4) Risk Characterization: determination of potential human health risks based on comparison of the estimated exposures to the exposure limits for the chemicals of potential concern.

Each of these steps of human health risk assessment, with respect to the current evaluation, are described in the following sections.

### ***3.2.1 Problem Formulation***

Problem formulation consisted of identification of the land use scenarios, receptors, exposure pathways and chemicals of concern to be evaluated in the human health risk assessment of the Polaris mine site.

#### ***3.2.1.1 Land Use Scenarios***

There are four general categories of land use that the various areas of the Polaris site could ultimately be used for:

- Residential;
- Parkland;
- Commercial; and
- Industrial.

However, there is also a fifth type of land use that is unique to the high Arctic. This has been termed “Historical Land Use” and consists of Inuit families residing on the land in tents for brief periods. During these periods, the Inuit are assumed to engage in hunting, fishing and gathering activities. This type of land use is of primary concern to the residents of communities surrounding the Polaris mine site, and has the potential to result in the greatest exposure of these residents to chemicals in the site soil and local biota. Therefore, the human health risk assessment focused on this land use scenario only. All other land use scenarios were considered improbable for the Polaris mine site.

In order to accurately depict the type of land use in the risk assessment, a questionnaire was prepared for distribution to the potentially-impacted Inuit communities of Resolute Bay and Grise Fiord. The questionnaire was designed to capture the expected land use of the LCI site following the decommissioning of the mine. A summary of the responses as well as the individual questionnaire results can be found in Appendix C. Nine community members of

Resolute Bay participated in the survey while five individuals participated from Grise Fiord. Some of the key land-use issues that arose from the consultation process include:

- When visiting LCI, most individuals are not expected to spend more than 3 days on the island;
- Respondents rarely eat wild game or fish harvested from LCI;
- Individuals were not expected to visit LCI more than three times per season;
- Community members of all age groups could visit LCI;
- Most people will miss the Cominco facilities once they have been removed (*i.e.*, opportunity to collect food, gas, *etc.*);
- LCI is generally viewed as a transitory stop while en route to more suitable hunting and fishing areas (*e.g.*, Bathurst Island)

#### 3.2.1.2 *Receptor Selection*

Considering that Inuit of all ages are expected to visit LCI following the mine closure, the most sensitive potential receptor is considered to be a female toddler. Young children exhibit strong hand-to-mouth activity and are expected to ingest greater amounts of soil than older age groups. Further, due to the lighter body weights, children would be exposed to higher doses of on-site metals on a “per unit body weight” basis.

#### 3.2.1.3 *Exposure Pathway Selection*

Results from the community consultation efforts indicate that an Inuit family may typically spend up to 3 days per visit on LCI. Potential exposure times of this duration are indicative of a short-term exposure scenario, as outlined by US EPA (1999, page 7).

For this land use scenario, exposure to chemicals in surface soil would occur via a variety of direct soil exposure pathways (*i.e.*, dermal exposure to soil, incidental ingestion of soil, and inhalation of dusts derived from soil).

Due to the typically brief exposure duration and the unlikelihood of receptors consuming either game or fish from LCI, these ingestion pathways were excluded from the assessment.

For an Inuit family assumed to camp at the site, the main sources of exposure to chemicals will be from surface soil (and associated pathways). It is recognized that aquatic (*i.e.*, marine) animals (*e.g.*, fish, seals, walrus, whales, *etc.*) could also represent potential sources of exposure; however, there are insufficient data available to estimate exposures from such potential sources at this time. However, the main contaminants of concern associated with the site are lead, zinc, cadmium and copper. Zinc and copper do not bioaccumulate in aquatic animals to any significant extent, while lead and cadmium only bioaccumulate to a very small degree. Due to the migratory nature of these animals, their exposures to these substances as a results of the Polaris Mine would be expected to be small and would not be expected to significantly impact existing tissue concentrations. Thus, the current human health risk assessment focuses only on terrestrial sources of human exposure to site chemicals (*i.e.*, soil).

Based on the intended land use of the site, the following exposure pathways were evaluated:

- inhalation of metals in re-suspended dusts from on-site soils;
- ingestion of metals in surface soils and airborne dusts;
- dermal contact with metals in soil and airborne dusts.

#### *3.2.1.4 Chemical Selection*

Initial screening of contaminant levels in soil with respect to potential environmental impacts was conducted using the appropriate guidelines or criteria established by Canadian regulatory agencies (*e.g.*, CCME, B.C. MELP). Only those metals which were detected in surface soil were considered. Maximum measured surface soil concentrations were employed in the human health risk assessment due to the nature of the exposure scenario (see section 3.2.2).

The soil quality criteria for a residential/parkland use, obtained from the CCME (1991, 1997), are considered protective of both human health and the environment. A relatively high level of conservatism is inherent within the values in order to ensure a safe environment for all terrestrial species.

It is important to note that an exceedance of soil guidelines does not automatically imply that the environment is not healthy or capable of sustaining a viable, active ecosystem. As mentioned, the inherent conservatism of the guidelines generally biases the assessment towards overestimating the potential risks. Although contaminant concentrations at levels below the guidelines indicates a negligible risk, where an exceedance is noted, further consideration of site specific issues must be considered before rendering a verdict. It is not uncommon for natural background levels of metals to exceed guidelines with no ill effects on the ecosystem.

Extensive environmental sampling was conducted as part of the environmental site assessment of the Polaris Mine Operation completed by Gartner Lee Limited (1999, 2000). The existing data was compiled to aid in the chemical selection for the screening level human health risk assessment using the methodology described below.

If chemicals exceeded regulatory guidelines for soil (for residential/parkland use), they were compared to more specific guidelines designed to specifically protect for human health. Chemicals which then exceeded these guidelines were in turn carried forward to the quantitative human health risk assessment.

A comparison of maximum measured site soil metal concentrations to regulatory soil quality criteria is presented in Table 1. To ensure that no chemicals of potential concern were excluded from the quantitative assessment, measured concentrations included samples taken at depth.



**Table 1. Maximum measured metal concentrations in soil sampled from the Polaris Mine site**

Chemical	Concentration (mg/kg)	Guideline <sup>1</sup> (mg/kg)	Location
Antimony	<DL	20 <sup>2</sup>	--
Arsenic	<DL	12	--
Barium	388	500	Site J (0 – 0.01m, 2000 sample)
Beryllium	1.2	4 <sup>2</sup>	TP140 (0.5 – 0.8m, 2000 sample)
Cadmium	318	10	Foldaway snow dump FSD-2 (sfc grab, 2000 sample)
Chromium	44	64	TP77 (0.1 – 0.3m raised bore, 1999 sample)
Cobalt	20	50 <sup>2</sup>	TP140 (0.5 – 0.8m, 2000 sample)
Copper	196	63	TP40 (0.1 – 0.3m concentrate building, 1999 sample)
Lead	16,500	140	TP80 (0.3 – 0.6m outdoor oil storage, 1999 sample)
Molybdenum	<DL	10 <sup>2</sup>	--
Nickel	43	50	#59, 1401E,; 1267N (1999 sample) and TP140 (0.5 – 0.8m, 2000 sample)
Selenium	<DL	3 <sup>2</sup>	--
Silver	<DL	20 <sup>2</sup>	--
Tin	<DL	50 <sup>2</sup>	--
Zinc	139,000	200	Foldaway snow dump FSD-2 (sfc grab, 2000 sample)

Note: <DL indicates all measured soil concentrations were less than the laboratory detection limits for both the 1999 and 2000 sampling programs; shaded cells identify chemicals for which the measured concentrations exceeds the applicable guideline

<sup>1</sup> guidelines based on CCME 1999 Canadian Environmental Quality Guidelines for residential/parkland land use (unless otherwise noted)

<sup>2</sup> based on B.C. MELP generic numerical soil standard



Metals exceeding environmental soil standards/guidelines:

- Cadmium
- Copper
- Lead
- Zinc

Further examination indicated that the soil quality guideline for copper considered protective of human health is actually greater than the maximum measured soil concentration (CCME, 1999). The human health-based soil quality guideline for copper is 1,100 mg/kg. This guideline is based on the soil ingestion pathway.

Therefore, metals evaluated in greater detail as part of the quantitative human health risk assessment included:

- Cadmium
- Lead
- Zinc

### **3.2.2            *Exposure Assessment***

Exposure assessment involves the estimation of the amount of chemical received by human receptors per unit time (*i.e.*, the quantity of chemical and the rate at which that quantity is received). Exposure assessment was conducted for the chemicals, human receptors and exposure pathways identified to be of greatest concern during the problem formulation stage of the risk assessment. The rate of exposure to chemicals from the various environmental media is usually expressed in units of amount of chemical intake per body mass per time (*e.g.*, µg chemical/kg body weight/day).

For the quantitative human health risk assessment, the exposure assessment was conducted using deterministic or point estimate exposure analysis techniques. Deterministic techniques utilize single “reasonable worst-case” values for each parameter considered in the risk assessment. An alternative to deterministic analysis techniques is stochastic (or probabilistic) exposure analysis which utilizes probability density functions (or distributions) to characterize input parameters (*i.e.*, each input parameter is represented by a distribution of values, such as the distribution of body weight observed in a population of people). The main advantage of stochastic analysis is that it produces results that are believed to more realistically represent the characteristics that actually exist for the various risk determination parameters than the use of single worst-case or upper bound values that are used in deterministic analysis techniques. However, the deterministic approach is much less data-intensive than the stochastic approach and can serve as an efficient “first-cut” evaluation to determine whether or not further, more detailed assessment of risks is warranted.

Considering that the intended use of the closed mine site area is representative of a “short-term” exposure scenario, chemicals were evaluated using deterministic risk analysis techniques. The

deterministic approach considered a “worst-case” scenario which in turn ensured that potential health risks to future users of the site would not be underestimated. Cominco Ltd. is committed to remediating any soils considered to harbour metal concentrations that were identified to pose greater than negligible health risks. Soil quality remediation objectives or soil concentrations that would pose no unacceptable health risks were subsequently calculated employing deterministic techniques.

The exposure estimate considers how people might be exposed to metal-contaminated surface soils. Various environmental and receptor parameters are incorporated into the calculation of total chemical exposure. Environmental parameters were largely based on the environmental concentrations of the various chemicals already measured at the site and reported in the site assessment reports (GLL, 1999 & 2000). Parameters such as physical constants and bioavailability were acquired from literature sources and/or from CANTOX ENVIRONMENTAL’s in-house database.

The degree of environmental chemical exposure depends on the interactions of a number of parameters, including:

- the various physical, chemical and biological factors that determine the ability of the receptor to take the chemicals into the body from the exposure pathways (e.g., bioavailability of the chemicals from particles and soil);
- the physical/chemical characteristics of the chemical which determine the interaction and behaviour of a chemical with its surrounding environment (e.g., tendency to bind to particles);
- the concentrations of chemicals in various compartments of the environment (e.g., surface soil) impacted by activities from the site as well as the normal ambient, or background concentrations<sup>1</sup>;
- the behavioural and lifestyle characteristics of the human receptors in the environment that determine the actual exposures through interactions of the receptors with the various pathways (e.g., respiration rate, soils/dusts ingested); and,
- the various exposure pathways for the transfer of the chemicals from the different environmental compartments to the organisms such as humans (e.g., inhalation of air, soils and dusts; ingestion of soils/dusts; dermal exposure to dust/soil).

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<sup>1</sup> Since the intended land use of the closed Cominco mine site is not representative of a chronic or long-term exposure scenario, it was deemed inappropriate to include background concentrations in the total short-term exposure analysis.

The contribution of each exposure route (i.e., ingestion, inhalation, dermal) to total exposure is based on the individual characteristics of each of the human receptors (e.g., soil/dust consumption, breathing rate, body weight, time spent on-site, *etc.*). Receptor characteristics were based on published literature sources (e.g., Health Canada, 1999; US EPA, 1997, O'Connor, 1997; *etc.*) but primarily through correspondence with local people. Receptor characteristics were selected to reflect local behaviours and life style activities where possible, however, data for individuals from other regions were used when local data were insufficient. Depending on the pathway of exposure, different receptors may have a potentially greater relative exposure (e.g., children consuming more chemically-impacted soil/dust than members of older age-groups). Thus, significant age or sex specific differences in receptor characteristics that affect chemical exposures are also taken into account.

All mathematical formulae, input parameters and assumptions used in the exposure assessment are provided in Appendix B.

### 3.2.2.1 *Bioavailability*

The bioavailability of a chemical defines that portion of the exposure concentration of chemical (e.g., the quantity inhaled or ingested) that enters the body and is assumed to be available to produce toxic effects in target tissues. In other words, the toxic effects of a chemical are determined by the actual concentration of the chemical at the site of action rather than the amount that gains access to the body. In the majority of cases, however, the available data are inadequate to define the concentrations of chemicals at their site of action. Therefore, the bioavailable dose is used as a first approximation of the amount of the chemical at its site of action.

The bioavailability of a chemical is dependent on the chemical form as well as the tissue/animal species with which the chemical interacts. Thus, when applying exposure limits derived from animal studies to estimate potential risks following exposures of humans, it is necessary to consider the bioavailability of each chemical to the test animal, and its bioavailability to the human receptors. This allows normalization of exposures with respect to the route and allows the bioavailable doses to humans to be compared with acceptable bioavailable doses determined from animal studies or human epidemiological data. For example, if the exposure limit for a particular chemical was established from animal studies where the chemical was injected into the trachea in a solution, but the exposure pathways considered was the inhalation of chemical adsorbed to airborne particles (dusts/soils) by humans, the exposure limit and exposure would be adjusted by the amount of chemical absorbed *via* tracheal injection in animals, and *via* dust inhalation in humans.

The bioavailability of chemicals following oral ingestion, inhalation, or dermal contact, was estimated from the published literature, where available. In situations where chemical specific data are lacking, it was assumed that the chemical is 100% bioavailable. This assumption would tend to over-estimate exposure rates and therefore over-estimate the potential health risks associated with the chemicals of concern.

### 3.2.3 Toxicity Assessment

Toxicity is the potential for a chemical or agent to produce any type of damage, permanent or temporary, to the structure or functioning of any part of the body. The toxicity of a chemical depends on the amount of chemical taken into the body (referred to as the “dose”) and the duration of exposure (*i.e.*, the length of time the person is exposed to the chemical). For every chemical, there is a specific dose and duration of exposure necessary to produce a toxic effect in humans (this is referred to as the “dose-response relationship” of a chemical). In the toxicity assessment, information relating to the dose-response relationships of each chemical is evaluated (usually from laboratory animal studies and studies of human exposure in the workplace) in order to determine the maximum dose of chemicals to which humans can be exposed that would be associated with a very low probability of experiencing adverse health effects. These toxicity estimates are called exposure limits and indicate an exposure that will not likely result in harmful effects.

There are two main types of dose-response relationships for chemicals:

- **Threshold Response Chemicals:** For some chemicals, there is a dose-response threshold below which no adverse effects would be expected to occur. This relationship is true for most chemicals which do not cause cancer by altering genetic material (*e.g.*, most metals). Thresholds are generally assumed for non-carcinogens because for these types of effects, it is generally believed that homeostatic, compensating, and adaptive mechanisms must be overcome before toxicity is manifested. Exposure limits derived for threshold-response chemicals are called reference doses (RfD), acceptable daily intakes (ADI) or permissible daily intakes (PDI) and are generally derived by regulatory agencies such as Health Canada and the U.S. Environmental Protection Agency (EPA).
- **Non-threshold Response Chemicals:** For some chemicals, there is no dose-response threshold. This means that any exposure greater than zero is assumed to cause some type of response, or damage. This relationship is typically used for chemicals which can cause cancer by damaging genetic material. Since, in theory, any exposure has the potential to cause damage, it is necessary to define an “acceptable” degree of risk associated with these types of exposures. This “acceptable” degree of risk is usually defined as a risk of one-in-one hundred thousand to one-in-one-million. These numbers can be better explained as the dose rate that may cause an increased risk of mortality related to cancer in one person out of one hundred thousand people, or one person out of one million people. The acceptable level of risk is a policy rather than a scientific decision, and is set by regulatory agencies, as opposed to risk assessors.

#### 3.2.3.1 Selection of Exposure Limits

Exposure limits for chemicals considered to have a threshold response are based on scientific information, professional judgement and technical review by a number of experienced scientists with expertise in a wide range of scientific disciplines. They are usually expressed as the quantity of chemical per unit body weight per unit time (*i.e.*,  $\mu\text{g chemical/kg body weight/day}$ ) when considering a chronic or long-term duration of exposure. Short-term exposure limits are

typically expressed as a concentration. This could be as the quantity of chemical per unit body weight or as the quantity of chemical per some measurement of environmental quantity (*e.g.*, air volume, soil/food mass, water volume).

Chronic exposure limits incorporate the notion of “per unit time” because these limits are considered to be protective of toxicological impact over a substantial fraction of the receptor’s life span (*i.e.*, exposure on an ongoing basis). Short-term limits ensure that exposure to that particular chemical dose for a brief span of time (*i.e.*, exposure that is not ongoing) will not result in measurable toxicological impact.

The exposure limits are derived based on the most sensitive endpoints in individuals (*e.g.*, organ damage, neurological effects, reproductive effects, *etc.*), and are typically termed reference doses (RfD) or tolerable daily intakes (TDI), which indicate concentrations of chemicals that individuals can be exposed to on a daily basis without the occurrence of adverse health effects. Large safety or uncertainty factors (*i.e.*, 100-fold or greater) are often used in the estimation of the RfD or TDI for threshold-type chemicals. Thus, exceedance of the exposure limit does not necessarily mean that adverse effects will occur; rather, it means that the safety factor beyond the no-effect exposure is somewhat reduced. Usually, exposure rates less than the RfD or TDI are not likely to be associated with adverse health effects, and are therefore, less likely to be of concern. As the frequency or magnitude of exposures exceeding the RfD or TDI increase, the probability of adverse health effects in a human population increases. However, it should not be categorically concluded that all exposures below the RfD or TDI will be unlikely to result in adverse health effects, and that all doses above the RfD or TDI are likely to result in adverse health effects.

The response of the body to chemicals depends in part, on the quantity of the chemicals that enter the tissues and cells. Only a fraction of the chemicals ingested, or inhaled, or in contact with the skin are actually absorbed into the body. The fraction of the exposure received that is absorbed by the body is known as the bioavailable fraction.

The bioavailability of a chemical is dependent on the chemical form, the environmental medium, as well as the tissue/organ with which the chemical interacts. Thus, when applying exposure limits, it is necessary to consider the bioavailability of each chemical in the particular study from which the exposure limit is derived to obtain reasonable estimates of the quantity of the chemical entering the body. This allows for the normalization of exposures with respect to exposure route, and comparison of the bioavailable doses to humans with the exposure limits determined from animal studies or human epidemiological data.

The following hierarchical approach was used to identify exposure limits for use in the current risk assessment:

- Canadian regulatory agencies (*e.g.*, CCME, Health Canada, Environment Canada);
- Another recognized regulatory authority or expert organization [*e.g.*, US EPA, World Health Organization, American Conference of Governmental Industrial Hygienists (ACGIH)]; and,

Given the acute (short duration and infrequent) rather than chronic (continuous exposure), nature of the intended land use, acute exposure limits were selected *when possible*. Where acute limits were not available, chronic exposure limits were used. The use of chronic exposure limits for acute exposure scenarios is a conservative approach that likely overestimates potential risks.

Table 2 provides a summary of the exposure limits used in the current screening level human health risk assessment. These exposure limits are described in further detail in Appendix A .

**Table 2. Acute exposure limits for the metals of potential concern at Cominco's mine site**

Chemical	Exposure Limit	Potential Effects <sup>1</sup>	Reference
Cadmium	4.3 µg/kg <sup>2</sup>	GI irritation and vomiting in children	Calabrese <i>et al.</i> 1997
Lead	20 µg/dL (short-term "benchmark" blood level)	GI irritation, irritability, lethargy, tremors	CDC 1991 Health Canada 1994
Zinc	300 µg/kg <sup>3</sup>	GI distress and diarrhoea	Health Canada 1996

<sup>1</sup> toxic effects are *possible* at these levels but not certain

<sup>2</sup> ten-fold safety factor applied to acute dose to protect for most-sensitive sub-groups

<sup>3</sup> due to a paucity of data, limit is based on chronic exposure

Although Table 2 identifies toxic impacts associated with exposure to the metals, it does not mean that receptors exposed to the stated levels would automatically experience these adverse effects. Sensitivity to chemical exposure varies substantially within a population. It should be stressed that these effects are reversible and would therefore not be expected to continue beyond the duration of on-site exposure.

### 3.2.4 Risk Characterization

Risk characterization is the quantification and evaluation of the estimated risks from exposure to chemicals from the site. Risk characterization involves the estimation of risks associated with site-related exposure to chemicals of concern by comparisons between the estimated exposure and the toxicity estimate (*i.e.*, exposure limit).

Risk estimates were expressed as Hazard Quotients (HQs) which were determined according to the following formula:

$$\text{HQ} = \frac{\text{EXPOSURE ESTIMATE } (\mu\text{g/kg body weight})}{\text{EXPOSURE LIMIT } (\mu\text{g/kg body weight})}$$

#### 3.2.4.1 *Interpretation of Hazard Quotients*

Once hazard quotients have been determined, they are compared to a benchmark indicator of safety, also called the critical hazard quotient. In general, if the total chemical exposure from all pathways is equal to or less than the exposure limit, then the HQ would be 1.0 or less, and no adverse health effects would be expected. Therefore, the benchmark of safety would be 1.0, assuming that one has estimates of exposure from all *relevant* exposure pathways.

For threshold chemicals, the exposure limits represent the level of total exposure which would not result in adverse health effects, regardless of the source or pathways of exposure. As most risk assessments do not account for all possible sources of contamination and potential exposure pathways, the selection of a critical HQ value of 1.0 may not fully safeguard receptors in a chronic exposure scenario. In an attempt to address this issue, some regulatory agencies (*e.g.*, OMOE, CCME, Health Canada) recommend that 20% of total exposure be apportioned to any one source or pathway. However, a critical HQ value of 1.0 is considered appropriate for evaluating short-term or acute exposure scenarios.

Therefore, if threshold chemicals are determined to have HQ values less than 1.0, exposure levels are considered to be less than the exposure limit, and no adverse health effects would be expected to occur in the receptors and scenarios evaluated in the risk assessment. If HQ values are greater than 1.0, the estimated exposure is considered to exceed the exposure limit, indicating the potential for adverse effects in sensitive individuals.

It should be noted that HQ values greater than 1.0 do not necessarily indicate a risk of adverse health effects. Rather, for those chemicals which exceed the critical HQ, or benchmark of safety, assumptions and data used in the risk assessment would need to be re-examined prior to making final conclusions on potential risks.

Based on the rationale provided above, the following criteria were used to evaluate risk estimates:

- For on-site metals with total Hazard Quotient values greater than 1.0, risk management options should be considered.
- Metals with total Hazard Quotient values less than 1.0 could be excluded from future risk management plans or future risk assessment.



### **3.2.5            *Limitations, Uncertainty and Conservative Assumptions Used in the Human Health Risk Assessment***

In any risk assessment, the findings are based on available data from the specific study area, and the scientific literature, in conjunction with a number of assumptions. Every effort is made to ensure these assumptions and data adequately represent conditions at the site. However, data are often limited, resulting in uncertainty in the assessment. Where uncertainty exists, assumptions are made, and data are selected so as to err on the conservative side. Some key sources of uncertainty associated with the current assessment included:

- The community consultation efforts indicated that the members of the affected Inuit communities would use the site for only a few days per season (*i.e.*, not more than 3 days per visit).
- Results of the community consultation also suggested that Inuit visiting LCI would be more likely to camp away from the mine site (*i.e.*, northern part of LCI).
- It was assumed that humans would spend their time in contact with soil at the maximum measured concentrations on the site.
- It was assumed that Inuit children visiting the site would exhibit high soil ingestion tendencies (*i.e.*, the upper end of the soil ingestion range).
- It was conservatively assumed that the bioavailability related to ingesting soil was the same as the oral bioavailabilities reported for ingestion studies. Typically, metals will sorb strongly to soil and hence their overall bioavailabilities would be greatly reduced. However, to ensure that risks were not underestimated, this was not incorporated into the exposure calculations.

### **3.3                Results Of The Human Health Risk Assessment**

Based on the methodology described above, the risks to human health were estimated for the intended/future use of Cominco's mined area following decommissioning of the site. Results are presented for those chemicals considered to pose a potential threat to human health (*i.e.*, exceeded regulatory guidelines/standard, see section 3.2.1.4 for added detail). Potential health risks for the four metals of concern are presented in Table 3. Risks resulting from exposure to the on-site metal soil concentrations are discussed individually below. The derivation of these results (*i.e.*, the calculation of exposure and hazard quotients) are outlined in detail in Appendix B.



**Table 3. Human health risks associated with exposure to on-site metal surface soil concentrations following decommissioning of Cominco's Polaris mine site**

	Cadmium	Lead	Zinc
Soil conc. (µg/g) <sup>1</sup>	105.0	16,500.0	59,300.0
Exposure (µg/day) <sup>2</sup> :			
Oral	7.6	3,498.0	9,488.0
Dermal	1.8	12.4	1,855.9
Inhalation	0.0	1.4	4.1
Total	9.4	3,511.8	11,348.0
<b>Hazard Quotient</b>	<b>0.7</b>	<b>8.8</b>	<b>5.8</b>

Note: shaded cells indicate HQ's that exceed the critical value of 1.0

<sup>1</sup> soil concentrations are maximum measured surface soil concentration (GLL 1999 & 2000). These concentrations differ from those presented in Table 1 because they were taken strictly from surface soil samples.

<sup>2</sup> exposure based on an Inuit child weighing 16.4 kg

### 3.3.1 Cadmium

The hazard quotient for cadmium was estimated to be below the critical value of 1.0. This suggests that Inuit children exposed to cadmium-contaminated surface soils at the mine site on LCI should not be subject to unacceptable human health risks (*i.e.*, health risks are negligible).

### 3.3.2 Lead

The estimated hazard quotient for lead exceeded the critical value of 1.0. When Inuit children were assumed to be exposed to the maximum measured soil concentration, the health risk value would potentially be seven times greater than the target risk level.

Health risks may be overestimated as a result of several conservative assumptions, including:

- Children are exposed to the maximum measured surface soil concentration for the entire time they are on-site
- Soil bioavailability is equal to the typical lead oral bioavailability

This may suggest that the mine site does not necessarily harbour indisputable lead-related health risks. However, Cominco has indicated a commitment to remediate its actively mined area to the extent wherein no environmental health risks remain following closure of its mine. This is discussed in further detail in section 5.

### 3.3.3 Zinc

The hazard quotient for zinc also exceeded the critical risk value of 1.0. Inuit children exposed to zinc-contaminated surface soils (*i.e.*, maximum measured) for the intended land use (as indicated by the results of the community consultation) results in a health risk approximately six times greater than the target risk level.

Again, this does not necessarily mean that these calculated health risks are unacceptable, as several conservative assumptions were employed in the risk assessment. These are outlined in section 4.3 above and not excluding:

- The use of an exposure limit for zinc that is based on chronic (*i.e.*, continuous) exposure. These toxicological limits are often much less (*i.e.*, more stringent) than those for an acute or short-term exposure duration.

However, since the estimated risk exceeds the targeted level, risk management should ensure negligible health risks in the future (*i.e.*, no expected adverse impacts). This is discussed further in section 5.

## 3.4 Conclusions of the Human Health Risk Assessment

Initial screening of the contaminant concentrations in soils on, and surrounding, the actively mined area of Little Cornwallis Island identified four metals of potential environmental concern. These included:

- Cadmium
- Lead
- Zinc

At various sampling locations, soil concentrations of these metals exceeded soil guidelines designed to be protective of both ecological and human health.

The intended use of Cominco's Polaris site following closure of the mine was evaluated through active consultation with members of the communities of Resolute Bay and Grise Fiord. These efforts concluded that:

- Respondents do not intend to use the site for more than three days per visit. Visits will be infrequent and of short duration.
- Respondents consider LCI to be a transition area to more gainful hunting areas (*e.g.*, Bathurst Island).
- Visitors would tend to camp away from the mine site (*i.e.*, further north on LCI).

The intended land use was therefore best described as being representative of a short-term exposure scenario.

Results of the risk assessment identified exceedance of the targeted/acceptable risk level of 1.0 for both lead (8.8) and zinc (5.8). The exposure limit for zinc was based on chronic or continuous exposure, suggesting that risk associated with intake of zinc-contaminated soils was likely overestimated. In an attempt to ensure that health risks to Inuit children were not underestimated, the assessment did not consider the reduced availability of metals tightly sorbed to ingested soils.

The derivation of soil quality remediation objectives is outlined in section 5.

## **4.0 ECOLOGICAL RISK ASSESSMENT**

### **4.1 Introduction**

An ecological risk assessment (ERA) based on frameworks described by the Canadian Council of Ministers of the Environment and Environment Canada (Gaudet *et al.*, 1994; CCME, 1996) was conducted to identify the potential risks to Valued Ecosystem Components (VECs) found at the Polaris Mine Site. VECs are resources or environmental features that are important to human populations (*i.e.*, intrinsic, economic, and/or social value); have local, regional, provincial, national, and/or international profiles; or if altered from their existing status, will be important in evaluating the impacts of development and in focusing management or regulatory policy (CCME, 1996). Their significance to ecosystem health was also considered.

The ERA was conducted to determine the potential risks to ecological receptors as a result of on-site contamination of soils with heavy metals. Given the nature of the chemical contamination, the ERA focused on potential risks to terrestrial wildlife species.

### **4.2 Objectives of the Ecological Risk Assessment**

The objectives of the ERA were:

- 3) to determine whether the on-site soil contamination at the Polaris Mine site would likely pose risks to indigenous terrestrial wildlife species.
- 4) to identify areas within the site which may potentially require remediation based on potential ecological risks and to subsequently determine soil quality remediation objectives to guide the reclamation plans at the Polaris Mine site.

### **4.3 Ecological Risk Assessment Methodology**

The methods used to identify potential risks to terrestrial wildlife were based on ecological risk assessment procedures endorsed by regulatory agencies including the Canadian Council of Ministers of the Environment (CCME), British Columbia Ministry of Environment, Land and Parks (BC MELP), Alberta Environmental Protection (AEP), the Ontario Ministry of Environment and Energy (MOEE) and the United States Environmental Protection Agency (U.S.

EPA). The terrestrial wildlife risk assessment follows the framework of a preliminary quantitative ERA (Gaudet *et al.*, 1994; CCME, 1996). While the methods for conducting a human health and ecological risk assessment are similar, the terminology used for these two types of assessment differs slightly. In an ecological risk assessment, the problem formulation stage, as outlined in the following pages, is divided into the Planning Stage and Receptor Characterization Stage. To be consistent with the concurrent human health assessment, however, the terminology used in this chapter will be that used for human health assessments. The risk assessment frameworks include the following steps:

- 1) Problem Formulation: In this step, information was gathered to characterize the sites and to identify valued ecological receptors, potential exposure pathways, chemicals of concern, and appropriate assessment and measurement endpoints.
- 2) Exposure Assessment: In this step, site-specific rates of exposure to ecological receptors were estimated for the chemicals of concern.
- 3) Toxicity (or Hazard) Assessment: In this step, the toxicity of the chemicals of concern to the selected ecological receptors was determined through identification and assessment of dose-response information obtained from the scientific literature. Recommended upper limits of exposure (*i.e.*, maximum exposure rates below which significant ecological health risks are not expected to occur) were derived from this assessment.
- 4) Risk Characterization: In the final step, the potential risks to terrestrial wildlife were estimated by comparing the site-specific chemical exposures derived in the exposure assessment to the exposure limits established in the toxicity assessment.

At each stage, conservative, yet realistic, assumptions were used in an effort to ensure that predicted risks associated with the Polaris Mine site would not be underestimated.

#### **4.3.1 Problem Formulation**

The problem formulation phase of an ERA acts as an information gathering and interpretation stage. It is conducted to plan and focus the approach of the assessment on critical areas of concern for the sites being evaluated. Key tasks requiring evaluation within the problem formulation phase include the following:

- (i) Identification of Valued Ecosystem Components or VECs (*i.e.*, identify potential ecological receptors of chemical exposures).
- (ii) Exposure Pathway Analysis (*i.e.*, identify possible routes by which organisms may be exposed to chemicals at the site).

- (iii) Identification of Chemicals of Concern (*i.e.*, select chemicals which pose the greatest potential risk to receptors due to their toxic potency and measured concentrations); and,
- (iv) Identification of Assessment and Measurement Endpoints (*i.e.*, focus risk assessment on individual, population or ecosystem level effects and select practical measurements of those effects).

#### *4.3.1.1 Receptor Identification and Selection*

During the receptor identification stage of the ERA, key terrestrial wildlife receptors were identified following consideration of the indigenous species that are expected to utilize the habitat around the site. Emphasis was placed on those animals most likely to receive the greatest exposure to on-site contamination due to their habits and diet. In addition, information obtained from returned questionnaires by Resolute Bay and Grise Fiord residents was also used to identify and select ecological receptors.

Special consideration in ecological risk assessment is always given to any species which are classified as “endangered or threatened”. However, no such species were identified during field investigations (GLL 1999), and therefore, no endangered species were evaluated in the current ERA. As mentioned in Section 5.1, the ERA focused on potential risks to terrestrial wildlife species.

Due to the nature of the site (“insignificant” plant coverage, extreme latitude, *etc.*), toxicity to terrestrial plants and invertebrates were not incorporated into the risk assessment (and the subsequent calculation of the soil quality remediation objectives, see section 5 for further detail).

As outlined in the CCME protocol (1996):

Ideally, the selection of receptors should be compatible with, and reflect important characteristics of the ecosystem (*i.e.*, ecologically relevant) ... however, the selection of ecological receptors ... must focus on key receptors that maintain land use activities.

#### *Terrestrial Wildlife Receptors*

The selection of receptors is conducted so as to ensure assessment of the most sensitive or exposed species. This involves identifying representative species that have the greatest potential for exposure through various media, including the food chain, on-site habitation, as well as other behavioural and physiological factors that may result in increased exposures. Wildlife receptors were also selected based on sensitivity to chemical exposure and on availability of biological data describing their characteristics and behaviour.

For the current assessment, four representative species were selected as ecological receptors:

- Herbivores – lemming, arctic hare, caribou

- Carnivores – arctic fox.

Rationale for the selection of these species is presented in the following paragraphs.

*Collared lemmings (D. lentus)*

The lemming was selected as a wildlife receptor for this assessment as this species is relatively abundant in Canada's Arctic (the dominant rodent species). Several behavioural factors influence the extent of chemical exposure of the lemming which makes it a relatively sensitive receptor. Lemmings feed on ground level vegetation and seeds thus gaining access to contaminants that accumulate in vegetation. In addition, this species has a high potential for exposure to chemicals within surface soils, due to its burrowing and preening behaviour patterns. Lemmings are also quite territorial and migrate only out of necessity (a result of overcrowding). They are therefore quite sedentary with a limited home range, and as such, lemmings residing on-site would be likely to experience continual chemical exposures throughout their lives. Lemmings form the base of the terrestrial arctic food chain being prey for higher terrestrial wildlife species, and are thus important for the assessment of the effects of chemical exposures on higher trophic level organisms.

The foraging, defecation and burrowing behaviour of lemmings plays an integral role in the ecosystem of the high arctic. Their activity influences the productivity of vegetation by providing input of decomposed organic matter and dispersing soil nutrients. The lemming populations cycle and thereby influence the abundance of wildlife at higher trophic levels (*e.g.*, arctic fox) (GLL 2000).

Lemmings generally feed on willow, supplemented by grasses. The preferred vegetation units for lemming burrows include the willow and lichen-moss-algae habitats, and to a lesser extent the bare-lichen-moss-algae and wet meadow habitats (GLL 2000).

*Arctic hare (L. arcticus)*

The arctic hare was selected for assessment for several reasons. This animal, native to the arctic regions of Canada, is a herbivorous medium-sized mammal that is valued by humans (as indicated in the community consultation) and is fed upon by such predatory wildlife as the arctic food (*i.e.*, used in the evaluation of higher trophic level effects). The hare, like the lemming, does not migrate, has a relatively small home range and has a high potential for exposure *via* direct contact with soil.

*Peary caribou (R. tarandus)*

The caribou is representative of a large ungulate, found throughout Canada's arctic. Unlike the lemming and the arctic hare, caribou are migratory in nature. Migratory patterns of movement significantly decrease the exposure of caribou to any on-site contamination. However, during

calving, the herd may remain at a specific location for an extended period of time. In addition, these species were selected based on their value as a food source to both humans and other wildlife.

The caribou diet depends on seasonal availability, but consists largely of a combination of willow, herbs, mosses, and lichens. They prefer habitats where the vegetation is abundant and the ground conditions are dry. The population sizes of caribou herds fluctuate according to climatic conditions. BC Research's environmental baseline study (1975) noted only a sparse population of Peary caribou on LCI. Despite the reduced numbers on LCI, caribou were identified as valued ecosystem components in the community consultation and were included in the quantitative assessment for this reason.

#### *Arctic fox (A. lagopus)*

The arctic fox is one of the smaller predatory animals found inhabiting Canada's arctic. Although they may fall prey to owls, bears and other larger carnivores, the arctic fox is still considered to be a top predator, feeding on regionally-important species of small mammals such as arctic hare and lemmings. By virtue of its location in the food chain, the fox is susceptible to contaminants that accumulate in the tissue of its prey. Despite significant food shortages during the harsh winter months, the arctic fox does not migrate. Food caught during the summer is preserved in the permafrost and retrieved later. As a result, exposure to on-site contamination will occur on a continuous basis. The home range of the arctic fox is expected to be relatively large in comparison to the site area. Cominco has committed to removing all current facilities, ensuring that foxes will not be able to take advantage of the shelter offered by the abandoned structures on the site when choosing dens. This would otherwise place them and their young in relatively close proximity to the areas of contamination.

Anecdotal evidence by mine site personnel indicates that the population of arctic fox has increased since the mine opened, largely due to increased opportunities for shelter and year round food sources. Following mine closure, the likelihood of foxes remaining in the immediate vicinity should diminish (GLL 2000).

#### *Other terrestrial wildlife qualitatively, but not quantitatively, evaluated*

Although muskox and polar bears have been identified as being environmentally and culturally important, these receptors were excluded from further quantitative evaluation for the following reasons.

- 1) Quantified chemical risks to caribou can also be applied to muskox. Muskox are larger than caribou and when considering the reduced chemical dose on a "per unit body weight" basis, would actually be exposed to a lesser extent. Therefore, if chemical risks to caribou were considered "acceptable", the same could automatically be said for muskox.
- 2) Muskox were considered less likely to spend time foraging at the mine site in the future.

- 3) Polar bears have a diversified diet and cover a large territory while foraging for food. It is difficult to accurately quantify their chemical exposure for this reason. Arctic foxes were assumed to feed only small mammals that spent their entire lifetimes on the site. For this reason, the arctic fox was chosen to be the higher-trophic level animal that could potentially be at the highest chemical risk.

#### *4.3.1.2 Exposure Pathway Screening*

The possible pathways that terrestrial wildlife receptors could be exposed to chemical contaminants in soil and vegetation were evaluated for their contribution towards the total predicted exposure rate from the site. Pathways were selected based on the behavioural characteristics of the receptors, site characteristics, the source of contamination, the types of receptors present, and chemical fate. Only the most significant pathways were assessed in the ERA. If risks were found to occur with these major pathways, reassessment of less significant exposure pathways would be necessary.

The following sections summarize the pathways selected for the wildlife receptors identified for the ecological assessment:

##### *(i) Lemming, arctic hare, and caribou*

Potential exposure pathways for the lemming, arctic hare, and caribou were reviewed based on the nature of the site and the contaminants. Due to similarities in behavioural characteristics, the following major exposure pathways were identified for these species:

- Ingestion of contaminated soils or dusts;
- Ingestion of potentially contaminated vegetation; and,
- Direct dermal contact with soils or dusts (except for caribou).
- Inhalation of contaminated dusts.

The apparent absence of suitable drinking water areas at the mine site precluded the evaluation of the drinking water pathway.

##### *(ii) Arctic fox*

Potential exposure pathways for the arctic fox were reviewed. The following major exposure pathways were identified for this species:

- Ingestion of contaminated soils or dusts;
- Direct dermal contact with soils or dusts; and,
- Ingestion of potentially contaminated prey species (lemming);
- Inhalation of metal-contaminated dusts.



### *Chemical Screening*

Initial screening of contaminant levels in soil with respect to potential environmental impacts was conducted using the appropriate guidelines or criteria established by Canadian regulatory agencies (e.g., CCME, BC MELP). Only those metals which were detected in surface soils were considered. In cases where metals “qualified” for further evaluation (i.e., maximum surface soil concentration exceeded guideline), the mean site concentration was adopted for the ecological risk assessment.

Considering that the soil quality criteria for residential/parkland use (CCME 1999) are considered protective of both human health and the environment, the chemical screening methodology used for the human health risk assessment was also employed for the ecological risk assessment (see section 3.2.1.4 in the HHRA).

Metals that were identified as exceeding soil quality guidelines for environmental protection (CCME, 1999) and were subsequently included in the quantitative ecological risk assessment included:

- Cadmium
- Copper
- Lead
- Zinc

#### *4.3.1.3 Selection of Assessment and Measurement Endpoints*

In order to narrow the focus of the risk assessment, it was necessary to select assessment endpoints, which may include estimating risks at the individual, population, community or ecosystem level. The assessment endpoints for the ERA should be relevant to the site-specific contamination and should be capable of being assessed based on the available data (CCME, 1996). For the purposes of the proposed ERA, assessment endpoints were selected to evaluate the potential for site-specific toxic effects that could result in reduction of populations of valued ecological receptors, relative to comparable non-contaminated sites or background areas.

Assessment endpoints must be translated into measurement endpoints to practically conduct an ERA. Measurement endpoints are measurable environmental characteristics that are related to the assessment endpoint (CCME, 1996). For the proposed ERA, individual and population level measurement endpoints (i.e., effects on survival, reproduction, growth, etc.) were selected for the chemicals of concern in the toxicity assessment in order to determine exposure limits and toxicity benchmarks for the selected ecological receptors.

#### *4.3.2 Exposure Assessment*

Exposure assessment involves the estimation of the amount of chemical received by ecological receptors per unit time (i.e., the quantity of chemical and the rate at which that quantity is

received). Exposure assessment was conducted for all chemicals, ecological receptors and exposure pathways short-listed during the problem formulation phase of the ERA.

#### 4.3.2.1 *Exposure to Herbivorous Terrestrial Organisms*

The rate of contaminant exposure to herbivorous terrestrial organisms was evaluated based on mean soil and vegetation concentrations from the site. For migrating animals such as the caribou, consideration was given to the amount of time that these animals are expected to spend at the site in order to correct the exposure rate.

A summary of the sampled contaminant concentrations in soil and vegetation used in the ecological risk assessment is presented in Table 4.

Sampled vegetation included:

- Grasses (*Alopecurus alpinus*)
- Lichen (*Thamnolia subuliformis*)
- Willow (*Salix arctica*)

Vegetation samples were taken from 13 stations previously established in the environmental baseline study (BC Research 1975).

**Table 4. Mean soil and vegetation concentrations of the chemicals of potential concern at, and adjacent to, the Polaris mine site**

Chemical	Geometric mean of measured site concentration (mg/kg) <sup>1</sup>				
	Surface soil <sup>2</sup>	Grasses	Lichen	Willow root	Willow top
Cadmium	17.0	0.8	1.2	3.2	3.0
Copper	12.0	4.6	1.4	5.8	4.4
Lead	141.0	25.0	256.2	13.4	11.4
Zinc	3635.0	148.8	145.0	351.7	376.6

<sup>1</sup> all geometric means except for zinc (arithmetic mean), for which no geometric mean could be calculated

<sup>2</sup> surface soils indicate those samples less than or equal to 0.5 cm

#### 4.3.2.2 *Exposure to Predatory Wildlife Receptors*

The objectives of the exposure assessment for predatory wildlife, such as the arctic fox, was to determine the exposure rates for chemicals of concern *via* ingestion of prey (lemmings) that contain chemical residues as a result of feeding on contaminated vegetation. The exposure was estimated for the site sources *via* quantitative exposure modelling.

A deterministic approach was selected not only because it is a relatively time and cost efficient method for predicting exposure based on the narrow range of input parameters expected for

wildlife receptors in the proposed ERA, but also because it is a conservative approach for estimating risk.

Exposure rates were estimated based on the interactions of a number of parameters, including:

- the various physical, chemical, and biological factors that determine the uptake of chemicals into biological organisms (e.g., bioconcentration, bioaccumulation, bioavailability of the chemicals from soil particles and foods);
- the physical/chemical characteristics of the chemicals which determine their interaction and behaviour with the surrounding environment (e.g.,  $K_{ow}$ , tendency to bind to particles); and,
- the behavioural lifestyle and physical characteristics of the selected ecological receptors that determine the dose rates through the various exposure pathways (e.g., respiration rate, food intake, dietary composition, soils/dust intake, migratory patterns, etc.).

#### 4.3.2.3 *Chemical Bioavailability*

Every attempt was made to assess chemical bioavailability on a chemical specific, receptor specific and site specific basis. When ranges of data are indicated in the literature, maximum values were used in exposure modelling. However, if limited data on the bioavailability of chemicals was available in the published literature, it was assumed that the chemical was 100% bioavailable, in order to provide a conservative assessment of the potential risk.

#### 4.3.2.4 *Physical/Chemical Characteristics*

The physical/chemical characteristics of chemicals (tendency to bind to particles) determine their behaviour in the environment and their tendency to enter pathways by which receptors are exposed. Therefore, knowledge of the physical/chemical characteristics is critical to the estimation of the importance of exposure pathways and the magnitude of subsequent exposures. The most current physical/chemical characteristics were selected from the published literature for the chemicals of concern.

#### 4.3.2.5 *Receptor and Parameter Assumptions*

For each receptor, a number of conservative but realistic assumptions were used to facilitate the quantitative modelling process helping to ensure that the exposure levels predicted were not underestimated. The estimation of exposure to terrestrial receptors was based on the individual physiological characteristics of each receptor species (e.g., soil/dust consumption, breathing rate, body weight, food consumption). Where possible, receptor parameters published by regulatory agencies (e.g., US EPA) were used for lemmings, arctic hares, caribou, and arctic fox. When data regarding the above mentioned receptors were not available, data from similar species were used to represent the specific ecological receptor of concern. When ranges of data were reported

in the literature, values resulting in the most realistic scenario were assumed for the current ERA. For example, it would be unrealistic to apply the minimum body weight of an arctic hare and the maximum daily food intake rate when deriving an exposure ( $\mu\text{g/kg bw/day}$ ) from the consumption of chemically impacted vegetation.

Every attempt was made to ensure that the conservative assumptions used in the current assessment were realistic in nature. The following section discusses the assumptions used for each receptor of concern.

#### *Lemming*

For the purpose of the current assessment, lemmings were assumed to spend 100% of their time on-site. While on-site, the lemming's diet consisted of chemically-impacted grass and willow.

#### *Arctic hare*

For the purpose of the current assessment, hare were assumed to spend 100% of their time on-site. While on-site, the hare diet consisted solely of willow roots.

#### *Caribou*

For the purpose of the current ERA, caribou were assumed to spend 25% of their time on-site. While on-site, the caribou's diet was conservatively assumed to consist of vegetation taken strictly from within the boundaries of the site.

#### *Arctic fox*

For the purpose of the current assessment, the arctic fox was assumed to spend 100% of its time within the site boundary area. The exposure of the arctic fox *via* the food chain was estimated by conservatively assuming that the diet of the fox was comprised of 80% hare and 20% lemming and that these prey animals also spent 100% of their time on-site.

The individual physiological characteristics for each receptor species used in the current ERA are outlined in greater detail in Appendix B.

### **4.3.3**            Toxicity Assessment

#### **4.3.3.1**        *Toxicity to Plants and Soil-Dwelling Organisms*

Due to the nature of the site ("insignificant" plant coverage, extreme latitude, *etc.*), toxicity to plants and soil-dwelling organisms was not included in the current ERA. Rationale for the exclusion is discussed in greater detail in section 5.1.

The potential toxicological impact on plant life resulting from Cominco's operation of its Polaris mine is discussed in detail by C.E. Jones under separate cover (GLL 2000).

#### 4.3.3.2 Toxicity to Terrestrial Wildlife Receptors

A wildlife toxicity assessment was conducted to determine exposure limits for lemmings, arctic hare, caribou, and arctic fox for the chemicals of concern. The NOAEL (No-Observable-Adverse-Effect-Level) exposure limit refers to the rate at which a receptor can be exposed to a given chemical without the occurrence of adverse health effects. The LOAEL (Lowest-Observable-Adverse-Effect-Level) exposure limit is the rate of exposure associated with the occurrence of some adverse effect in the test species. The actual threshold of the occurrence of adverse in test species lies somewhere between the NOAEL and LOAEL. Exposure limits were expressed in terms of the quantity of chemical per body weight per day (*e.g.*, mg of chemical/kg body weight/day), and are presented in Table 5.

**Table 5. Exposure limits<sup>1</sup> (mg/kg/day) for terrestrial wildlife for the ecological risk assessment of Polaris mine site**

Chemical	Lemming	Arctic hare	Caribou	Arctic fox
Cadmium	5.0	5.0	5.0	1.7
Copper	80.0 <sup>2</sup>	80.0 <sup>2</sup>	4.2	8.0 <sup>2</sup>
Lead	0.6	0.6	5.0	1.0 <sup>2</sup>
Zinc	210.0	210.0	17.0	20.0

<sup>1</sup> exposure limit based on lowest-effect level unless otherwise stated

<sup>2</sup> exposure limit based on no-effect level

These exposure limits were derived based on published studies of toxicity in the receptor species or similar wildlife species, if available, or from studies in which laboratory animals were used. In cases in which more than one study was available and suitable for derivation of an exposure limit, the data yielding the most conservative exposure limit was used. NOAELs and LOAELs were identified, and if appropriate, uncertainty factors were used in the derivation of exposure limits (*i.e.*, ten-fold uncertainty factors were applied for the use of a LOAEL and use of a sub-chronic study). It was assumed that the exposure limits were protective of the mean or average population of lemmings, arctic hare, caribou, and arctic fox and that application of additional uncertainty factors for species sensitivity would result in a relatively conservative assessment.

The derivation of these exposure limits is explained further in Appendix A.

#### 4.3.4 Risk Characterization

The final process in the ERA for terrestrial wildlife consisted of a comparison of the exposure limits (i.e., either the NOAEL, the rate of exposure that would not produce adverse effects or the LOAEL, the rate of exposure that would potentially produce adverse effects) against the total estimated exposure. This comparison is expressed as a Hazard Quotient (HQ) and is calculated by dividing the predicted exposure by the exposure limit, as indicated in the following equation:

$$\text{Hazard Quotient} = \frac{\text{Estimated Exposure}}{\text{Exposure Limit}}$$

Due to exclusion of the drinking water pathway, the acceptable level of exposure was assumed to be 80% of the exposure limit. CCME (1996) protocol suggests that up to 20% of total exposure may come from drinking contaminated water. For this reason, an HQ value less than 0.8 indicates that the estimated total exposure is less than the upper limit of total exposure, and therefore no risk of adverse health effects is predicted. An HQ value greater than 0.8 indicates that the estimated exposure level exceeds the exposure limit, and may pose an unacceptable adverse health risk to the wildlife receptors. HQ values that are slightly less than or greater than 0.8 require re-evaluation of the conservative nature of the assumptions before the potential risks to terrestrial wildlife can be characterized.

As discussed earlier, the actual concentration where adverse effects begin to occur in the test species lies somewhere between the NOAEL and the LOAEL. Therefore, HQ values based on NOAEL-derived exposure limits provide an indication of the likelihood that adverse effects will not occur in the receptor species, while HQ values based on LOAELs indicate the likelihood of occurrence of adverse effects in receptor species if future predicted exposures are not overestimated. Therefore, HQ values associated with on-site exposures were calculated based on either (dependent on the available toxicological information):

- (i) HQ values for the Protection of Ecological Health (HQ<sub>NOAEL</sub>): The predicted exposure was compared to the exposure limits derived from study NOAELs. HQ values calculated in this manner indicated the likelihood that adverse effects will not occur in terrestrial wildlife.
- (ii) HQ values for the Likelihood of Adverse Effects (HQ<sub>LOAEL</sub>): The predicted exposure was compared to the exposure limits derived from study LOAELs. HQ values calculated in this manner indicated the likelihood that adverse effects may occur in terrestrial wildlife.

#### 4.4 General Assumptions Used in the Ecological Risk Assessment

Attempts to estimate absolute risk levels from a single exposure situation are affected by the uncertainties in the estimation of the exposure limits, chemical concentrations in various media, receptor characteristics, and the ultimate exposure levels predicted. Therefore, it is necessary to

outline a set of assumptions to guide the ERA process. The underlying basis of these assumptions in an ERA is to conduct an assessment that is conservative and yet realistic, taking into account the uncertainties in the chemical, biological and toxicological data. The conservative assumptions used in the current ERA are presented in the following sections.

#### **4.4.1            *Assumptions Associated with Exposure Estimations***

The following conservative assumptions were used in the estimation of exposures to terrestrial wildlife (see section 5.3.2):

- (i) All wildlife receptors (except caribou) were assumed to spend 100% of their time feeding on-site.
- (ii) Oral, inhalation and dermal bioavailability parameters used for estimating chemical exposure in terrestrial wildlife were based on other mammalian or human species when no species-specific data were available. Dermal bioavailability was assumed to be substantially higher in fur-bearing animals, in comparison to humans. One hundred percent bioavailability was assumed where chemical specific data were unavailable.
- (iii) The most conservative values for chemical and biological parameters (*e.g.*, maximum chemical bioavailability, maximum food consumption) were used where ranges were reported in the literature.

Generally, the assumptions made in selecting the various receptor characteristics and environmental fate parameters for the chemicals of concern tend to provide conservative, yet realistic, estimates of chemical exposure in the receptors.

#### **4.4.2            *Assumption Associated with the Derivation of Exposure Limits***

The following conservative assumptions were used in the derivation of exposure limits for terrestrial receptors:

- (i) A literature review was conducted to identify appropriate exposure limits for rodents and large mammals. Due to the limited toxicity database for large mammals, the exposure limits used for the current assessment were at times derived *via* the application of safety factors (see Appendix A).
- (ii) The exposure limits selected for terrestrial wildlife were assumed to be protective of the mean or average population.

### **4.5                Results of the Ecological Risk Assessment**

Based on the methodology described above, the risks to ecological health were estimated for the intended/future use of Cominco's mined area following decommissioning of the site. Results are

presented “species-by-species” for those chemicals considered to pose a potential threat to ecological health. Risks resulting from exposure to the on-site metal soil concentrations are discussed for each wildlife receptor below. The derivation of these results are outlined in greater detail in Appendix B.

#### 4.5.1 *Lemming*

Health risks to lemmings exposed to contaminant-impacted surface soils and vegetation are outlined in Table 6.

**Table 6. Health risks to lemmings exposed to metal concentrations at Cominco’s Polaris mine site**

	Cadmium	Copper	Lead	Zinc
Exposure (µg/kg/day):				
Plant ingestion	57.5	595.0	1285.8	17675.1
Soil ingestion	1.4	0.5	5.7	293.5
Dust inhalation	0.0	0.0	0.0	0.0
Dermal contact	4.3	7.2	2.0	1641.2
Total exposure	63.2	602.7	1293.5	19609.8
<b>Hazard Quotient</b>	<b>0.1</b>	<b>0.0</b>	<b>5.0</b>	<b>0.2</b>

As indicated in Table 6, the only metal that resulted in an exceedance of the targeted HQ value of 0.8 was lead (HQ = 5.0). Approximately 99% of the exposure (and hence risk) is due to ingestion of lead-contaminated vegetation.

The risk assessment did not account for reduced foraging activity during the colder months of the year. The assessment assumed that the level of feeding activity remained constant throughout the year (*i.e.*, no hibernation). This assumption probably led to a substantial overestimation of potential lead-related health risks. For these reasons, along with the low estimated hazard quotients for the other metals, no adverse effects are expected for the LCI lemming population as a result of Cominco’s on-site soil contamination.

#### 4.5.2 *Arctic hare*

Health risks to hare exposed to contaminant-impacted surface soils and vegetation are outlined in Table 7.



**Table 7. Health risks to hare exposed to metal concentrations at Cominco's Polaris mine site**

	Cadmium	Copper	Lead	Zinc
Exposure (µg/kg/day):				
Plant ingestion	46.5	367.7	453.9	11355.3
Soil ingestion	0.6	0.2	2.3	117.3
Dust inhalation	0.0	0.0	0.0	0.0
Dermal contact	0.9	1.5	0.4	354.5
Total exposure	48.0	369.4	456.6	11827.1
<b>Hazard Quotient</b>	<b>0.1</b>	<b>0.0</b>	<b>1.8</b>	<b>0.1</b>

As outlined in Table 7, the only metal that resulted in an exceedance of the targeted HQ value of 0.8 was lead (HQ = 1.8). Similar to the lemming, approximately 99% of exposure to the arctic hare is due to the ingestion of lead-contaminated vegetation.

The risk assessment assumed that arctic hare spend all their time foraging at the Polaris Mine site area. However, the barren nature of the site would suggest that the area is unlikely to naturally sustain an arctic hare population. For this reason, lead-related risks to arctic hare were likely substantially overestimated and hence no adverse effects are expected for any LCI hare populations as a result of Cominco's on-site soil contamination.

#### **4.5.3 Peary caribou**

Health risks to Peary caribou exposed to contaminant-impacted surface soils and vegetation at the mine site are outlined in Table 8.

**Table 8. Health risks to caribou exposed to metal concentrations at Cominco's Polaris mine site**

	Cadmium	Copper	Lead	Zinc
Exposure (µg/kg/day):				
Plant ingestion	13.3	174.2	4358.9	4343.4
Soil ingestion	0.5	0.2	2.1	107.4
Dust inhalation	0.0	0.0	0.0	0.0
Total exposure	13.8	174.4	4361.0	4450.8
<b>Hazard Quotient</b>	<b>0.0</b>	<b>0.1</b>	<b>2.1</b>	<b>0.7</b>

Estimated HQ values only exceeded the critical value of 0.8 for lead for caribou. Close to 100% of the risk was due to ingestion of lead-contaminated plants.

The caribou population on LCI is small. For those few caribou that will visit the site, the assessment conservatively assumed that they would spend an entire season foraging solely on the mining site's chemically impacted vegetation.

The exposure limit for lead is based on a "lowest-observed-effect" level, with the measured effects included a slight reduction in calves' weight along with decreased ALAD levels. Both effects are of uncertain ecological relevance and so population effects would be considered unlikely.

Due to the limited number of caribou expected to regularly visit the mine site and the conservativeness of the exposure limits, effects, albeit unlikely, would be expected to occur only on an "individual-animal-basis". Considering that ecological risks are measured on whether or not a population could sustain itself at the estimated chemical risk level, current on-site surface soil concentrations are not expected to deleteriously impact the survivability and sustainability of the local caribou population.

#### 4.5.4 *Arctic fox*

Health risks to arctic fox exposed to contaminant-impacted surface soils and vegetation at the mine site are outlined in Table 9.

**Table 9. Health risks to arctic fox exposed to metal concentrations at Cominco's Polaris mine site**

	Cadmium	Copper	Lead	Zinc
Exposure (µg/kg/day):				
Prey ingestion	105.1	16.5	736.4	269.5
Soil ingestion	0.5	0.2	2.0	102.5
Dust inhalation	0.0	0.0	0.0	0.0
Dermal contact	0.5	0.8	0.2	179.2
Total exposure	106.1	17.5	738.6	551.2
<b>Hazard Quotient</b>	<b>0.4</b>	<b>0.0</b>	<b>1.8</b>	<b>0.1</b>

The only metal that exhibited an elevated HQ value was lead (1.8). Again, close to 100% of the risk can be attributed to the ingestion route of exposure. Prey (*e.g.*, lemmings and hare) retain lead (*i.e.*, bioaccumulate) which in turn leads to exposure to higher trophic animals such as the arctic fox.

The assessment assumed that lemmings and hare spent 100% of their time feeding on-site. The assessment did not consider a reduction in herbivorous feeding activity during the winter months (with snow cover and lower temperatures) which may have resulted in the over prediction of lemming and hare tissue concentrations. Arctic fox were also considered to spend all their time on-site, with no reduced feeding activity during the colder months.

The fox exposure limit for lead is based on a “no-observed-effect” level, suggesting that actual risks resulting from lead exposure would likely be substantially less (up to 10 fold).

Due to the conservative nature of the assessment, and the apparent “beneficial” impact Cominco’s mine has had on the “abundance” of foxes in the immediate vicinity, no population-level effects are expected for foxes exposed to on-site surface soil concentrations of the evaluated metals.

#### **4.6 Ecological Risk Assessment Conclusions**

The results of the ecological risk assessment indicated elevated risks associated with wildlife exposure to lead-contaminated soils and vegetation.

Due to the barren nature of the mine site, it is unlikely that the evaluated wildlife receptors would spend a significant portion of their time foraging on-site. The assumption wherein wildlife spent 100% of their time on-site most likely led to the overestimation of potential ecological risks.

Predicted ecological risks and potential deleterious effects are confined to the individual level. This means that the metal contamination of the surface soils surrounding Cominco’s Polaris mine is not expected to impact the survivability or sustainability of any of the evaluated terrestrial wildlife populations (*i.e.*, no measurable effects on the ecological populations).

It should be stressed that the estimated risk to chemical exposure at the mine site was primarily due to plant/prey ingestion. Removal of the plant ingestion pathway would result in acceptable risks to lemmings, hare, caribou and subsequently arctic fox. However, due to the sensitive nature of the high arctic ecosystem, removal of any plants on LCI, along with the remediation of soils supporting these plants, would likely do more harm than good. This is discussed further in section 5.

#### **5.0 SOIL QUALITY REMEDIATION OBJECTIVES**

In an attempt to ensure LCI’s overall environmental health, Cominco has committed to remediating the area’s soils to the extent where no future ecological or human health impacts would be seen. Soil quality remediation objectives (SQROs) are chemical soil concentrations that would not pose a risk to critical (*i.e.*, most sensitive) receptors spending time on-site.

SQROs for metals of potential concern were determined within the permitted framework as governed by the Canadian Environmental Quality Guidelines (CCME 1999). This framework, outlined in the National Contaminated Sites soil protocol, provides the opportunity to move from generic soil guidelines to site-specific remediation objectives.

The unique nature of the Polaris mine site suggests that the application of generic soil quality guidelines is not appropriate for remediative purposes. The “barren and rugged” terrain of LCI (Graham 1982) precludes the regular use of the area by both wildlife and human receptors. Further, the atypical characteristics and unusual exposure scenarios indicative of the high arctic location of the mine site necessitated the development of site-specific SQROs.

SQROs are based on the most sensitive receptor (ecological or human) to a chemical of potential concern. For example, a SQRO for chemical “X” of 100 mg/kg may be considered a safe soil concentration for herbivores, but may not be stringent enough for predatory mammals or the most sensitive members of surrounding Inuit communities. For this reason, the lowest SQROs for either ecological or human receptors were chosen for the metals of concern.

## **5.1 Basis of the Soil Quality Remediation Objectives**

It should be stressed that although unacceptable ecological risks were predicted for cadmium, lead and zinc, the soil quality remediation objectives were largely based on the results of the human health risk assessment.

Risks to wildlife receptors were primarily due to ingestion of on-site metal-contaminated plants. The high arctic is an extremely sensitive ecosystem and removal of any vegetation, or soils supporting that vegetation, in an attempt to eliminate exposure resulting from plant ingestion would result in comparatively greater environmental impact to the area. A study by CE Jones (GLL 2000) evaluated the potential impact that the operation of the Polaris Mine may have had on the study area’s vegetation.

Considering that exposure to both lead and zinc posed potentially unacceptable human health risks, SQROs were derived for these two metals.

## **5.2 Soil Quality Remediation Objective for Lead**

Results from the risk assessment identified Inuit children acutely exposed to lead- and zinc-contaminated soils as being the most sensitive receptors. Considering that the most sensitive receptors of exposure to lead are human foetuses, infants, and children up to six years of age (ATSDR, 1988), the site-specific SQRO for lead is intended to protect Inuit from measurable health effects associated with the future use of the site.

The “benchmark” probability that U.S. EPA uses to guide remediation decisions is a less than 5% probability that an individual’s blood lead level will exceed 10 µg/dL for long-term, chronic exposure periods (White *et al.*, 1998). This target blood lead level is appropriate for long-term, chronic exposures and effects (*e.g.*, effects on cognitive abilities). However, sensitive receptors such as Inuit children are only expected to visit the Mine site for perhaps one to three days per

year (B.C. Research, 1975; LaVigne, 1980; results from community consultation, 2000). This type of short-term or acute exposure period suggests a different toxicological impact. Ingestion of soil while visiting the Mine site could result in larger, temporary increases in blood lead levels, possibly giving rise to such clinical effects as gastrointestinal distress, weight loss, tremors, irritability, and lethargy. For the purposes of this risk assessment, a more relevant short-term “benchmark” of 20 µg/dL was used as a target blood lead level (CDC, 1991; Health Canada, 1994).

The following relationship describes the uptake of lead into blood:

$$[\text{Pb}]_{\text{blood}} = \alpha + \beta(\text{uptake})$$

where Pb = concentration of Pb in blood (µg/dL)

$\alpha$  = y intercept (baseline blood-Pb level = 4 µg/dL)

$\beta$  = slope of the regression line (statistical regression technique was used to fit a curve to absorbed lead versus blood-Pb levels assume 0.04, US EPA 1989)

Assuming that a “safe” level or target level is 20 µg/dL ...

$$20 \text{ µg/dL} = 4 + 0.04 (\text{uptake Pb}),$$

$$\text{uptake Pb} = 400 \text{ µg/day}$$

So total exposure during an exposure episode should not exceed 400 µg or 0.4 mg. An Inuit child visiting the site can be exposed to lead through a number of pathways which include:

- Incidental ingestion of soil;
- Inhalation of soil and dusts;
- Dermal contact with soil.

The SQRO was estimated by back-calculating for soil concentrations that would result in an uptake of 400 µg/day.

The assessment incorporated the upper percentile of U.S. EPA recommended soil ingestion rates (400 mg/day; U.S. EPA, 1996), as well as a base-line blood lead level of 4 µg/dL (Wood, 2000) to calculate a site-specific SQRO for lead concentrations in soil of 2,000 ppm. Lead concentrations in surficial soil that are equal to or below this recommended objective are not expected to cause adverse health effects to children visiting the Mine site for brief, infrequent periods of time.

It should be noted that this SQRO is not protective of children exhibiting very high soil ingestion rates (*e.g.*, pica). This condition is typically not addressed in risk assessments, nor is it commonly considered when setting regulatory soil quality guidelines. Recent studies by Calabrese *et al.* (1997) suggest that certain children will ingest up to 50 g of soil per day during

episodes of pica behaviour. At this ingestion rate, soil guidelines set by such regulatory agencies as CCME and U.S. EPA may not be stringent enough to protect the health of pica children.

### **5.3 Soil Quality Remediation Objective for Zinc**

The risk assessment identified the receptor (wildlife or human) deemed most sensitive to zinc toxicity given their behavioural patterns and likelihood of exposure. In deriving a site-specific SQRO for zinc, the risk assessment:

- Determined a daily exposure to zinc that would result in either no measurable health effects on human individuals or wildlife populations; and in turn,
- Resolved a zinc soil concentration that, when ingested/inhaled/contacted, would not result in adverse effect.

Results from the risk assessment identified humans to be the most sensitive receptors under the assumed exposure scenarios. Zinc is a nutritionally essential metal and exhibits relatively low toxicity to terrestrial mammals and humans. Zinc does not accumulate with continued exposure, and body content is modulated by homeostatic mechanisms (Klaassen, 1996).

Zinc toxicity from excessive ingestion is uncommon, but gastrointestinal distress and diarrhoea have been reported following ingestion of large doses. Due to the lack of overall human zinc toxicity data, the Health Canada provisional tolerable daily intake of 0.30 mg/kg bw/day was used as an acute exposure limit for zinc (Health Canada, 1996). It is recognized that this likely overestimates the potency of zinc on an acute basis, however, at this time no further information was readily available.

When considering this acute exposure limit and the set of exposure assumptions, the results of the risk assessment indicate that the site-specific soil quality remediation objective for zinc at the Polaris Mine that would adequately protect both human and environmental health is approximately 10,000 mg/kg.

It should also be noted that plants at the mine site have been shown to survive at a zinc soil concentration of 10,000 mg/kg.

## **6.0 SUMMARY OF RISK ASSESSMENT RESULTS**

For the ecological and human health risk assessment, the intended use of Cominco's Polaris site following closure of the mine was evaluated through active consultation with members of the communities of Resolute Bay and Grise Fiord. Participants in the community survey identified a number of valued ecosystem components (specifically wildlife receptors) that were subsequently evaluated in the ecological risk assessment. Participants also outlined the intended land use of the mine site area, which can best be described as being representative of short-term exposure scenario.

Results of the ecological risk assessment indicated elevated risks for several of the evaluated wildlife receptors. However, further evaluation found that any predicted ecological risks and potential deleterious effects would be confined to the individual organism level. The metal contamination of the surface soils surrounding the mine is not expected to impact the survivability of the evaluated wildlife populations.

Results of the human health risk assessment identified exceedance of the targeted risk level of 1.0 for both lead (8.8) and zinc (5.8).

To ensure the overall environmental well-being of Little Cornwallis Island, soil quality remediation objectives were derived. The SQROs were based on the most sensitive receptor spending time on-site, which in the case of lead and zinc were found to be Inuit children.

The surface soil concentrations considered safe for the most sensitive receptors were estimated to be:

- Lead                      2,000 mg/kg
- Zinc                        10,000 mg/kg

Lead and zinc concentrations in surficial soil that are equal to or below these recommended objectives are not expected to cause adverse health effects to Inuit children visiting the mine site under the conditions identified through the consultation process.

## **7.0 REFERENCES**

- B.C. Research 1975. Environmental Study of Polaris Mine, Little Cornwallis Island. Submitted to Cominco Ltd., Project No. 1642. Prepared by Division of Applied Biology, B.C. Research.
- Bryant Environmental Consultants Ltd. 1997. Environmental Evaluation Report: The Eclipse Report (Draft). Vol I and II. Yellowknife, Northwest Territories.
- Calabrese, E.J., Stanek, E.J., James, R.C. and S.M Roberts, 1997. Soil ingestion: A concern for acute toxicity in children. *Environ. Health Persp.*, 105(12): 1354-1357.
- CCME (Canadian Council of Ministers of the Environment) 1996. A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines. The National Contaminated Sites Program. CCME-EPC-101E. March, 1996.
- CDC (U.S. Centre for Disease Control and Prevention), 1991. Preventing lead poisoning in young children. U.S. Department of Health and Human Services, Public Health Service.

Available online at

<http://www.cdc.gov/nceh/pubcatns/1994/cdc/books/plpsn/plpsn7.htm#sum>

- Efroymson, R.A. and G.W. Suter II. 1999. Finding a niche for soil microbial toxicity tests in ecological risk assessment. *Human and Ecological Risk Assessment* 5(4):715-727.
- Efroymson, R.A., Will, M.E. and Suter II, G.W. 1997. Toxicological benchmarks for contaminants of potential concern for effects on soil and litter invertebrates and heterotrophic processes: 1997 revision. ES/ER/TM-126/R2. Oak Ridge National Laboratory, Oak Ridge, TN.
- GLL (Gartner Lee Ltd.) 1999. Cominco Ltd. – Polaris Operations. Draft Environmental Assessment Report. November, 1999. GL 99-902.
- Graham, K.A. 1982. Eastern Arctic Study, Case Study Series: The Development of the Polaris Mine. Joint Project of the Institute of Local Government and the Centre for Resource Studies. Queen's University, Kingston, Ontario.
- Health Canada, 1994. Uptake of evidence for low-level effects of lead and blood lead intervention levels and strategies – Final report of the Working Group. Federal-Provincial Committee on Environmental and Occupational Health, Environmental Health Directorate, Ottawa, Ontario.
- Health Canada 1996. Health based tolerable daily intakes / concentrations and tumorigenic doses / concentrations for priority substances. Environmental Health Directorate, Health Protection Branch, Health Canada, Ottawa. ISBN 0-662-24858-9.
- Kapustka, L.A. 1999. Microbial endpoints: The rationale for their exclusion as ecological assessment endpoints. *Human and Ecological Risk Assessment* 5(4):691-696.
- Klaassen, C.D. (Ed) 1996. Casarett & Doull's Toxicology: The Basic Sciences of Poisons. Fifth Edition. McGraw-Hill. Toronto.
- LaVigne, M. 1980. Potential Socio-Economic Impacts of the Polaris Mine Project. Prepared by Outcrop Ltd. (Yellowknife) in association with DPA Consulting Ltd. (Vancouver).
- U.S. EPA (U.S. Environmental Protection Agency), 1996. Exposure Factors Handbook. Vol 1: General Factors, page 4-24.
- White, P.D. *et al.* 1998. The conceptual structure of the integrated exposure uptake biokinetic model for lead in children. *Environ. Health Persp.*, 106: 1513-1530.
- Wood, C., 2000. Alaska State Health – Epidemiology Department (Head of lead task force). Personal Communication.



**APPENDIX A**  
**TOXICITY PROFILES**

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**A-1.0 HUMAN TOXICITY PROFILES****A-1.1 Cadmium****A-1.1.1 Bioavailability****A.-1.1.1.1 Oral route of exposure**

Absorption of cadmium via the gastrointestinal tract appears to be a saturable process (Nordberg *et al.*, 1985). The amount of metal absorbed as a direct result of soil ingestion depends on the individual, the form of the metal within the soil, and the soil's physical characteristics (Ashmead *et al.*, 1985). Calabrese *et al.* (1997) determined that ingestion of 5 g of soil during a soil pica episode in young children would result in a dose of 0.03 mg cadmium/kg body weight. It has been concluded in several studies that approximately 5% of orally ingested cadmium is absorbed by the gastrointestinal tract (Rahola *et al.*, 1972; Friberg and Kjellstrom, 1981; Leonard *et al.*, 1984; Kazantzis, 1987). Estimates of cadmium absorption by ingestion based on human studies using radiolabelled cadmium indicated an average absorption of 4.6% (range of 0.7 to 15.6%) (McLellan *et al.*, 1978). Other assessments report estimates of 5 to 10% absorption (IARC, 1976; NAS, 1977). For the current exposure assessment the fraction of cadmium absorbed via ingestion was assumed to be 15.6%.

**A-1.1.1.2 Inhalation route of exposure**

WHO (1984) concluded that on average, 25% of cadmium in the general air would be retained in the respiratory system. In occupational exposures, absorption from the lung has been reported to range from 20 to 50%, depending on respirable particle sizes (Elinder *et al.*, 1976; Kazantzis, 1987). US EPA (1980) concluded that 25% of respirable particles would be absorbed; however, the absorption of cadmium fumes (*i.e.*, from cigarette smoke and industrial sources) may be as high as 50%. Based on the dynamics of total airborne particles (including respirable and non-respirable particles) in the respiratory system, about 13% of airborne cadmium on particles would be retained and absorbed in the lungs following inhalation. In addition, approximately 60% of airborne particles would be cleared by the mucociliary apparatus and swallowed. If 0.7 to 15.6% of the swallowed cadmium were absorbed from the gastrointestinal tract, based on oral bioavailability, then 0.42 to 9.36% (0.7% of 60%; 15.6% of 60%) of airborne cadmium would be bioavailable by this route. Therefore, based on airborne particle dynamics, the total bioavailability of airborne cadmium from particles would be 13.42 to 22.36% (13% from the respiratory system plus 0.42 to 9.36% of the cleared material). For the current assessment, absorption of cadmium via the respiratory system was assumed to be 22%.

**A-1.1.1.3 Dermal route of exposure**

Studies show that absorption of cadmium via the skin is not substantial (CEC, 1978; Carson *et al.*, 1986, Skog and Wahlberg, 1964; Wahlberg, 1965). Westar *et al.* (1992) calculated a dermal absorption of 0.5% to 1.0 % for cadmium chloride from water and soil using a model of human cadaver skin and human plasma. In estimating dermal exposure to cadmium for the current exposure assessment, the amount of cadmium absorbed by the skin was considered to be 1%.

### **A-1.1.2 Toxicology**

Cadmium is transported in the blood and widely distributed in the body but accumulates primarily in the liver and kidneys (Goyer, 1991). Metabolic transformations of cadmium are limited to its binding to protein and nonprotein sulfhydryl groups, and various macromolecules, such as metallothionein, which is especially important in the kidneys and liver (ATSDR, 1989). Cadmium is excreted primarily in the urine.

#### **A-1.1.2.1 Short-term toxicity**

Acute oral exposures as low as 20 to 30 mg/kg body weight have caused fatalities in humans (Young, 1991), but fatal poisonings from cadmium are generally rare (ATSDR, 1989). Oral exposure to lesser amounts may result in gastrointestinal irritation, nausea, vomiting, salivation, abdominal pain, diarrhoea, vertigo and gastroenteritis (Klaassen, 1996; Fergusson, 1990; Lauwerys, 1979). Kidney damage has been observed in people who are exposed to large doses of cadmium either through inhalation or ingestion.

ATSDR (1989) identified the human health effects resulting from short-term cadmium ingestion (*i.e.*, less than or equal to 14 days) to be related primarily to gastrointestinal irritation at 0.05 to 0.5 mg/kg/day.

Exposure to cadmium may affect several organ systems, including the lungs, gastrointestinal tract and the liver. Symptoms involving the gastrointestinal tract may include nausea, vomiting, salivation, abdominal pain, diarrhoea, vertigo and gastroenteritis (Klaassen, 1996; Fergusson, 1990; Lauwreys, 1979). Inhalation of cadmium may produce an acute chemical bronchitis, pneumonitis and pulmonary oedema (Klaassen, 1996; Fergusson, 1990). Toxaemia of the liver may result from acute exposure to cadmium. Toxic effects resulting from cadmium exposure may be fatal.

#### **A-1.1.2.2 Long-term toxicity**

The long term effects of cadmium exposure may include hypertension, anaemia, prostate cancer chronic obstructive pulmonary disease, chronic kidney disease and effects on the cardiovascular and skeletal systems (Klaassen, 1996; Fergusson, 1990). Lung damage (*e.g.*, emphysema) has been observed in factory workers inhaling air with high cadmium concentrations (ATSDR, 1989).

The US EPA (2000) has classified cadmium as a probable human carcinogen (Group B1) for the inhalation route of exposure. Cadmium was not classifiable as to human carcinogenicity for the oral route of exposure (*i.e.*, Group D).

The evidence of a relationship between the incidence of prostate and lung cancer and exposure to cadmium appears to be weak. Furthermore, the negative mutagenicity and genotoxicity data for cadmium in *in vitro* and *in vivo* test systems indicate a non-genotoxic mechanism for cadmium carcinogenesis in the lung. This would suggest that the carcinogenicity of cadmium may act in a “threshold-response” carcinogenic mechanism (*i.e.*, tumorigenic growth does not occur until a certain threshold or dose is exceeded).

Long-term oral exposure to cadmium primarily affects the kidneys, resulting in tubular proteinosis. However, skeletal conditions such as “itai-itai” disease may also occur (Young, 1991).

Although reproductive and developmental effects have been observed in animals treated with cadmium, these effects have not been seen in human subjects (ATSDR, 1989).

#### ***A-1.1.3 Exposure Limit Used In The Current Assessment***

Considering the intended land use of the Polaris site once Cominco has decommissioned its mine on Little Cornwallis Island, an exposure limit should be protective of short-term durations of exposure to cadmium-contaminated soils.

Based on research completed by Lauwerys (1979) and Calabrese *et al.* (1997), the short-term exposure limit used in the current risk assessment was 4.3 µg/kg body weight/day. Lauwerys (1979) estimated the “no-effect” level of cadmium administered as a single oral dose to be 3 mg or approximately 43 µg/kg body weight (*i.e.*,  $3 \text{ mg} \div 70 \text{ kg adult} \approx 0.043 \text{ mg/kg}$  or 43 µg/kg). To ensure the protection of sensitive individuals within the potentially exposed population, a safety factor of 10 was applied to this dose, resulting in a short-term exposure limit of 4.3 µg/kg body weight/day. This limit also corresponds with the range of effects identified by ATSDR (1989) at various oral cadmium doses for short-term exposures (*i.e.*, up to 14 days). ATSDR (1989) estimated a “no-effect” level of ingested cadmium of slightly greater than 10 µg/kg body weight/day.

#### ***A-1.1.4 References***

- Ashmead, H.D., Graff, D.J. and Ashmead, H.H. 1985. In: Intestinal Absorption of Metal Ions and Chelates; Charles C. Thomas: Springfield, IL, pp 76-77. Cited In: Hamel *et al.* 1998.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1989. Toxicological profile for cadmium. ATSDR/U.S. Public Health Service, ATSDR/TP-88/08.
- Calabrese, E.J., Stankek, E.J., James, R.C., and Roberts, S. 1997. Soil ingestion: A concern for acute toxicity in children. *Environ Health Perspect* 105:1354-1358.
- Carson, B.L., Ellis III, H.V., and McCann, J.L. (Eds.) 1986. Toxicology and Biological Monitoring of Metals in Humans. Including Feasibility and Need. Lewis Publishers, Inc. Chelsea, MI.
- CEC. 1978. Criteria (Dose/Effect Relationships) for Cadmium. Commission of the European Communities. Rep Working Group of Experts Prepared for the Commission of the European Communities, Directorate-General for Social Affairs, Health and Safety Directorate, Pergamon Press.

- Elinder, C.G., Kjellstrom, T., Friberg, L., Lind, B., and Linnman, L. 1976. Cadmium in kidney cortex, liver, and pancreas from Swedish autopsies. *Arch Environ Health* 30:292.
- Goyer, R. 1991. Toxic effects of metals. In: *Casarett and Doull's Toxicology*. 4<sup>th</sup> edition, Pergamon Press, New York, pp 623-680.
- Fergusson, J.E. 1990. The Heavy Elements: Chemistry, Environmental Impact and Health Effects. Pergamon Press, Toronto. pp 548-551.
- Friberg, L., and Kjellstrom, T. 1981. Cadmium. *Disord Miner Metab* 317-352. Cited In: American Petroleum Institute, 1985.
- IARC. 1976. Cadmium, Nickel, Some Epoxides, Miscellaneous Industrial Chemicals and General Considerations on Volatile Anaesthetics. Volume 2. International Agency for Research on Cancer, Lyon, France.
- Kazantzis, G. 1987. Cadmium. In: Fishbein, L., Furst, A., and Mehlman, M.A. (Eds.) *Genotoxic and Carcinogenic Metals: Environmental and Occupational Occurrence and Exposure*. Volume XI. Princeton Scientific Publishing Co., Inc., Princeton, NJ.
- Klaassen, C.D. 1996. *Casarett & Doull's Toxicology: The Basic Science of Poisons*. Fifth Edition. Eds: C.D. Klaassen, M.O. Amdur and J.Doull. McGraw-Hill, Toronto. P699.
- Lauwerys, R. 1979. Cadmium in man. In: Webb, N. (ed.) *The chemistry, biochemistry, and biology of cadmium*. Elsevier/North Holland Biomedical Press, pp 433-453.
- Leonard, A., Gerber, G.B., Jacquet, P., and Lauwerys, R.R. 1984. Carcinogenicity, Mutagenicity, and Teratogenicity of Industrially Used Metals. In: Kirsch-Volders, M. (Ed.) *Mutagenicity, Carcinogenicity, and Teratogenicity of Industrial Pollutants*. Plenum Press, NY, pp. 59-126.
- McLellan, J.S., Flanagan, P.R., Chamberlain, M.J., and Valberg, L.S. 1978. Measurement of dietary cadmium in humans. *J Toxicol Environ Health* 4:131.
- NAS. 1977. *Drinking Water and Health*. National Academy of Science, National Academy Press, Washington, DC.
- Norberg, G.F., T. Kjellstrom and M. Nordberg 1985. Kinetics and metabolism. In: Friberg, L., C.G. Elinder, T. Kjellstrom and G.F. Norberg, Eds. *Cadmium and Health: A toxicological and epidemiological appraisal*. Vol. 1. Exposure, dose and metabolism. CRC Press, Boca Raton, Fl., pp 103-178.
- Rahola, T., Aaran, R.K., and Miettinen, J.K. 1972. Half-time Studies of Mercury and Cadmium by Whole-body Counting. In: *Assessment of Radioactive Contamination in Man*. Symposium on Assessment of Radioactive Organ and Body Burdens, Stockholm, 1971.

International Atomic Energy Agency Proceedings Series, Vienna, pp. 553-562. IAEA Publication 290.

Skog, E., and Wahlberg, J.E. 1964. A comparative investigation of the percutaneous absorption of metal compounds in the guinea pig by means of radioactive isotopes. *J Invest Derm* 43:187.

Young, R.A. 1991. Toxicity Summary for Cadmium. Prepared for: Oak Ridge Reservation Environmental Restoration Program.

US EPA. 1980. Ambient Water Quality Criteria: Cadmium. (U.S.) Environmental Protection Agency, p. 177. NTIS PB-292 423/1GA. Cited In: NCI, 1985.

US EPA. 2000. Integrated Risk Information System (IRIS). US Environmental Protection Agency (US EPA). Home Page.

Wahlberg, J.E. 1965. Percutaneous toxicity of metal compounds - A comparative investigation in guinea pigs. *Arch Environ Health* 11:201.

Westar, R., Maibach, H., Sedik, L., Melendres, J., Dizio, S., and Wade, M. 1992. In vitro percutaneous absorption of cadmium from water and soil into human skin. *Fund Appl Toxicol* 19:1-5.

WHO. 1984. Guideline for Drinking-Water Quality. Health Criteria and Other Supporting Information. Volume 2. World Health Organization, Geneva.

## **A-1.2            Lead**

Lead toxicity to humans varies with the age and circumstances of the exposed individual, with the major risk related to possible toxic impact to the nervous system. The most susceptible populations or sensitive receptors are children, particularly toddlers, infants in the neonatal period, as well as the developing foetus (Goyer, 1996).

### **A-1.2.1            Bioavailability**

#### **A-1.2.1.1        Oral route of exposure**

In clinical studies, adults have been found to absorb approximately 10 to 15% of their total oral lead intake (Kehoe, 1961; Thompson, 1971; Karhausen, 1973; Blake, 1976; Chamberlain *et al.*, 1978). In a recent review, Mushak (1991) reported that small children and infants absorb as much as 42 to 53% of their lead intake (Karhausen, 1973; Alexander, 1974; Ziegler *et al.*, 1978). To ensure that oral exposure to lead resulting from the incidental ingestion of surface soils was not underestimated, the fraction of ingested lead absorbed was assumed to be 53% for children.

#### **A-1.2.1.2 Inhalation route of exposure**

In the past, the average Canadian adult had been reported to absorb approximately 31 to 38 µg lead/day, of which 30 to 45% was attributed to inhalation (NRCC, 1978). The World Health Organization (WHO, 1984) estimated that approximately 20 to 60% of the total amount of lead inhaled is deposited in the lung, with the majority of this being absorbed. Kehoe (1961) and Gross (1981) obtained respiratory system deposition rates for humans of 30 to 70% (mean of 48%) while Nozaki (1966), Chamberlain *et al.* (1975), Morrow *et al.* (1980), and Hammond *et al.* (1981) cited similar values of 30 to 50%. It is estimated that most of the lead deposited in the pulmonary region is either absorbed or cleared, since autopsies on human lungs have shown that very little lead is accumulated in the lung (Barry, 1975). Specific absorption values of lead from the respiratory system of children were not available, but Barltrop (1972) estimated that children inhale 40% more lead than adults. The actual lead deposition rate in 10-year-old children was given by James (1978) as 1.6 to 2.7 times that of adults, based on a number of modifying factors, including the differences in metabolic rates and airway dimensions. The percentage of inhaled lead swallowed following clearance from the respiratory system from the action of the mucociliary apparatus was cited by Chamberlain *et al.* (1975) as 6%.

For the purpose of the current exposure assessment, lead was assumed to be adsorbed to airborne particles or adsorbed to soil particles that have become suspended in the air. Therefore, the bioavailability of lead following inhalation would be determined by the dynamics of deposition, retention and clearance of airborne particles by the respiratory system. Approximately 13% of airborne particles would be retained within the respiratory system, while 60% of airborne particles would be cleared from the respiratory system and swallowed. In children, if 42 to 53% of the lead on the swallowed particulates were absorbed from the gastrointestinal tract, then 25.2 to 31.8% of airborne lead would be bioavailable by this route (42 of 60%; 53 of 60%). Assuming 100% bioavailability for the retained fraction of lead, 13% of inhaled lead would be absorbed by the respiratory system. Therefore, for children the total fraction of airborne lead that would be bioavailable following inhalation would be 38.2 to 44.8% (13% from the respiratory tract plus 25.2 to 31.8% from the gut). To ensure that inhalation exposure to lead was not underestimated, the current assessment used an inhalation bioavailability of 44.8 %.

#### **A-1.2.1.3 Dermal route of exposure**

The absorption of inorganic lead compounds through the skin was considered insignificant compared to that occurring from either inhalation or ingestion. Skin absorption of lead acetate was estimated to approach 0.06% (Moore *et al.*, 1980) while later studies reported up to 5% when in aqueous solution and up to 8% when in organic form as tetraethyl lead. An absorption fraction of 0.06% was selected for the exposure assessment for lead. This estimate is considered appropriate in that the lead at the site will not be in the form of soluble salts, as free ion, or as an organic compound, but will be intrinsically bound within a soil-like matrix and thus would be minimally absorbed.



### **A-1.2.2 Toxicology**

#### **A-1.2.2.1 Short-term toxicity**

The primary concern with short-term exposure via soil ingestion is the possible large increase in blood-lead level which may result in potential clinical effects such as:

- Gastrointestinal distress
- Weight loss
- Tremors
- Irritability
- Lethargy

Symptoms of acute exposure to lead may also include: nausea, vomiting, abdominal pains, anorexia, anaemia, co-ordination loss, restlessness, hyperactivity, confusion and impairment of memory (Fergusson, 1990).

The “benchmark” probability that US EPA uses to guide remediation decisions is a less than 5% probability that an individual’s blood lead level will exceed 10 µg/dL for long-term, chronic exposure periods (White *et al.*, 1998). However, this target blood lead level is more appropriate for long-term, chronic exposures and effects (*e.g.*, effects on cognitive abilities). Sensitive receptors such as Inuit children are only expected to visit the Mine site for perhaps one to three days per year (B.C. Research, 1975; LaVigne, 1980; results from community consultation 2000). This type of short-term or acute exposure period suggests a short-term toxicological impact. Ingestion of soil while visiting the Mine site could result in larger, temporary increases in blood lead levels, possibly giving rise to the described clinical effects. For the purposes of this risk assessment, a more relevant short-term “benchmark” of 20 µg/dL was used as a target blood lead level (CDC, 1991; Health Canada, 1994).

#### **A-1.2.2.2 Long-term toxicity**

The toxic effects of lead involve several organ systems and biochemical activities, however the most sensitive system is the nervous system. For adults with excess occupational exposure, or even accidental exposure, the concerns are peripheral neuropathy and/or chronic nephropathy (Klaassen, 1996). Other effects of long term exposure may include interference with haem synthesis (Fergusson, 1990).

The threshold PbB concentration for lead-induced elevation of FEP is in the range of 25 to 30 µg/dL whole blood in women and children (Nutrition Foundation, 1982) and in adult males. Studies of children living in proximity to a lead smelter indicated an apparent threshold of 60 µg/dL or greater for effects on FEP (McNeil *et al.*, 1975; Landrigan *et al.*, 1976). PbB concentrations are directly related to anaemia resulting from impairment of haem synthesis and acceleration of red blood cell destruction. The threshold for this effect in children has been reported to be 50 µg/dL (Tsuchiya, 1979; Nutrition Foundation, 1982).

Decreased IQ values among children with PbB concentrations from 5.6 to 25 µg/dL have been reported (Yule *et al.*, 1981; Fulton *et al.*, 1987; Hatzakis *et al.*, 1987). Cooney *et al.* (1989) reported that at PbB concentrations of 0.25 µg/dL, there were no adverse effects on neurobehavioral development. Several authors suggest a LOAEL of 10 to 15 µg/dL perinatal PbB concentrations (Wolf *et al.*, 1985; Bellinger *et al.*, 1987; Dietrich *et al.*, 1987; Wigg *et al.*, 1988).

Bellinger *et al.*, (1987) conducted a prospective cohort study of children (n=249) living in the Boston area, from birth to 2 years of age. While postnatal exposure was not associated with detrimental effect, prenatal exposure was reported to impair early cognitive development assessed using the Bayley Mental Development Index (MDI). In a follow-up to this Boston perspective study, Bellinger *et al.* (1991) found that prenatal PbB concentrations  $\geq 10$  µg/dL in cord blood was associated with a slower cognitive development in children up until at least 2 years of age. After 57 months of age, however, prenatal exposure was not related to intelligence tests results.

Based on a study to assess the effects of chronic low to moderate foetal lead exposure in lead-hazardous areas of Cincinnati (n=305), Dietrich *et al.* (1987) considered that there was a direct relationship between prenatal, umbilical and new-born PbB concentrations and deficits on the Bayley MDI at either 3 or 6 months. Male infants and infants from the poorest families were especially sensitive to lead. However, once the regression analysis was adjusted for confounding variables, no significant effects of foetal lead exposure on Bayley Psychomotor Developmental Index (PDI) were found. Further study suggested that the neurobehavioral deficits were partly mediated by lead-related reductions in birth weight and gestation. However, in a further follow-up study of the Cincinnati lead study cohort, Dietrich *et al.* (1993) found that even after adjustments were made for covariates, there remained a statistical significance between postnatal lead blood concentrations and Performance IQ when compared to children with mean blood concentrations (<10 µg/dL).

Studies of the effect of environmental exposure to lead were conducted on children born near a lead smelter, near Port Pirie in South Australia. Results demonstrated that child development at ages 2, 3 and 4 appeared to be inversely related to postnatal PbB concentrations based on the McCarthy Scales of Children's Abilities (McMichael *et al.*, 1988). Reductions in perceptual performance and memory scores were also reported. In a follow-up to the McMichael *et al.* (1988) cohort study, an increase in PbB concentration from 10 to 30 µg/dL was shown to cause a decrease in General Cognitive Index score on the McCarthy Scales (combines scores for verbal, perceptual-performance, and memory and motor performance subscales) for children (McMichael *et al.*, 1992). However, no significant differences in children's abilities with respect to blood concentrations of lead and mental development effects, were observed when considering different environmental modifying factors

It was noted in a European multi-center study on lead neurotoxicity in children by the World Health Organization, Regional Office for Europe (WHO/EURO) and the Commission of the European Communities (Winneke *et al.*, 1990) that neurobehavioral effects of environmental lead exposure in children represent weak signals in a noisy background and consequently many published cross-sectional studies suffer from insufficient power to detect subtle effects. School-

aged children (with PbB concentrations of <5 to 50 µg/dL) were found to have detectable exposure-related behavioural and cognitive effects. Based on this study, no threshold could be identified from the data for neurotoxicity in school-aged children.

A report to the U.S. Congress on childhood lead poisoning in the U.S. (Mushak *et al.*, 1989) indicates that PbB concentrations of 10 to 15 µg/dL are associated with many effects, including alterations in neurobehavioral development and electrophysiological function, disturbances in haem biosynthesis and deficits in growth and maturation, may occur both prenatally and later in childhood.

#### **A-1.2.2.3 Baseline blood-lead level**

Discussion with Charles Wood (program leader for blood-Pb program) at the State of Alaska Epidemiology Group (State Health Department):

The state of Alaska has an active, ongoing blood lead screening effort in some of the northern most regions of the state (*e.g.*, Barrow Village, Fairbanks, Fort Yukon). Twenty-two children aged 1 to 2 years (included Inuit children, but participants were not differentiated by race) were screened for blood Pb levels. Results from this program indicated that no children had blood lead levels in excess of 10 µg/dL, and that both the arithmetic and geometric means were below 4 µg/dL.

#### **A-1.2.3 Exposure Limit Used In The Current Assessment**

Based on the nature of the intended use of the site, the benchmark blood-lead level considered most appropriate for the current assessment was 20 µg/dL. Inuit children in the area are not expected to have a baseline blood-lead level that exceeds 4 µg/dL. This translates into an “allowable” daily intake of lead of 400 µg for a child for brief durations of time.

#### **A-1.2.4 References**

- Alexander, F.W. 1974. The uptake of lead by children in differing environments. *Environ Health Perspect* 7:155-159.
- Barltrop, D. 1972. Transfer of Lead to the Human Foetus. In: Barltrop, D., and Burland, W.L. (Eds.) *Mineral Metabolism in Pediatrics*. Philadelphia, PA. Davis Co. pp. 135-151.
- Barry, P.S.I. 1975. A comparison of concentrations of lead in human tissues. *Brit J Ind Med* 32:119-139.
- B.C. Research 1975. Environmental Study of Polaris Mine, Little Cornwallis Island. Submitted to Cominco Ltd., Project No. 1642. Prepared by Division of Applied Biology, B.C. Research.
- Bellinger, D., Leviton, A., Waternaux, C., Needleman, H., and Rabinowitz, M. 1987. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. *N Engl J Med* 316(17):1037-1043.

- Bellinger, D., Sloman, J., Rabinowitz, M., Needleman, H.L., and Waternaux, C. 1991. Low-level lead exposures and children's cognitive function in the preschool years. *Pediatrics* 82(2). Cited In: Volpe *et al.*, 1992.
- Blake, K.C.H. 1976. Absorption of <sup>203</sup>Pb from gastrointestinal tract of man. *Environ Res* 11:1-4.
- CDC (U.S. Centre for Disease Control and Prevention), 1991. Preventing lead poisoning in young children. U.S. Department of Health and Human Services, Public Health Service. Available online at <http://www.cdc.gov/nceh/pubcatns/1994/cdc/books/plpsn/plpsn7.htm#sum>
- Chamberlain, A.C., Clough, W.S., Heard, M.J., Newton, D., Stott, A.N.B., and Wells, A.C. 1975. Uptake of lead by inhalation of motor exhaust. *Proc R Soc Lond Ser B Biol Sci* 192:77. Cited In: HWC, 1980.
- Chamberlain, A.C., Heard, M.J., Little, P., Newton, D., Wells, A.C., and Wiffen, R.D. 1978. Investigations into Lead From Motor Vehicles. United Kingdom Atomic Energy Authority, Harwell, United Kingdom. Report No. AERE-R9198.
- Cooney, G.H., Bell, A., McBride, W., and Carter, C. 1989. Low-level exposures to lead: The Sydney lead study. *Develop Med Child Neurol* 31(1):640-649.
- Dietrich, K.N., Krafft, K.M., Bornschein, R.L., Hammond, P.B., Berger, O., Succop, P.A., and Bier, M. 1987. Low-level foetal lead exposure effect on neurobehavioral development in early infancy. *Pediatrics* 80(5):721-730.
- Dietrich, K.N., Berger, O.G., Succop, P.A., Hammond, P.B., and Bornschein, R.L. 1993. The developmental consequences of low to moderate prenatal and postnatal lead exposure: Intellectual attainment in the Cincinnati lead study cohort following school entry. *Neurotoxicol Teratol* 15:37-44.
- Fergusson, J.E. 1990. *The Heavy Elements: Chemistry, Environmental Impact and Health Effects*. Pergamon Press, Toronto. pp 551-553.
- Fulton, M., Raab, G., Thomson, G., Laxen, G., Hunter, D., and Hepburn, W. 1987. Influence of blood lead on the ability and attainment of children in Edinburgh. *Lancet* 8544:1221-1226. Cited In: Davis *et al.*, 1990.
- Goyer, R.A. 1996. Toxic Effects of Metals. In: Casarett and Doull's Toxicology: The Basic Science of Poisons. C.D. Klaassen, M.O. Amdur, J. Doull, Eds. Fifth Edition. McGraw-Hill, Toronto, Chapter 23.
- Gross, S.B. 1981. Human oral and inhalation exposures to lead: summary of Kehoe balance experiments. *J Toxicol Environ Health* 8:333-337.

- Hammond, P.B., O'Flaherty, E.J., and Gartside, P.S. 1981. The impact of air-lead on blood-lead in man - a critique of the recent literature. *Food Cosmet Toxicol* 19:631-638.
- Hatzakis, A., Kokkevi, A., Katsouyanni, K., Maravelias, K., Salaminios, F., Kalandidi, A., Koutselinis, A., Stefanis, K., and Trichopoulos, D. 1987. Psychometric intelligence and attentional performance deficits in lead-exposed children. *Heavy Metal Environ* 1:204-209.
- Health Canada, 1994. Uptake of evidence for low-level effects of lead and blood lead intervention levels and strategies – Final report of the Working Group. Federal-Provincial Committee on Environmental and Occupational Health, Environmental Health Directorate, Ottawa, Ontario.
- James, A.C. 1978. Lung Deposition of Sub-Micron Aerosols Calculated as a Function of Age and Breathing Rate. In: National Radiological Protection Board Annual Research and Development Report. National Radiological Protection Board, Harwell, United Kingdom, pp. 71-75.
- Karhausen, L. 1973. Intestinal Lead Absorption. In: International Symposium, Environmental Health Aspects of Lead. Commission of European Communities, C1D, Luxembourg, pp. 427-440. Cited In: HWC, 1980.
- Kehoe, R.A. 1961. The metabolism of lead in man in health and disease. In: The Harben Lectures (1960). *J R Inst Public Health Hyg* 24:1.
- Klaassen, C.D. 1996. Casarett & Doull's Toxicology: The Basic Science of Poisons. Fifth Edition. Eds: C.D. Klaassen, M.O. Amdur and J.Doull. McGraw-Hill, Toronto. P705.
- Landrigan, P.J., *et al.* 1976. Increased lead absorption with anaemia and slowed nerve conduction in children near a lead smelter. *J Pediat* 89:904-910. Cited In: Nutrition Foundation, 1982.
- LaVigne, M. 1980. Potential Socio-Economic Impacts of the Polaris Mine Project. Prepared by Outcrop Ltd. (Yellowknife) in association with DPA Consulting Ltd. (Vancouver).
- McMichael, A.J., Baghurst, P.A., Wigg, N.R., Vimpani, G.V., Robertson, E.F., and Roberts, R.J. 1988. Port Pirie cohort study: Environmental exposure to lead and children's abilities at the age of four years. *N Engl J Med* 319(8):468-475.
- McMichael, A.J., Baghurst, P.A., Vimpani, G.V., Robertson, E.F., Wigg, N.R., and Tong, S. 1992. Sociodemographic factors modifying the effect of environmental lead on neuropsychological development in early childhood. *Neurotoxicol Teratol* 14:321-327.

- McNeil, *et al.* 1975. Evaluation of long-term effects of elevated blood lead concentrations in asymptomatic children. *Arch Ind Hyg Toxicol* 26:97-118. Cited In: Nutrition Foundation, 1982.
- Moore, M.R., Meredith, P.A., Watson, W.S., Sumner, D.J., Taylor, M.K., and Goldberg, A. 1980. The percutaneous absorption of lead-203 in humans from cosmetic preparations containing lead acetate, as assessed by whole-body counting and other techniques. *Food Cosmet Toxicol* 18:399-405.
- Morrow, P.E., Beiter, H., Amato, F., and Gibb, F.R. 1980. Pulmonary retention of lead: An experimental study in man. *Environ Res* 21:373-384.
- Mushak, P. 1991. Gastro-intestinal absorption of lead in children and adults: Overview of biological and biophysico-chemical aspects. *Chem Speciation Bioavailab* 3(3/4):87-104.
- Mushak, P., Davis, J.M., Crocetti, A., and Grant, L. 1989. Prenatal and postnatal effects of low-level lead exposure: Integrated summary of a report to the U.S. Congress on childhood poisoning. *Environ Res* 50:11-36.
- Nozaki, K. 1966. Method for studies on inhaled particles in human respiratory system and retention of lead fume. *Ind Health* 4:118. Cited In: HWC, 1980.
- NRCC. 1978. Effects of Arsenic in the Canadian Environment. National Research Council Canada, Associate Committee on Scientific Criteria for Environmental Quality, Subcommittee on Heavy Metals and Certain Other Compounds. NRCC No. 15391.
- Nutrition Foundation 1982. Assessment of the Safety of Lead and Lead Salts in Food. A Report of the Nutrition Foundation's Expert Advisory Committee, June, 1982. Nutrition Foundation, Washington, DC.
- Thompson, J.A. 1971. Balance between intake and output of lead in normal individuals. *Brit J Ind Med* 28:189-194.
- Tsuchiya, K. 1979. Lead. Freiberg *et al.* (Eds). In: *Handbook on the Toxicology of Metals*. Elsevier/North-Holland Biomedical Press, pp. 451-84.
- WHO. 1984. Guidelines for Drinking-Water Quality. Health Criteria and Other Supporting Information. Volume 2. World Health Organization, Geneva.
- White, P.D. *et al.* 1998. The conceptual structure of the integrated exposure uptake biokinetic model for lead in children. *Environ. Health Persp.*, 106: 1513-1530.
- Wood, C., 2000. Alaska State Health – Epidemiology Department (Head of lead task force). Personal Communication.



- Wigg, N.R., Vimpani, G.V., McMichael, A.J., Baghurst, P.A., Robertson, S.F., and Roberts, R.J. 1988. Port Pirie cohort study: Childhood blood lead and neuropsychological development at age two years. *J Epidemiol Community Health* 42:213-219.
- Winneke, G., Brockhaus, A., Ewers, U., Kramer, U., and Neuf, M. 1990. Results from the European multicenter study on lead neurotoxicity in children: Implications for risk assessment. *Neurotoxicol Teratol* 12:553-559.
- Wolf, A.W., Ernhart, C.B., and White, C.S. 1985. Intrauterine Lead Exposure and Early Development. In: Lekkas, T.D. (Ed.) *International Conference: Heavy Metals in the Environment*. CEP Consultants, Ltd. Edinburgh, pp. 153-155. Cited In: Davis *et al.*, 1990.
- Yule, W., Lansdown, R., Millar, I.B., and Urbanowicz, M.A. 1981. The relationship between blood lead concentrations, intelligence and attainment in a school population: a pilot study. *Develop Med Child Neurol* 23:567-576.
- Ziegler, E.E., Edwards, B.B. Jensen, R.L., Mahaffey, D.R., and Foman, S.J. 1978. Absorption and retention of lead by infants. *Pediatr Res* 12:29-34. Cited In: Muskak, 1991.

### **A-1.3            Zinc**

#### **A-1.3.1           Bioavailability**

##### **A-1.3.1.1        Oral exposure route**

Absorption of zinc from dietary sources reportedly ranged from 10 to 40% (Solomons, 1982). Synder *et al.* (1975) suggests that 35% is the best value pertaining to the bioavailability of zinc from food sources. For the current assessment, the oral bioavailability of zinc was conservatively assumed to be 40%.

##### **A-1.3.1.2        Inhalation exposure route**

In a study with humans, zinc oxide fumes and powder were reported to be deposited in the alveoli. Increased concentrations of zinc in serum and plasma indicated pulmonary absorption (Sturgis *et al.*, 1927, Drinker *et al.*, 1927a). Based on airborne particle dynamics in the respiratory system, about 13% of airborne zinc on particles would be bioavailable through the lung. Approximately 60% of airborne particles would be cleared by the mucociliary apparatus and swallowed. If 10 to 40% of the swallowed zinc were absorbed from the gastrointestinal tract, then an additional 6 to 24% (10% of 60%; 40% of 60%) of airborne zinc would be bioavailable by this route. Therefore, based on airborne particle dynamics, the total bioavailability of airborne zinc from particles would be 19 to 37% (13% from the respiratory system plus 6 to 24% of the cleared material). To ensure that exposure resulting from inhalation of zinc was not underestimated, an inhalation bioavailability of 37% was used for the current assessment.

### **A-1.3.1.3 Dermal exposure route**

Studies of dermal exposure to zinc are very limited. Stokinger (1981) reported that  $\approx 2.5\%$  of dermally applied zinc was absorbed through the skin in various species (not specified). For the current exposure assessment, a value of 2.5% was selected to represent the dermal absorption fraction of zinc.

### **A-1.3.2 Toxicology**

Zinc is considered to be a relatively non-toxic metal and is an essential element in the diet of humans for normal growth and development (NRC, 1980). Zinc toxicity from excessive ingestion is uncommon, but gastrointestinal distress and diarrhoea have been reported following ingestion of beverages standing in galvanised cans or from use of galvanised utensils (Klaassen, 1996). In cases of low dietary copper, there is potential for zinc toxicity in the form of hypocupraemia (NRC, 1980). The Canadian recommended dietary intake of zinc varies between 4 and 10 mg/day depending on the age of the individual (Health and Welfare Canada, 1980). In the United States, the recommended minimum daily intake level is 10 mg/day for children and 15 mg/day for adults (Health and Welfare Canada, 1980). Much evidence exists demonstrating that zinc deficiency, both acute and chronic, is a much larger and more serious risk to the human population (Henkin *et al.*, 1975) than any environmental exposure to excess zinc (Walsh *et al.*, 1994).

#### **A-1.3.2.1 Short-term toxicity**

Metal fume fever, an acute disease, causes temporary pulmonary function impairment but does not lead to chronic lung disease (Drinker *et al.*, 1927b; Brown, 1988; Malo *et al.*, 1990). The symptoms of this fever include dryness and throat irritation, flu-like symptoms such as chills, muscle and joint aches, weakness, sweating and high fever (Daig and Challen, 1964), as well as coughing, fatigue and shortness of breath (Rom, 1983). Other reported symptoms are reduced lung volumes, a decreased capacity of CO and an increase in bronchiolar leukocytes (Vogelmeier *et al.*, 1987; Malo *et al.*, 1990) with symptoms for this fever usually disappearing within 1 to 4 days (Drinker *et al.*, 1927b; Sturgis *et al.*, 1927; Brown, 1988). Many authors have reported “metal fume fever” in humans acutely exposed to zinc chloride or zinc oxide at 600 mg/m<sup>3</sup> for 5 hours (Daig and Challen, 1964; Key *et al.*, 1977; Rom, 1983). An experimental study by Gordon *et al.* (1992), demonstrated that all 4 subjects exposed to ultrafine zinc oxide particles at the current TLV of 5 mg/m<sup>3</sup> (ACGIH, 1997) for 2 hours *via* inhalation, experienced metal fume fever symptoms. These symptoms were absent after 24 hours, and are the predominant effect of inhalation exposure to zinc oxide (Gordon *et al.*, 1992).

#### **A-1.3.2.2 Sub-Chronic and Chronic Toxicity**

In a study by Samman and Roberts, (1987), 47 volunteers were given 150 mg of zinc sulphate daily as 2 mg zinc/kg/day for 6 weeks. Out of the 26 people, 47 experienced symptoms such as headache, nausea, stomach cramps and diarrhoea. NAS (1980) reported that patients treated with



150 mg Zn/day, showed no adverse effects. However, elevated chronic zinc intakes coupled with low dietary copper concentrations may reduce copper absorption and hence, produce hypocupraemia. Long-term oral administration of zinc supplements caused anaemia in humans (Hale *et al.*, 1988; Broun *et al.*, 1990; Gyorffy and Chan, 1992) and so did zinc sulphate exposure at 2 mg zinc/kg/day for 10 months (Hoffman *et al.*, 1988). Several case studies have been reported involving sickle cell anaemia patients taking 150 mg Zn/day (equivalent to 10 times the U.S. recommended daily adult intake) for treatment of ulcerations and sickling of red blood cells (US EPA, 1984). In general, no toxicity was observed over periods of 3 to 24 months depending on the individual study. If any toxicity was noted (*i.e.*, microcytic anaemia, neutropenia and hypocupraemia), it was reversed with the reduction of zinc intake and copper supplementation.

Haematological effects including decreased hematocrit, serum ferritin, and erythrocyte superoxide dismutase activity were observed in women given daily supplements of 50 mg of zinc as zinc gluconate (0.83 mg/kg/day) for 10 days (Yadrick *et al.*, 1989).

There are no reports that specifically look at the carcinogenicity of zinc compounds in humans (US EPA, 1998). US EPA (1998) concluded that zinc is not classifiable as to human carcinogenicity based on inadequate evidence in both humans and animals.

#### ***A-1.3.3 Exposure Limit***

ATSDR (1997) has reported an oral intermediate level (*i.e.*, 15 to 364 days) called a minimum risk level (MRL) for zinc of 0.3 mg/kg/day based on the study by Yadrick *et al.*, (1989). The chronic oral MRL was derived from the sum of the reported supplemental dose of 0.83 mg/kg/day in Yarick *et al.* and the dietary estimate from the FDA Total Diet Study (for 1982-1986) of 0.16 mg/kg/day (Pennington *et al.*, 1989) for a total dose of 1 mg/kg/day. The total dose was then divided by an uncertainty factor of 3 based on minimal LOAEL from a study of the most sensitive humans and the consideration that zinc is an essential dietary nutrient (TERA, 2000)

An RfD of 0.3 mg/kg/day (300 µg/kg/day) for soluble zinc salts was also established by US EPA (1998) based on the human diet supplement study by Yadrick *et al.* (1989) study. An uncertainty factor of 3 was applied to the LOAEL of 1.0 mg/kg/day due to the short duration of the study and the fact that zinc is an essential nutrient. This RfD was reviewed and agreed upon by both nutritionists and toxicologists (US EPA, 1998), but is based on soluble salts, as opposed to less soluble forms (such as those bound to particulate). In addition, while it was set to allow for adequate zinc intake in adolescents and adults, it may not supply the recommended daily allowance to groups requiring greater zinc intake at certain stages in their lives (*i.e.*, lactating women, infants, *etc.*) (US EPA, 1998). As a result, this RfD may be overly conservative for the current assessment.

The Health Canada provisional tolerable daily intake of 0.3 mg/kg/day (300 µg/kg/day) was conservatively used in this assessment as a short-term exposure limit for zinc (Health Canada, 1996). This happens to also equal the oral intermediate level recommended by ATSDR (1997) and is the same RfD value recommended by the US EPA (1998). It is recognised that this likely overestimates the potency of zinc on an acute basis; however, at this time no further information was readily available.

#### **A-1.3.4      References**

- ACGIH. 1997. Threshold Limit Values for Chemical Substances and Physical Agents. Biological Exposure Indices. 1997 TLVs® and BEIs®. American Conference of Governmental Industrial Hygienists Inc., Cincinnati, OH.
- ATSDR, 1997. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Cobalt. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.
- Broun, E.R., Greist, A., Tricot, G. *et al.* 1990. Excessive zinc ingestion: A reversible cause of sideroblastic anaemia in saline waters by atomic-absorption spectrophotometry. *Talanta* 14:809-816. Cited In: ATSDR, 1997.
- Brown, J.J. 1988. Zinc fume fever. *Br J Radiol* 61:327-329. Cited In: ATSDR, 1997.
- Carson, B.L., Ellis (III), H.V., and McCann, J.L. (Eds). 1987. Toxicology and Biological Monitoring of Metals in Humans. Including Feasibility and Need. Lewis Publishers, Inc. Chelsea, MI.
- Daig, A.T., and Challen, P.J.R. 1964. Respiratory hazards in welding. *Ann Occup Hyg* 7:223-231. Cited In: Walsh *et al.*, 1994.
- Drinker, P., Thomson, R.M., and Finn, J.L. 1927a. Metal fume fever: IV. Threshold doses of zinc oxide, preventive measures, and the chronic effects of repeated exposures. *J Ind Hyg* 9:331-345. Cited In: ATSDR, 1997.
- Drinker, K.R., Thompson, P.K., and Marsh, M. 1927b. An investigation of the effect upon rats of long-continued ingestion of zinc compounds, with a special reference to the relation of zinc excretion to zinc intake. *Am J Physiol* 81:284-306.
- Gordon, T., Chen, L.C., Fine, J.M., Schlesinger, R.B., Su, W.Y., Kimmel, T.A., and Amdur, M.O. 1992. Pulmonary effects of inhaled zinc oxide in human subjects, guinea pigs, rats and rabbits. *Am Ind Hyg Assoc J* 53(8):503-509.
- Gyorffy, E., and Chan, H. 1992. Copper deficiency and microcytic anaemia resulting from prolonged ingestion of over the counter zinc. *Am J Gastroenterology* 87(8):1054-1055. Cited In: ATSDR, 1997.

- Hale, W.E., May, F.E., Thomas, R.G. *et al.* 1988. Effect of zinc supplementation on the development of cardiovascular disease in the elderly. *J Nut Elder* 8(2):49-57. Cited In: ATSDR, 1997.
- Health Canada. 1996. Health-based tolerable daily intakes / Concentrations and tumorigenic doses / Concentrations for priority substances. Environmental Health Directorate, Health Protection Branch, Health Canada (HC), Ottawa. ISBN 0-662-24858-9.
- Health and Welfare Canada. 1980. Guidelines for Canadian Drinking Water Quality. Health and Welfare Canada. Supporting Documentation, Ottawa, ON.
- Henkin, R.I., Patten, B.M., Re, P.K., and Bronzert, D.A. 1975. A syndrome of acute zinc loss. Cerebellar dysfunction, mental changes, anorexia, and taste and smell dysfunction. *Arch Neurol* 32:745-751. Cited In: Walsh *et al.*, 1994.
- Hoffmann, H.N. (II), Phyliky, R.L., and Fleming, C.R. 1988. Zinc-induced copper deficiency. *Gastroenterology* 94:508-512. Cited In: ATSDR, 1997.
- Klaassen, C.D. 1996. Casarett & Doull's Toxicology: The Basic Science of Poisons. Fifth Edition. Eds: C.D. Klaassen, M.O. Amdur and J.Doull. McGraw-Hill, Toronto. P721.
- Key, M.M., Henschel, A.F., Butler, J., Ligo, R.N., and Tabershaw, I.R. 1977. Occupational diseases: A guide to their recognition. U.S. Department Health Education, Welfare. Washington, DC. Pp 408-410. Cited In: Walsh *et al.*, 1994.
- Malo, J-L., Malo, J., Cartier, A. *et al.* 1990. Acute lung reaction due to zinc inhalation. *Eur Res K* 3:111-114. Cited In: ATSDR, 1997.
- NAS. 1980. Recommended Dietary Allowances. Ninth Edition. National Academy of Sciences, Printing and Publishing Office, Washington, DC. Cited In: Carson *et al.*, 1987.
- NRC. 1980. Drinking Water and Health. Volume 3. National Research Council. Board on Toxicology and Environmental Health Hazards. National Academy Press, Washington, DC.
- Pennington, J.A.T., Young, B.E., and Willson, D. 1989. Nutritional elements in US diets: Results from the total diet study, 1982 to 1986. *J Am Diet Assoc* 89(5):659-664. Cited In: IRIS 1993 and TERA 2000.
- Rom, W.N. 1983. Environmental and Occupational Medicine. Little Brown and Co., Boston, MA. Cited In: Walsh *et al.*, 1994.
- Samman, S., and Roberts, D.C.K. 1987. The effects of zinc supplements on plasma zinc and copper levels and the reported symptoms in healthy volunteers. *Med J Australia* 146:246-249. Cited In: Walsh *et al.*, 1994.

- Solomons, N.W. 1982. Biological availability of zinc in humans. *Am J Clin Nut* 35:1048-1075.  
Cited In: Carson *et al.*, 1987.
- Stokinger, H.E. 1981. The Metals. In: Clayton, G.D., and Clayton, F.E. (Eds.) *Patty's Industrial Hygiene and Toxicology*, Third Edition Volume 2A. John Wiley and Sons, Inc., New York, NY.
- Sturgis, C.C., Drinker, P., and Thomson, R.M. 1927. Metal fume fever: I. Clinical observations on the effect of the experimental inhalation of zinc oxide by two apparently normal persons. *J Ind Hyg* 9:88-97.
- Snyder, W.S., Cook, M.J., Nassett, E.S., Karhausen, L.R., Howells, G.P., and Tipton, I.H. 1975. International Commission of Radiological Protection. Report of the task group on reference man, New York, NY. ICRP Publication 23. Cited In: Carson *et al.*, 1987.
- TERA (Toxicology Excellence for Risk Assessment). 2000. Noncancer Oral Risk Values – More Information. <http://www.tera.org>.
- US EPA. 1984. Health Effects Assessment for Zinc (and Compounds). (U.S.) Environmental Protection Agency, Cincinnati, OH. NTIS PB86-134590.
- US EPA, 1998. Integrated Risk Information System. IRIS Database On-line Search. (U.S.) Environmental Protection Agency, Cincinnati, OH.
- Vogelmeier, C., Konig, G., Bencze, K. *et al.* 1987. Pulmonary involvement in zinc fume fever. *Chest* 92:946-949. Cited In: ATSDR, 1997.
- Walsh, C.T., Sandstead, H.H., Prasad, A.S., Newberne, P.M., and Fraker, P.J. 1994. Zinc: Health effects and research priorities for the 1990s. *Environ Health Perspect* 102(2):5-2046.
- Yadrick, M.K., Kenny, M.A., and Winterfeldt, E.A. 1989. Iron, copper, and zinc status: Response to supplementation with zinc or zinc and iron in adult females. *Am J Clin Nutr* 49:1450-150. Cited In: IRIS, 1993.

## **A-2.0 ECOLOGICAL TOXICITY PROFILES**

### **A-2.1 Introduction**

Exposure limits are chemical concentrations or doses which are equivalent to acceptable exposure levels for ecological receptors. That is, if calculated exposure levels do not exceed the exposure limits, no unacceptable risks to ecological receptors would be expected. If an exposure level does exceed an exposure limit, it does not imply unacceptable ecological risks. Rather, the chemical is retained as a chemical of concern (COC) for that particular receptor, and further assessment is conducted and assumptions re-evaluated to determine if potential effects are likely.

Exposure limits were derived for each ecological receptor. It was necessary to group arctic hare with lemmings because of a paucity of data for particular species. It is assumed that species which have similar gut physiology will have similar toxic responses to chemicals.

Toxicity data were used to develop exposure limits for the screening-level ecological risk assessment. The exposure limits are presented below (Table A-1) for the four metals of potential concern, and the full rationale for their derivation is provided in subsequent sections of this report.

Guidance manuals authored by CCME (1996), US EPA (2000), and BC MELP (1998) were consulted in the derivation of the ecological exposure limits. Ecological Soil Screening Levels (Eco-SSLs) are defined as screening values that can be used to identify chemicals in soil requiring further evaluation in a comprehensive ecological risk assessment. Therefore, the methodology presented in the Eco-SSL guidance would be appropriate to use to evaluate chemicals present in soils surrounding the actively mined area of LCI.

**Table A-1 Exposure limits<sup>1</sup> (mg/kg/day) for wildlife for the ecological risk assessment**

<b>Metal</b>	<b>Lemming</b>	<b>Arctic hare</b>	<b>Caribou</b>	<b>Arctic fox</b>
Cadmium	5.0	5.0	5.0	1.7
Copper	80.0 <sup>2</sup>	80.0 <sup>2</sup>	4.2	8.0 <sup>2</sup>
Lead	0.6	0.6	5.0	1.0 <sup>2</sup>
Zinc	210.0	210.0	17.0	20.0

<sup>1</sup> Exposure limit based on lowest-effect level of EC<sub>20</sub> unless otherwise indicated

<sup>2</sup> Exposure limit based on no-effect level

The general process which was followed for deriving exposure limits is outlined below. First, methods described in BC MELP (1998) guidance for development of Toxicity Threshold Values (TRVs) were used. TRVs are defined as “concentrations above which a detrimental effect will occur in an organism” and which therefore are similar to what we refer to as “exposure limits”. In general, “the EC<sub>20</sub>, or the concentration that affects 20% of the organisms exposed” is recommended as the TRV. If an EC<sub>20</sub> is not available, BC MELP (1998) provides guidance for deriving an EC<sub>20</sub> or developing an alternative TRV. For example, if an EC<sub>20</sub> is not reported, the guidance recommends that a concentration-response curve be generated from the data available in the literature and that the EC<sub>20</sub> be calculated. If these data are not available, a lowest observed adverse effect level (LOAEL) is recommended as the TRV, without dividing by any uncertainty factors. If the resulting TRV is less than regional background concentrations (BC MELP, 1997), the background concentration is used as the exposure limit. In addition, there is guidance specific to terrestrial mammalian receptors:

- use an EC<sub>20</sub>;
- give preference to those with the same feeding group;
- give preference to feeding studies (not single dose studies, or injection studies), particularly of weeks to months in duration;

- if the data are from similar animals (e.g., rodent data to compare with rodents or duck data to compare to other waterfowl), do not use any uncertainty factors. If your animals are not so closely related, divide the value by 10; and,
- if the ONLY data available for any animal species are from injection or oral dosing studies, convert the dose to concentration in food, assuming an average body weight (bw in g) for the species and an average food consumption rate (g/day dry weight). Food consumption may be estimated from the following equations:

$$F = 0.621 (bw)^{0.564} \text{ for rodents}$$

$$F = 0.577 (bw)^{0.727} \text{ for mammalian herbivores}$$

$$F = 0.235 (bw)^{0.822} \text{ for other mammals}$$

The Eco-SSL approach was used in addition to the BC approach, when sufficient data were available. This approach utilises all of the available data, instead of simply selecting one LOAEL, and assumes that any differences in the data are due to study design, variability in measurement, *etc.* rather than on differences in how the animal is reacting to the chemical. The geometric mean of the growth/reproduction effect data is calculated instead of selecting the lowest LOAEL. Then, a safety factor of between 1 and 5 is applied (CCME, 1996) (e.g., to extrapolate to animals not in the data set) to derive the exposure limit.

#### **A.2.1.2 References**

BC MELP (British Columbia Ministry of Environment, Lands and Parks). 1997. Waste Management Act, Contaminated Sites Regulation. BC Reg. 375/96.

BC MELP (British Columbia Ministry of Environment, Lands and Parks). 1998. Recommended Guidance and Checklist for Tier 1 Ecological Risk Assessment of Contaminated Sites in British Columbia. January, 1998.

CCME (Canadian Council of Ministers of the Environment). 1996. A protocol for the derivation of environmental and human health soil quality guidelines. CCME-EPC-101E.

US EPA (United States Environmental Protection Agency). 2000. Ecological Soil Screening Level Guidance. Draft. July 10, 2000. U.S. EPA, Office of Emergency and Remedial Response, Washington, DC.

#### **A-2.2 Cadmium**

One study was conducted on wild small mammals, bank voles, although histological changes and survival, not reproduction, were assessed (Swiergosz *et al.*, 1998). The effect concentration of 15 mg/kg diet was converted to a dose of 2.1 mg/kg/day, assuming a body weight of 30 g (Swiergosz *et al.*, 1998) and calculating a food ingestion rate of 0.0042 kg/d using the equation listed in Section A-2.1. In addition, a long-term study of reproductive success in mice was conducted, although exposure was through cadmium salts in water, and not the diet (Schroeder and Mitchner, 1971). The effect level for reproductive success was 2.5 mg/kg/d. This value is in agreement with the LOAEL of 4 mg/kg/d from a 2-year rat study which measured effects on



growth (Loser, 1980). Therefore, three LOAELs for small mammals were between 2.1 and 4 mg/kg/d.

A 30% decrease in weight gain was reported at 4.8 mg/kg/d for calves exposed to cadmium in the diet (Powell *et al.*, 1964). This value is in close agreement with the effect concentration for decreased weight gain of 6.4 mg/kg/d for calves, which was derived from exposure only 3 times per week, rather than daily exposure (Lynch *et al.*, 1976). It also agrees with the effect concentration for decreased weight gain derived for lambs of 4.6 mg/kg/d (Doyle *et al.*, 1974). Therefore, three effect levels for ruminants were between 4.6 and 6.4 mg/kg/d.

The LOAEL of 2.1 mg/kg/d may be divided by 10 to derive the exposure limit for arctic foxes. As a check for arctic foxes, which are closely related to dogs, the lowest effect level for renal histopathology of 5 mg/L drinking water (Anwar, 1961) was converted to a LOAEL of 0.26 mg/kg/d by assuming a body weight of 12.7 kg and a water ingestion rate of 0.652 L/d (Sample *et al.*, 1996). These two LOAELs are comparable. However, renal histopathological effects may not adversely impact growth or reproduction, and therefore this endpoint may not be ecologically relevant. In fact, the highest concentration tested (10 mg/L) did not affect growth rate in this study (Anwar, 1961).

The Eco-SSL method also may be used to derive an exposure limit for mammals. There were eight effect-levels that did not include histopathological endpoints (CEI, 2000), or severe effects (*e.g.*, greater than 70% decrease in body weight). Five of these were for rats and mice, one for lambs and two for cows. All low-effect levels were between 2.5 and 10 mg/kg/d. The geometric mean of the rodent, ruminant, and all values were 4.8, 5.2 and 5 mg/kg/d, respectively. Therefore, 5 mg/kg/d may be selected as the exposure limit for small mammals and ruminants. For those species not closely related to small mammals and ruminants (*e.g.*, arctic foxes), an uncertainty factor of 3 may be applied to derive an exposure limit of 1.7 mg/kg/d.

Because histological changes are not as ecologically relevant as growth and reproductive effects, exposure limits derived using the Eco-SSL method were selected. In addition, the exposure limit of 5 mg/kg/d for small mammals is in close agreement with the individual LOAELs reported from three studies. Similarly, the exposure limit for caribou of 5 mg/kg/d is in close agreement with individual LOAELs reported from three studies.

#### **A-2.2.1      *References***

- Anwar, R.A., Langham, R.F., Hoppert, C.A., Alfredson, B.V., and Byerrum, R.U. 1961. Chronic Toxicity studies. III. Chronic toxicity of cadmium and chromium in dogs. *Arch Environ Health* 3:456-467.
- CEI (Cantox Environmental Inc.). 2000. Ecotoxicological Profiles, Ecological Risk Assessment Program: Cominco Trail Operations, BC. Prepared for Cominco Ltd., Trail, BC. Revised January 2001.
- CCME. 1996. Canadian Soil Quality Guidelines for Cadmium: Environmental Supporting Document - Final Draft December 1996.

- Doyle, J.J., Pfander, W.H., Grebing, S.E. and Pierce, J.O. 1974. Effects of cadmium on growth, cadmium absorption and cadmium tissue levels in growing lambs. J Nutr 104:160-166. Cited In: CCME, 1996.
- Loser, E. 1980. A 2-year oral carcinogenicity study with cadmium on rats. Cancer Lett 9:191-198.
- Lynch, A.J., Smith, D.F., Fisher, M., Pike, T.L. and Weinland, B.T. 1976. Physiological responses of calves to cadmium and lead. J Animal Sci 42:410-421. Cited In: CCME, 1996.
- Powell, G.W., Miller, W.J., Morton, J.D., and Clifton, C.M. 1964. Influence of dietary cadmium level and supplemental zinc on cadmium toxicity in the bovine. J Nut 84:205-214.
- Sample, B.E., Opresko, D.M., Suter II, G.W. 1996. Toxicological Benchmarks for Wildlife: 1996 Revision. Oak Ridge National Laboratory. Oak Ridge, Tennessee. ES/ER/TM-86/R3.
- Schroeder, H.A. and Mitchener, M. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch Environ Health 23:102-106.
- Swiergosz, R., Zakrzewska, M., Sawicka-Kapusta, K., Bacia, K. and Janowska, I. 1998. Accumulation of cadmium in and its effect on bank vole tissues after chronic exposure. Ecotoxicology and Environmental Safety 41:130-136.

### **A-2.3          Copper**

Reproductive effects (decreased litter size and abnormal fetuses) were observed in a study of mice at 3,000 mg CuSO<sub>4</sub>/kg diet (220 mg Cu/kg/d), whereas no effects were observed at 2,000 mg CuSO<sub>4</sub>/kg diet (146 mg Cu/kg/d) (Lecyk, 1980). The maximum tolerable level in the diet of rats has been estimated at 1,000 mg/kg (NRC, 1980) which is equivalent to a dose of 80 mg/kg/d. This last level was selected as the exposure limit for small mammals. This value was divided by 10 to derive the exposure limit of 8 mg/kg/d for arctic foxes.

Sheep are particularly sensitive to copper, as sheep have more sulphate-reducing bacteria in their rumen which make the copper more bioavailable. The dietary requirement of ruminants has been estimated at between 8 and 10 mg/kg diet (NRC, 1980). Reduced weight gain has been observed at 200 mg Cu/kg diet in cattle (Jenkins and Hidirolou, 1989). This LOAEL was converted to a dose of 4.2 mg/kg/d using the body weight of 43.4 kg and a food ingestion rate of 0.92 kg dw/day from the study. This LOAEL is used as the exposure limit for caribou, as there is no evidence that caribou are as sensitive as sheep.

#### **A-2.3.1          References**

- Jenkins, K.J. and Hidirolou, H. 1989. Tolerance of the calf for excess copper in milk replacer. Journal of Dairy Science 72:150-156.



Lecyk, M. 1980. Toxicity of cupric sulfate in mice embryonic development. *Zool Pol* 28(2):101-105.

NRC (National Research Council). 1980. Mineral tolerance of domestic animals. Subcommittee on mineral toxicity in animals - Agriculture and renewable resources commission on natural resources. National Academy Press. Washington, D.C.

#### **A-2.4            Lead**

Growth inhibition in offspring was observed in bank voles exposed to 730 mg Pb/kg diet (Zakrzewska, 1988) or 22 mg/kg/day. However, this experiment lasted only five days. In another experiment, mice were exposed from conception throughout their lives to lead in their drinking fluid. The dose of 0.62 mg/kg/d did not affect reproductive success, although there was increased mortality prior to weaning (Donald *et al.*, 1987). The LOAEL of 0.62 mg/kg/d was selected as the exposure limit for small mammals.

One study found overt signs of toxicity in dogs exposed to 0.32 mg/kg/d, although other experiments found no signs of toxicity at 1.0 mg/kg/d (Zook, 1978). Because the 0.32 mg/kg/d dose is the lowest reported, and no effects were observed at the higher dose in other experiments, the dose of 1.0 mg/kg/d was selected as the exposure limit for arctic foxes.

Chronic toxicity has been reported in calves at doses of 5 mg/kg/d (Demayo *et al.*, 1982). A lower dose of 2.7 mg/kg/d in milk caused mortality in calves (Zmudzki *et al.*, 1983), however, direct exposure at Polaris will not be through milk. The dose of 5 mg/kg/d is in agreement with those of Lynch *et al.* (1976), where weight reduction and decreased ALAD were reported at doses of 3.9 and 7.7 mg/kg/d. Therefore, 5 mg/kg/d was selected as the exposure limit for caribou.

#### **A-2.4.1            References**

Demayo, A., Taylor, M.C., Taylor, K.W., and Hodson, P.V. 1982. Toxic effects of lead and lead compounds on human health, aquatic life, wildlife plants, and livestock. *Crit Rev Environ Control* 12(4):257-305 .

Donald, J.M., Cutler, M.G. and Moore, M.R. 1987. Effects of lead in the laboratory mouse. Development and social behaviour after lifelong exposure to 12  $\mu$ M lead in drinking fluid. *Neuropharmacology* 26(4):391-9.

Lynch, G.P., Smith, D.F., Fisher, M., Pike, T.L., and Weinland, B.T. 1976. Physiological responses of calves to cadmium and lead. *J Anim Sci* 42:410-421.

NRC (National Research Council). 1980. Mineral tolerance of domestic animals. Subcommittee on mineral toxicity in animals - Agriculture and renewable resources commission on natural resources. National Academy Press. Washington, D.C.

- Zakrzewska, M. 1988. Effect of lead on postnatal development of the bank vole (*Clethrionomys glareolus*). Arch. Environ. Contam. Toxicol. 17:365-371.
- Zmudzki, J., Bratton, G.R., Womac, C., and Rowe, L. 1983. Lead poisoning in cattle: Reassessment of the minimum toxic oral dose. Bull Environ Contam Toxicol 30:435-441.
- Zook, B.C. 1978. Lead intoxication in dogs. Chapter 10 in: Oehme, F.W. (ed). Toxicity of heavy metals in the environment. Part 1. Marcel Dekker, Inc. New York, NY. Pp. 179-190.

## **A-2.5 Zinc**

The lowest-effect level for a small mammal is 195 mg/kg/d for anaemia in ferrets (Straube *et al.*, 1980). Other LOAELs are at a similar level, with a growth endpoint for mice at 260 mg/kg/d and a reproductive endpoint for rats at 320 mg/kg/d (Schlicker and Cox, 1968; Mulhern *et al.*, 1986). No-effect levels for growth in mink and reproduction in rats range from 160 mg/kg/d to over 300 mg/kg/d (Aulerich *et al.*, 1991; Schlicker and Cox, 1968). Two lower values were for loss of fur in mink at 20.8 mg/kg/d (Bleavins *et al.*, 1983) and histological effects in testes of rats at 40 mg/kg/d (Saxena *et al.*, 1989). Excluding the fur loss and histological endpoints, five no- and low-effect levels for four different species are within a small concentration range (160 to 327 mg/kg/d), and therefore the geometric mean of these values, 210 mg/kg/d, was selected as the exposure limit for small mammals.

A dietary concentration of 900 mg/kg caused a decrease in weight gain, and a concentration of 500 mg/kg had no effect other than to increase tissue zinc concentrations in young cattle (NRC, 1980). The 900 mg/kg concentration was converted to an exposure limit of 17 mg/kg/d by assuming a body weight of 250 kg and estimating a food ingestion rate of 4.8 kg/d using the equation in Section A-2.1. This dose is consistent with effects observed in other ruminants, such as reproductive effects observed in sheep at 20 mg/kg/d (Campbell and Mills, 1979) and decreased weight gain in sheep at 34 mg/kg/d (Davies *et al.*, 1977). The exposure limit of 17 mg/kg/d was also used for caribou.

There are no toxicological data for arctic fox. The small mammal exposure limit was developed using data from smaller animals, but from four different species which are herbivores, omnivores and carnivores. The caribou exposure limit was developed from data on larger herbivores. The small mammal exposure limit divided by 10 (rounded to 20 mg/kg/d) is consistent with the caribou exposure limit (17 mg/kg/d) and was therefore selected as the exposure limit for arctic foxes.

### **A-2.5.1 References**

- ATSDR, 1997. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Zinc. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

- Aulerich, R.J., Bursian, S.J., Poppenga, R.H., Braselton, W.E. and Mullaney, T.P. 1991. Toleration of high concentrations of dietary zinc by mink. *J. Vet. Diagn. Invest.* 3:232-237.
- Bleavins MR Aulerich RJ; Hochstein JR; *et al.* 1983. Effects of excessive dietary zinc on the intrauterine and postnatal development of mink. *J Nutr* 113:2360-2367.
- Campbell, J.K. and Mills, C.F. 1979. The toxicity of zinc to pregnant sheep. *Environmental Research* 20: 1-13. Cited In: CCME, 1996
- CCME. 1996. Canadian Soil Quality Guidelines for Zinc: Environmental. Supporting Document - Final Draft, December, 1996. Canadian Council of Ministers of the Environment.
- Davies, N.T., Soliman, H.S., Corrigan, W., Flett, A. 1977. The susceptibility of suckling lambs to zinc toxicity. *Br. J. Nutr.* 38:153-157. Cited In: CCME, 1996
- Mulhern SA; Stroube WB; Jacobs RM. 1986. Alopecia induced in young mice by exposure to excess dietary zinc. *Experientia* 42:551-553. Cited In: ATSDR, 1997.
- NRC (National Research Council). 1980. Mineral tolerance of domestic animals. Subcommittee on mineral toxicity in animals - Agriculture and renewable resources commission on natural resources. National Academy Press. Washington, D.C.
- Saxena, R., Bedwal, R.S. and Mathur, R.S. 1989. Zinc toxicity and male reproduction in rats: A histological and biochemical study. *Trace Elements Med* 6(3):119-133.
- Schlicker, S.A. and Cox, D.H. 1968. Maternal dietary zinc, and development and zinc, iron, and copper content of the rat fetus. *J Nutr* 95:287-294. Cited In: Sample *et al.*, 1996.
- Straube, E.F., Schuster, N.H., and Sinclair, A.J. 1980. Zinc toxicity in the ferret. *J Comp Pathol* 90:355-361. Cited In: ATSDR, 1997.

**APPENDIX B**  
**TECHNICAL INFORMATION RELATED TO THE RISK ASSESSMENT**

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## **B-1.0 INTRODUCTION TO APPENDIX B**

This appendix provides technical information related to the assessment of the potential human health and ecological risks associated with exposure to chemicals found in surficial soil and vegetation at Cominco's Polaris Mine site on Little Cornwallis Island. The appendix also outlines in detail the derivation of the soil quality remediation objectives.

The estimation of exposure to chemicals of concern at the site was based on the following parameters:

- The chemical-specific physical, chemical and biological factors that determine the ability of the organism to take the chemicals into the body;
- The characteristics of the site and surrounding area;
- The characteristics of the environmental compartments at the site (*e.g.*, air, soil *etc.*), as well as the quantities of chemicals entering the compartments from various sources, and their persistence in these compartments;
- The behavioural and lifestyle characteristics of the human and ecological receptors that determine the actual exposures through interactions of the receptors with the various pathways (*e.g.*, respiration rate, body weight *etc.*); and,
- The equations and algorithms used to predict exposures to the receptors.

## **B-2.0 CHEMICAL CONCENTRATIONS**

Refer to the main report for details regarding on-site soil and vegetation chemical concentrations.

## **B-3.0 INPUT PARAMETERS FOR THE HUMAN HEALTH RISK ASSESSMENT**

The various model input parameters and assumptions used in the estimation of exposures are provided below.

### **B-3.1 Scenario and Exposure Pathway Assumptions for Human Receptors**

The "intended land use" exposure scenario assumes a family would spend short durations of time on Little Cornwallis Island once the mine site has been decommissioned by Cominco (described in greater detail in Appendix C). For this land use, short-term exposures from chemicals in surface soil were estimated for a variety of different pathways. Under this land use scenario, it was assumed that the Inuit receptor most at risk to chemicals from the site would be a pre-school child.

Based on the “intended land use” of the site and receptor characteristics for this land use scenario, the following exposure pathways were included:

- Inhalation of chemicals which resuspend as dusts from the soil surrounding the site;
- Ingestion of chemicals in soil; and,
- Dermal contact with chemicals in soil and airborne dusts.

Consideration was given to identifying receptors that would be at the greatest potential risk from the site, either through having the greatest probability of exposure to chemicals from the site or through having the greatest sensitivity to these chemicals.

### **B-3.2 Selected Values for Human Receptor Parameters**

For the current assessment, the potential health risks associated with individuals visiting the decommissioned Polaris Mine site were assessed for the most sensitive female pre-school Inuit child (6 months < 5 years). The pre-school child receptor typically has the greatest exposure to weight ratio, thereby making it a more sensitive potential receptor. The pre-school child is also recognised as being vulnerable to the toxic action of some of the evaluated metals of concern (*e.g.*, lead). The characteristics used in the risk assessment are outlined in Table B-1.0.

**Table B-1.0 Female Inuit pre-school child exposure characteristics**

Parameter	Value	Units	Reference
Body weight	16.4	kg	O'Connor Associates 1997
Soil ingestion rate	0.4	g/day	US EPA 1996
Air inhalation rate	8.8	m <sup>3</sup> /day	O'Connor Associates 1997
Area of exposed skin	0.214	m <sup>2</sup>	O'Connor Associates 1997
Soil adherence factor	5.85	g/m <sup>2</sup> /day	US EPA 1992

### **B-3.3 Equations and Algorithms Used to Estimate Human Exposures**

This section provides the equations and algorithms that were used to estimate exposure rates to human receptors from the site.

The following is a worked example of the potential health risks associated with an Inuit female pre-school child visiting the decommissioned Cominco Polaris Mine site. The example presents exposure and subsequent risk estimates for lead and zinc-impacted soils.

The soil concentrations and chemical-specific bioavailabilities employed in the calculation of exposure and risk are presented in Table B-2.0. Derivation of chemical bioavailabilities are discussed in detail in Appendix A.



**Table B-2.0 Soil concentrations and bioavailabilities used in the worked example**

	<b>Lead</b>	<b>Zinc</b>
Soil concentration (µg/g)	16,500	59,300
Oral bioavailability (unitless)	0.53	0.4
Inhalation bioavailability (unitless)	0.448	0.37
Dermal bioavailability (unitless)	0.0006	0.025

**B-3.3.1 Assumptions Defining Dust Levels Generated by Soils**

Background outside dust level: - 42 µg/m<sup>3</sup> (MOEE 1994)  
Percent of dust produced from soil: - 50% (Hawley 1985)

The ambient dust level can then be calculated as follows:

$$42 \text{ µg/m}^3 \times 0.50 \div 1,000,000 \text{ µg/g} = 2.1\text{E-}05 \text{ g/m}^3$$

Exposure contributions from soil on the site were considered for three routes of exposure: inhalation of soil, incidental ingestion, and dermal contact with surficial soils.

**B-3.3.2 Inhalation Exposure from On-Site Dust**

Exposure resulting from inhalation of dusts is described by the following equation:

$$\text{EXP(inh)} = \frac{\text{SC} \times \text{AI} \times \text{BDL} \times \text{BIO(inh)}}{\text{BW}}$$

where:

EXP(inh) = inhalation exposure to chemical from airborne dust (in µg/kg/day)  
SC = soil concentration of chemical (in µg/g, see Table B-2.0)  
AI = air inhalation rate (8.8 m<sup>3</sup>/day)  
BDL = background dust level (0.000021 g/m<sup>3</sup>)  
BIO(inh) = chemical-specific inhalation bioavailability (unitless)  
BW = body weight of female pre-school child (16.4 kg)

*According to the above equation and input parameters, exposure to lead and zinc from inhalation of on-site dusts [i.e., EXP(inh)] was estimated to be 0.1 and 0.3 µg/kg/day, respectively.*

**B-3.3.3 Incidental Ingestion Exposure from On-Site Soil**

Exposure resulting from incidental ingestion of on-site soil is described by the following equation:

$$\text{EXP(oral)} = \frac{\text{SC} \times \text{IR} \times \text{BIO(oral)}}{\text{BW}}$$

where:

EXP(oral)	=	exposure resulting from incidental ingestion of soil (in µg/kg/day)
SC	=	soil concentration of chemical (in µg/g, see Table B-2.0)
IR	=	soil ingestion rate (0.4 g/day)
BIO(oral)	=	oral bioavailability of chemical (unitless)
BW	=	body weight of female pre-school child (16.4 kg)

*According to the above equation and input parameters, exposure to lead and zinc due to incidental ingestion of on-site soil [i.e., EXP(oral)] was estimated to be 213.3 and 578.5 µg/kg body weight/day, respectively.*

### **B-3.3.4 Dermal Exposure from On-Site Soil**

Exposure resulting from dermal contact with on-site soil is described by the following equation:

$$\text{EXP(derm)} = \frac{\text{SC} \times \text{SA} \times \text{SAF} \times \text{BIO(derm)}}{\text{BW}}$$

where:

EXP(derm)	=	exposure resulting from dermal contact with soil (in µg/kg/day)
SC	=	soil concentration of chemical (in µg/g, see Table B-2.0)
SA	=	exposed body surface area (0.214 m <sup>2</sup> )
SAF	=	soil adherence factor (5.85 g/m <sup>2</sup> /day)
BIO(derm)	=	dermal bioavailability of chemical (unitless)
BW	=	body weight of female pre-school child (16.4 kg)

*According to the above equation and input parameters, exposure to lead and zinc due to dermal contact with on-site soil [i.e., EXP(dermal)] was estimated to be 0.8 and 113.2 µg/kg body weight/day, respectively.*

### **B-3.3.5 Total Exposure to Chemicals from Evaluated Soil Pathways**

The short-term daily exposure to lead and zinc from soil was then calculated by summing the exposures from the three different soil routes of exposure.

$$\text{EXP(total)} = \text{EXP(inhal)} + \text{EXP(oral)} + \text{EXP(derm)}$$

*Summing the contributions from the three pathways resulted in total exposure to lead and zinc of 214.2 and 692.0 µg/kg body weight/day, respectively.*

### B-3.3.6 Risk Characterization

Hazard Quotients (HQs) were estimated using the equations provided below and the exposure values previously calculated.

$$HQ = \frac{\text{Estimated Exposure}}{\text{Exposure Limit}}$$

Lead-related risks were calculated differently than health risks resulting from exposure to cadmium and zinc.

For the purposes of this risk assessment, “acceptable exposure” to lead was identified as a short-term “benchmark” blood-lead level of 20 µg/dL. The following relationship describes the uptake of lead into blood:

$$[\text{Pb}]_{\text{blood}} = \alpha + \beta(\text{uptake})$$

where:

Pb	=	concentration of Pb in blood (µg/dL)
α	=	y intercept (baseline blood-Pb level = 4 µg/dL)
β	=	slope of the regression line (statistical regression technique was used to fit a curve to absorbed lead versus blood-Pb levels; assume 0.04, US EPA 1989)

Considering that a “safe” or target level is assumed to be 20 µg/dL ...

$$20 \text{ µg/dL} = 4 + 0.04 (\text{uptakePb}),$$

$$\text{uptake(Pb)} = 400 \text{ µg/day}$$

An uptake of lead of 400 µg/day corresponds to daily exposure of 24.4 µg/kg body weight/day for a female Inuit pre-school child. This level of lead uptake has already been adjusted for bioavailability. Therefore, the health risk to a pre-school child exposed to on-site lead concentrations is quantified as:

$$\begin{aligned}
 HQ &= \frac{214.2 \text{ µg/kg/day}}{24.4 \text{ µg/kg/day}} \\
 &= 8.8
 \end{aligned}$$

Health risks for such metals as zinc were determined as follows.

$$\begin{aligned}
 \text{HQ} &= \frac{\text{Estimated Exposure}}{\text{Exposure Limit}} \\
 &= \frac{692.0 \mu\text{g/kg/day}}{300 \mu\text{g/kg/day} \times 0.4 \text{ (adjusted for study bioavailability)}} \\
 &= 5.8
 \end{aligned}$$

*The Hazard Quotients for Inuit children exposed to lead and zinc at the Polaris Mine site are 8.8 and 5.8, respectively.*

#### **B-4.0 ECOLOGICAL EXPOSURE ESTIMATES**

For the purpose of the current ecological risk assessment, the following terrestrial receptors were evaluated using both qualitative and quantitative techniques:

- Lemming
- Arctic hare
- Caribou
- Arctic fox

Receptor specific parameters and assumptions used in the assessment are summarised in Table 3.0.

**Table 3.0 Ecological receptor parameters and assumptions used in the risk assessment**

Parameter	Lemming <sup>a</sup>	Arctic hare <sup>a</sup>	Caribou	Arctic fox
Body weight (kg)	0.02	1.35	86.10 <sup>b</sup>	5.75 <sup>c</sup>
Lung ventilation rate (m <sup>3</sup> /day)	0.02	0.63	57.82 <sup>c</sup>	1.70 <sup>a</sup>
Food consumption (g/day)	grass: 1.7 willow: 1.7	willow roots: 108.9	grass: 3,180.0 <sup>c</sup> lichen: 3,180.0 <sup>c</sup>	hare: 231.5 <sup>a</sup> lemming: 57.9 <sup>a</sup>
Soil/dust intake (g/day)	0.08	2.18	127.20 <sup>a</sup>	8.10 <sup>a</sup>
Surface area (m <sup>2</sup> )	0.009	0.125	NA	0.270 <sup>a</sup>
Soil adherence factor (g/m <sup>2</sup> /day)	42.0	42.0	42.0	42.0
Area use factor	1.0 <sup>d</sup>	1.0 <sup>d</sup>	0.25 <sup>d</sup>	1.0 <sup>d</sup>

<sup>a</sup> US EPA 1993

<sup>b</sup> Thomas and Kiliaan 1985 (cited in Environment Canada Wildlife Model)

<sup>c</sup> Stahl 1967 (cited in Environment Canada Wildlife Model)

<sup>d</sup> Assumed

<sup>e</sup> Environment Canada 2000

## B-4.1 Exposure Rate Estimates from Chemically-Impacted Soil

The following sections present the methodologies applied in estimating the dose rates of ecological receptors from chemically-impacted soil and potential terrestrial prey. Dose rate estimates may differ for each receptor, depending on their habits. It should be noted that soil exposure via dermal contact was not assessed quantitatively for caribou.

The methodology is described through the use of a worked example of the potential ecological health risks associated with contaminated soils at Cominco's Polaris Mine site. The example presents exposure and subsequent risk estimates for lead-impacted soils, using a lemming as a representative herbivorous receptor, and an arctic fox as a representative carnivorous receptor.

The soil and plant concentrations, chemical-specific bioavailabilities, and biological half-lives employed in the calculation of exposure and risk are presented in Table B-4.0. Derivation of chemical bioavailabilities are discussed in detail in Appendix A.

**Table B-4.0 Soil and plant concentrations, bioavailabilities, and biological half-lives used in the risk assessment**

Parameter	Cadmium	Copper	Lead	Zinc
Soil conc. (µg/g)	14.0	12.0	141.0	3,635.0
Plant conc. (µg/g)				
grass	0.8	4.6	25.0	148.8
willow top	3.0	4.4	11.4	376.6
willow root	3.2	5.8	13.4	352.0
lichen	1.2	1.4	256.0	145.0
Oral bioavailability	0.18	0.79	0.42	0.40
Inhalation bioavailability	0.22	0.60	0.45	0.37
Dermal bioavailability	0.014	0.033	0.0008	0.025
Soil bioavailability	0.02	0.01	0.01	0.02
Biological half-life (days)	206 <sup>a</sup>	-- <sup>b</sup>	39 <sup>c</sup>	-- <sup>b</sup>

<sup>a</sup> Moore et al. 1973

<sup>b</sup> copper and zinc are essential metals that are actively regulated and are not considered to bioaccumulate

<sup>c</sup> Sharma et al. 1982 (average of muscle, liver, and kidney half-life)

### B-4.1.1 Exposure from Consumption of Vegetation

Exposure to herbivorous wildlife from the consumption of chemically-impacted vegetation was considered for the current ecological risk assessment. Measured vegetation data and receptor specific food consumption rates were used to estimate potential exposures to wildlife receptors of concern. The following outlines the methodology used to estimate daily exposure to vegetation consumption.

$$\text{EXP(veg)} = \frac{\text{VC} \times \text{IR(veg)} \times \text{BIO(oral)}}{\text{BW}}$$

where:

EXP(veg)	=	exposure from the consumption of on-site vegetation (µg/kg body weight/day)
VC	=	vegetation concentration (see Table B-4.0)
IR(veg)	=	ingestion rate of vegetation (1.7 g/day grass, 1.7 g/day willow for lemming)
BIO(oral)	=	bioavailability via ingestion (0.42 for lead)
BW	=	body weight (0.02 kg for lemming)

*The above equation estimated lemming exposure to lead via vegetation consumption to be 1,285.8 µg/kg body weight/day.*

#### **B-4.1.2 Exposure from Ingestion of Soil**

Exposure to wildlife from the incidental ingestion of chemically-impacted soil was also evaluated for the current ecological risk assessment. Measured on-site soil concentrations and receptor-specific soil ingestion rates were used to estimate potential exposures to wildlife receptors of concern. The following outlines the methodology used to estimate daily lead exposure to the lemming and arctic fox resulting from soil ingestion.

$$\text{EXP(soil)} = \frac{\text{SC} \times \text{IR(soil)} \times \text{BIO(soil)}}{\text{BW}}$$

where:

EXP(soil)	=	exposure from the ingestion on-site soil (µg/kg body weight/day)
SC	=	soil concentration (see Table B-4.0)
IR(soil)	=	soil ingestion rate (0.08 g/day for lemming; 8.10 g/day for arctic fox)
BIO(soil)	=	bioavailability via soil ingestion (0.01 for lead)
BW	=	body weight (0.02 kg for lemming; 5.75 kg for arctic fox)

*The above equation estimated lemming and arctic fox exposure to lead via on-site soil ingestion to be 5.7 and 2.0 µg/kg body weight/day, respectively.*

#### **B-4.1.3 Exposure from Inhalation of Dust**

Exposure to terrestrial receptors of concern as a result of inhalation of on-site chemically-impacted soil was estimated as part of the assessment. Measured on-site soil concentrations and receptor-specific inhalation rates were used to estimate potential exposures to the wildlife receptors. The following outlines the methodology used to estimate daily lead exposure to the lemming and arctic fox resulting from soil inhalation.

$$\text{EXP(inhal)} = \frac{\text{SC} \times \text{AI} \times \text{BDL} \times \text{BIO(inhal)}}{\text{BW}}$$

where:

EXP(inhal)	=	exposure from the inhalation of on-site soil (µg/kg body weight/day)
SC	=	soil concentration (see Table B-4.0)
AI	=	inhalation rate (0.023 m <sup>3</sup> /day for lemming; 1.7 m <sup>3</sup> /day for arctic fox)
BDL	=	background dust level (0.000021 g/m <sup>3</sup> )
BIO(soil)	=	bioavailability via inhalation (0.45 for lead)
BW	=	body weight (0.02 kg for lemming; 5.75 kg for arctic fox)

*The above equation estimated lemming and arctic fox exposure to lead via on-site soil inhalation to be 0.0 and 0.0 µg/kg body weight/day, respectively.*

#### **B-4.1.4 Exposure from Dermal Contact with Soil**

Exposure to terrestrial receptors of concern as a result of dermal contact with on-site chemically-impacted soil was estimated as part of the assessment. Measured on-site soil concentrations and receptor-specific dermal contact rates were used to estimate potential exposures to the wildlife receptors. The following outlines the methodology used to estimate daily lead exposure to the lemming and arctic fox resulting from dermal contact with contaminated soil.

$$\text{EXP(dermal)} = \frac{\text{SC} \times \text{SA} \times \text{SAF} \times \text{BIO(dermal)}}{\text{BW}}$$

where:

EXP(dermal)	=	exposure from dermal contact with soil (µg/kg body weight/day)
SC	=	soil concentration (see Table B-4.0)
SA	=	body surface area (0.0086 m <sup>2</sup> for lemming; 0.27 m <sup>2</sup> for arctic fox)
SAF	=	soil adherence factor (42 g/m <sup>2</sup> /day for lemming and fox)
BIO(dermal)	=	bioavailability via dermal contact (0.0008 for lead)
BW	=	body weight (0.02 kg for lemming; 5.75 kg for arctic fox)

*The above equation estimated lemming and arctic fox exposure to lead as a result of dermal contact with on-site soil to be 2.0 and 0.2 µg/kg body weight/day, respectively.*

#### **B-4.1.5 Exposure from Consumption of Prey**

Exposure of carnivorous wildlife (e.g., arctic fox) to on-site metals as a result of consumption of impacted prey species (e.g., arctic hare and lemming) was quantified in the risk assessment. In order to do this, body burdens of prey species was first estimated.



Steady-state body burdens for lead and other bioaccumulative metals were estimated for the arctic hare and lemming using a first-order elimination rate law. It was assumed that, within the body, steady-state chemical concentrations were attained after an exposure period equal to seven half-lives of a chemical. The biological half-lives used in these calculations were presented in Table B-4.0. Due to the limited amount of data on biological half-lives in wildlife species, half-lives for laboratory animals were used.

Chemical concentrations of prey species, following either the lesser of seven half-lives (*i.e.*, 7 x  $t_{1/2}$ ) or expected prey lifespan of exposure, were used in the estimation of the higher trophic level receptor exposures.

$$BB = EXP(tot) \times [(1/k) \times (1 - e^{-kt})] \div 1,000 \text{ g/kg}$$

where:

$$\begin{aligned} BB &= \text{prey whole body burden } (\mu\text{g/g}) \\ EXP(tot) &= \text{total daily lead exposure of prey (1,293.5 } \mu\text{g/kg/day for lemming} \\ &\quad \text{and 456.6 } \mu\text{g/kg/day for hare)} \\ [(1/k) \times (e^{-kt})] &= \text{decay constant (day}^{-1}\text{)} \\ k &= \ln(2)/t_{1/2} \\ t &= \text{time (273 days for lemming and hare}^1\text{)} \end{aligned}$$

*The decay constant for lead was calculated to be 55.8 for both the lemming and arctic hare. This constant was then used to calculate whole body burdens of the lemming and arctic fox of 72.2 and 25.5  $\mu\text{g/g}$ , respectively.*

As indicated in Table B-3.0, the diet of an arctic fox was assumed to consist of 20% lemmings and 80% arctic hare on per weight basis. The equation below describes how exposure to lead through chemically-impacted prey consumption was calculated.

$$EXP(pre) = \frac{BB(pre) \times IR(pre) \times BIO(oral)}{BW}$$

where:

$$\begin{aligned} EXP(pre) &= \text{exposure from the consumption of prey } (\mu\text{g/kg body weight/day}) \\ BB(pre) &= \text{chemical body burden of prey (72.2 } \mu\text{g/g for lemming; 25.5 } \mu\text{g/g} \\ &\quad \text{for arctic hare)} \\ IR(pre) &= \text{ingestion rate of prey (231.5 g/day of hare; 57.9 g/day of lemming)} \\ BIO(oral) &= \text{bioavailability via ingestion (0.42 for lead)} \\ BW &= \text{body weight (5.75 for arctic fox)} \end{aligned}$$

<sup>1</sup> lifespans of lemming and hare are approximately 1 and 1.25 years, respectively. These lifespans exceed the time needed to reach steady-state body burdens for lead (*i.e.*, 7 x 39 days = 273 days)

The above equation estimated arctic fox exposure to lead via prey consumption to be 736.4 µg/kg body weight/day.

#### **B-4.1.6 Total Exposure to Ecological Receptors from All Quantified Pathways**

Total exposure to the receptors of concern were estimated by summing exposures from all the potential routes of exposure. For the herbivorous receptors (e.g., lemming), total exposure to lead was calculated as follows:

$$\text{EXP}(\text{total}) = \text{EXP}(\text{veg}) + \text{EXP}(\text{soil}) + \text{EXP}(\text{inhal}) + \text{EXP}(\text{dermal})$$

Summing the contributions from the four pathways resulted in total exposure to lead of 1,293.5 µg/kg body weight/day for the lemmings spending 100% of their time on the Cominco Polaris Mine site.

For the arctic fox, total exposure to lead was calculated as follows:

$$\text{EXP}(\text{total}) = \text{EXP}(\text{prey}) + \text{EXP}(\text{soil}) + \text{EXP}(\text{inhal}) + \text{EXP}(\text{dermal})$$

Summing the contributions from the four pathways resulted in total exposure to lead of 738.6 µg/kg body weight/day for the arctic foxes spending 100% of their time at the Cominco Polaris Mine site.

#### **B-4.1.7 Risk Characterization**

Hazard Quotients (HQs) were estimated using the equations provided below and the exposure values previously calculated.

$$\text{HQ} = \frac{\text{Estimated Exposure}}{\text{Exposure Limit}}$$

The HQ for lemmings exposed to lead was calculated as:

$$\begin{aligned} \text{HQ} &= \frac{1,293.5 \text{ } \mu\text{g/kg body weight/day}}{620 \text{ } \mu\text{g/kg body weight/day} \times 0.42} \\ &= 5.0 \end{aligned}$$

The HQ for arctic foxes exposed to lead was determined to be:

$$\begin{aligned} \text{HQ} &= \frac{738.6 \text{ } \mu\text{g/kg body weight/day}}{1,000 \text{ } \mu\text{g/kg body weight/day} \times 0.42} \\ &= 1.8 \end{aligned}$$

The Hazard Quotients for lemmings and arctic foxes exposed to lead at the Polaris Mine site were determined to be 5.0 and 1.8, respectively.

## **B-5.0 SOIL QUALITY REMEDIATION OBJECTIVES**

The methodology used to estimate soil quality remediation objectives is described in detail in section 5 in the main report.

## **B-6.0 REFERENCES**

- Environment Canada 2000. Wildlife Service. Hinterland Who's Who: The Arctic Fox.  
<http://www.cws-scf.ec.gc.ca/hww-fap/afox/arfox.html>
- Hawley, J.K. 1985. Assessment of health risks from exposure to contaminated soil. *Risk Anal* 5: 289 – 302.
- MOEE (Ontario Ministry of Environment and Energy) 1994. Proposed Guidelines for the Clean-up of Contaminated Sites in Ontario. July 1994.
- Moore, W., Stara, J.F., Crocker, W.C., Malanchuk, M., and Ilris, R. 1973. Comparison of cadmium retention in rats following different routes of administration. *Environ Res* 6: 473. Cited In: Neathery, M.W., and Miller, W.J. 1975. Metabolism and toxicity of cadmium, mercury, and lead in animals: A review. *J Dairy Sci* 58(12): 1767 – 1781.
- O'Connor Associates 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment. Richardson, G.M. (Ed) O'Connor Associates Environmental Inc., Ottawa, ON.
- Sharma, R.P., Street, J.C., Shupe, J.L., and Bourcier, D.R. 1982. Accumulation and depletion of cadmium and lead in tissues and milk of lactating cows fed small amounts of these metals. *J Dairy Sci* 65(6): 972-979.
- Stahl, W.R. 1967. Scaling of respiratory variables in mammals. *J Appl Physiol* 22: 453 – 460.
- Thomas, D.C. and Kiliaan, H.P.L. 1984. Physical condition of the Beverly herd of caribou in March 1984. Canadian Wildlife Service, pp. 17-18, 20-21.
- US EPA (U.S. Environmental Protection Agency) 1989. *Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation*, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina.
- US EPA (U.S. Environmental Protection Agency) 1996. Exposure Factors Handbook. Vol 1: General Factors, page 4-24.
- US EPA (U.S. Environmental Protection Agency) 1993. Wildlife Exposure Factors Handbook. Volume I of II. Office of Health and Environmental Assessment. EPA/600/R-93/187. December 1993.

**APPENDIX C**  
**HIGH ARCTIC COMMUNITY QUESTIONNAIRE RESULTS**

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## HIGH ARCTIC COMMUNITY QUESTIONNAIRE RESULTS

On November 2-4, 2000, Mr. Stephen Morison of Gartner Lee Ltd. met with members of the communities of Resolute Bay and Grise Fiord to discuss the intended land use of the mined area on LCI following Cominco's closure of its Polaris mine. In an attempt to gain a clearer understanding of the "site-specific" input parameters of the exposure assessment (e.g., frequency of land use, duration of exposure, age classes exposed etc.), CANTOX ENVIRONMENTAL INC. authored a community consultation questionnaire designed to reduce uncertainties in the risk assessment through its exploration of the high arctic Inuit lifestyle. Both Inuit and English versions of the questionnaire are presented in Appendix D.

Resolute Bay community members that participated in the consultation process include:

1. George Eckalook
2. Mark Amarualak
3. Simonie Amagoalik
4. Nathaniel Kalluk
5. Ludy Pudluk
6. Isaac Kulluk
7. Herodier Kalluk
8. Nangat Idlout
9. David Kulluk

Grise Fiord community members that participated include:

1. Liza Ningiuk
2. Abraham Pijamini
3. Layneckee Kakkee
4. Larry Audlaluk
5. Jaypatee Akeeagok

Results of the discussions that are specifically relevant to the risk assessment are presented in Tables C-1 and C-2.

Members of Resolute Bay tend to use Little Cornwallis Island as a "stop-over" en-route to areas they consider to more suitable for both hunting and fishing (e.g., Bathurst Island). Due to the low numbers of caribou, LCI (and specifically the southern part where the mine currently sits) was considered to be a poor area for hunting. Therefore, visits to the island are typically brief and in the past have been for the purpose of gathering supplies at the Cominco facilities.

Several participants commented that they would miss Cominco's presence on LCI.

**Table C-1 Questionnaire responses from members of Resolute Bay**

Questions	Response	Comments
HUNTING AND FISHING PATTERNS ...		
Do you hunt for food?	<ul style="list-style-type: none"> <li>• yes ... 9</li> <li>• no ...</li> </ul>	
Do you fish for food?	<ul style="list-style-type: none"> <li>• yes ... 9</li> <li>• no ...</li> </ul>	
Where do you travel to hunt/fish for food?	Somerset Isl., Devon Isl., Prince of Wales Isl. Surrounding Resolute Bay. Creswell Bay. LCI (for seals).	
Do you ever hunt on LCI?	<ul style="list-style-type: none"> <li>• yes ... 8</li> <li>• no ... 1</li> </ul>	[see map for locations] “Stopped hunting when mine opened” “Only hunt in summer” “Only hunt in the northern part of LCI since the south part is not suitable for caribou”
How often do you hunt on LCI?	<ul style="list-style-type: none"> <li>• often ... 2</li> <li>• occasionally ... 4</li> <li>• rarely ... 2</li> <li>• never ... 1</li> </ul>	Often = one time per month. Occasionally = once in a season, up to two to three times per year. Rarely = once per year
What animals do you hunt on LCI?	Caribou (summer & winter); muskox; seal; duck/geese (summer); ptarmigan; polar bear All hunting was “opportunity-dependent”	



Questions	Response	Comments
Do you ever fish on LCI?	<ul style="list-style-type: none"> <li>• yes ... 4</li> <li>• no ... 5</li> </ul>	<p>[see map for locations]</p> <p>One respondent commented that there were no fish on LCI</p>
How often do you fish on LCI?	<ul style="list-style-type: none"> <li>• often ... 1</li> <li>• occasionally ... 1</li> <li>• rarely ... 2</li> <li>• never ... 1</li> </ul>	<p>Often = once per month</p> <p>Occasionally = once in a season, up to two to three times per year</p> <p>Rarely = once per year</p>
How often do you eat wild game from LCI?	<ul style="list-style-type: none"> <li>• often ... 8</li> <li>• occasionally ... 1</li> <li>• rarely ...</li> <li>• never ...</li> <li>• undecided ...</li> </ul>	<p>Often = once per month</p> <p>Occasionally = once in a season, up to two to three times per year</p> <p>Rarely = once per year</p> <p>“Few caribou on LCI”</p> <p>“Animals may not be good to eat because of the ‘dirty snow’”</p> <p>“No caribou”</p>
How often do you eat fish from LCI?	<ul style="list-style-type: none"> <li>• often ... 3</li> <li>• occasionally ... 5</li> <li>• rarely ...</li> <li>• never ... 1</li> <li>• undecided ...</li> </ul>	<p>“Fish are very small”</p>

Questions	Response	Comments
LAND USE ...		
How many times a season do you visit LCI now?	<ul style="list-style-type: none"> <li>• 1-3 times per winter ... 5</li> <li>• 1-3 times per spring ... 2</li> <li>• 1-3 times per summer ... 6</li> <li>• 1-3 times per fall ... 1</li> <li>• never ...</li> </ul>	Appears to be largely weather-dependent.
How long do you typically stay per visit?	<ul style="list-style-type: none"> <li>• a few hours ...</li> <li>• 1 to 3 days ... 8</li> <li>• 3 to 14 days ...</li> <li>• 14 to 30 days ...</li> <li>• 30 to 90 days ...</li> <li>• more than 90 days ...</li> </ul>	LCI is commonly used as a stop en-route Bathurst.
Once Cominco removes its facilities, would you be:	<ul style="list-style-type: none"> <li>• less likely to visit ... 3</li> <li>• equally likely to visit ... 3</li> <li>• more likely to visit ... 3</li> <li>• unsure ...</li> </ul>	<p>“Use LCI as a stop en-route to Bathurst Isl.”</p> <p>“More likely to visit if the buildings stay”</p> <p>“Have stopped on LCI while en-route to Bathurst to look for caribou”</p> <p>“Will miss mine”</p>

Questions	Response	Comments
Once the mine has been closed, how many times per season would you visit LCI?	<ul style="list-style-type: none"> <li>• 1-5 times per winter ... 3</li> <li>• 1-5 times per spring ... 2</li> <li>• 1-5 times per summer ... 5</li> <li>• 1-5 times per fall ... 2</li> <li>• unsure ... 1</li> </ul>	
Once the mine has closed, how long would you stay per visit?	<ul style="list-style-type: none"> <li>• a few hours ...</li> <li>• 1 to 3 days ... 5</li> <li>• 3 to 14 days ... 2</li> <li>• 14 to 30 days ...</li> <li>• 30 to 90 days ...</li> <li>• more than 90 days ...</li> </ul>	Appears to be weather-dependent.
What types of shelter do you use while camping?	Double-walled tents in the summer. Occasional use of igloos in the winter.	
Does the shelter have an open floor?	<ul style="list-style-type: none"> <li>• yes ... 2</li> <li>• no ... 5</li> </ul>	
What are the ages of the people that accompany you while camping?	<ul style="list-style-type: none"> <li>• 0 to 6 months ... 1</li> <li>• 6 months to 5 years ... 1</li> <li>• 5 years to 12 years ... 3</li> <li>• 12 years to 20 years ... 4</li> <li>• 20 years and above ... 2</li> </ul>	Children of all ages are commonly brought along on hunting/fishing camping trips.

Questions	Response	Comments
How do those that do not hunt/fish spend their time on LCI?	Some stay near the camp to help organize. Otherwise may come along to learn how to hunt/fish. Very small children may be carried to ensure their safety when polar bears are near.	
Do you bring food from home when you travel to hunt or fish?	<ul style="list-style-type: none"> <li>• yes ... 9</li> <li>• no ...</li> </ul>	
Where do you get your water from when you travel to hunt or fish?	Ice/snow ... available everywhere.	

Grise Fiord participants indicated that historically they have not traveled to LCI for hunting or fishing. Instead, they prefer to hunt closer to their community of Grise Fiord. The one person who voiced possible interest in visiting LCI following the closure of the mine also confirmed that the exposure scenario would be of a brief nature.

**Table C-2 Questionnaire responses from members of Grise Fiord**

Questions	Response	Comments
HUNTING AND FISHING PATTERNS ...		
Do you hunt for food?	<ul style="list-style-type: none"> <li>• yes ... 5</li> <li>• no ...</li> </ul>	
Do you fish for food?	<ul style="list-style-type: none"> <li>• yes ... 5</li> <li>• no ...</li> </ul>	
Where do you travel to hunt/fish for food?	In the immediate area surrounding Grise Fiord. Also at Bathurst Isl., Devon Isl., and Ellesmere Isl.	

Questions	Response	Comments
Do you ever hunt on LCI?	<ul style="list-style-type: none"> <li>• yes ...</li> <li>• no ... 5</li> </ul>	
How often do you hunt on LCI?	<ul style="list-style-type: none"> <li>• often ...</li> <li>• occasionally ...</li> <li>• rarely ...</li> <li>• never ... 5</li> </ul>	
What animals do you hunt on LCI?	--	
Do you ever fish on LCI?	<ul style="list-style-type: none"> <li>• yes ...</li> <li>• no ... 5</li> </ul>	
How often do you fish on LCI?	<ul style="list-style-type: none"> <li>• often ...</li> <li>• occasionally ...</li> <li>• rarely ...</li> <li>• never ... 5</li> </ul>	
How often do you eat wild game from LCI?	<ul style="list-style-type: none"> <li>• often ...</li> <li>• occasionally ...</li> <li>• rarely ...</li> <li>• never ... 5</li> <li>• undecided ...</li> </ul>	

Questions	Response	Comments
How often do you eat fish from LCI?	<ul style="list-style-type: none"> <li>• often ...</li> <li>• occasionally ...</li> <li>• rarely ...</li> <li>• never ... 5</li> <li>• undecided ...</li> </ul>	
LAND USE ...		
How many times a season do you visit LCI now?	<ul style="list-style-type: none"> <li>• 1-3 times per winter ...</li> <li>• 1-3 times per spring ...</li> <li>• 1-3 times per summer ...</li> <li>• 1-3 times per fall ...</li> <li>• never ... 5</li> </ul>	
How long do you typically stay per visit?	<ul style="list-style-type: none"> <li>• a few hours ...</li> <li>• 1 to 3 days ...</li> <li>• 3 to 14 days ...</li> <li>• 14 to 30 days ...</li> <li>• 30 to 90 days ...</li> <li>• more than 90 days ...</li> </ul>	

Questions	Response	Comments
Once Cominco removes its facilities, would you be:	<ul style="list-style-type: none"> <li>• less likely to visit ...</li> <li>• equally likely to visit ...</li> <li>• more likely to visit ... 1</li> <li>• unsure ... 3</li> </ul>	One respondent thought there may be better hunting once the mine closes.
Once the mine has been closed, how many times per season would you visit LCI?	<ul style="list-style-type: none"> <li>• 1-5 times per winter ...</li> <li>• 1-5 times per spring ... 1</li> <li>• 1-5 times per summer ...</li> <li>• 1-5 times per fall ...</li> <li>• unsure ... 3</li> </ul>	
Once the mine has closed, how long would you stay per visit?	<ul style="list-style-type: none"> <li>• a few hours ...</li> <li>• 1 to 3 days ... 1</li> <li>• 3 to 14 days ...</li> <li>• 14 to 30 days ...</li> <li>• 30 to 90 days ...</li> <li>• more than 90 days ...</li> </ul>	
What types of shelter do you use while camping?	Double-walled tents in the summer. Occasional use of igloos in the winter.	
Does the shelter have an open floor?	<ul style="list-style-type: none"> <li>• yes ...</li> <li>• no ... 4</li> </ul>	



Questions	Response	Comments
What are the ages of the people that accompany you while camping?	<ul style="list-style-type: none"> <li>• 0 to 6 months ...</li> <li>• 6 months to 5 years ...</li> <li>• 5 years to 12 years ...</li> <li>• 12 years to 20 years ...</li> <li>• 20 years and above ...</li> </ul>	Children of all ages are commonly brought along on hunting/fishing camping trips.
How do those that do not hunt/fish spend their time on LCI?	To learn about hunting/fishing and camping.	
Do you bring food from home when you travel to hunt or fish?	<ul style="list-style-type: none"> <li>• yes ... 3</li> <li>• no ... 1</li> </ul>	
Where do you get your water from when you travel to hunt or fish?	Ice/snow ... 4	

Figures 1 through 10 portray the historical and intended land use of the Cominco mine site area on LCI. Participants were asked to outline their preferred areas to hunt, fish and camp when visiting LCI. Each figure/map is briefly described below.

Figure 1 shows the preferred route to Bathurst Island under different weather conditions. The figure also displays locations of the two communities of Resolute Bay and Grise Fiord, along with the various islands that were identified as being good hunting areas by the respondents.

Figure 2 identifies the north-eastern portion of LCI to be the preferred area for camping. The area of the mine site is identified as being poor for hunting. Caribou are expected to be found north of the mine, in the more vegetated areas.

Figure 3 outlines the areas at which caribou may be found. Fishing on LCI was considered to be poor.

Figure 4 identifies the vegetated area most likely to host actively grazing caribou. The preferred camping area was again on the north-eastern portion of LCI.

Figure 5 shows that caribou have been spotted as far south as Frustration Lake and Lois Lake during the summer and fall. Walrus have been hunted north of Garrow Bay. Fishing was considered fair.

A potential camping area near the mine is presented in Figure 6. Seals appear to be actively hunted along the southern shore-line of LCI. Camping also took place on the north-eastern portion of the island.

Figure 7 identifies an area near the mine that had previously been camped at. The participant also identified caribou hunting to have taken place in those areas surrounding Frustration Lake and Lois Lake.

Figure 8 outlines the area where caribou were most likely to be found in the past. At present, hunting for caribou was considered poor. The waters near Garrow Bay were considered unfavourable for hunting seals.

Figure 9 identifies polar bears, seals, hare and caribou on LCI. The preferred camping area is north-east of the mine.

Figure 10 shows areas that were fished prior to the start-up of the Cominco Polaris mine. Caribou were hunted north of the present mine site.

**APPENDIX D**  
**HIGH ARCTIC COMMUNITY QUESTIONNAIRE**



**Section 2 - Hunting and Fishing Patterns**

Do you hunt or fish for food?

hunting:	<input type="radio"/>	yes	<input type="radio"/>	no
fishing:	<input type="radio"/>	yes	<input type="radio"/>	no

How often do you hunt for food?

<input type="text"/> times per week	<input type="text"/> times per year
<input type="text"/> times per month	<input type="text"/> rarely

How often do you fish for food?

<input type="text"/> times per week	<input type="text"/> times per year
<input type="text"/> times per month	<input type="text"/> rarely

Where do you travel to hunt/fish for food?

<input type="text"/>	<input type="text"/>
<input type="text"/>	<input type="text"/>
<input type="text"/>	<input type="text"/>
<input type="text"/>	<input type="text"/>
<input type="text"/>	<input type="text"/>

Do you ever hunt on Little Cornwallis Island?

<input type="radio"/>	yes	<input type="radio"/>	no
-----------------------	-----	-----------------------	----

If yes, where do you hunt on the Island (please mark on map)?

<input type="text"/>
<input type="text"/>

How often do you hunt on the Island?

<input type="text"/> times per week	<input type="text"/> times per year
<input type="text"/> times per month	<input type="text"/> rarely

What animals do you hunt on Little Cornwallis Island?

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Do you ever fish on Little Cornwallis Island?

Q      yes

Q      no

If yes, where do you fish on the Island? (please mark on map)

---

---

How often do you fish on the Island?

\_\_\_\_ times per week  
\_\_\_\_ times per month

\_\_\_\_ times per year  
\_\_\_\_ rarely

What fish do you catch there?

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How often do you eat wild game?

\_\_\_\_ times per day  
\_\_\_\_ times per week

\_\_\_\_ times per month  
\_\_\_\_ rarely

How often do you eat wild game from Little Cornwallis Island?

\_\_\_\_\_ times per day

\_\_\_\_\_ times per month

\_\_\_\_\_ times per week

\_\_\_\_\_ rarely

How much of this meat do you usually eat for a single meal?

☐ small serving

☐ medium serving

☐ large serving

How often do you eat fish?

\_\_\_\_\_ times per day

\_\_\_\_\_ times per month

\_\_\_\_\_ times per week

\_\_\_\_\_ rarely

How often do you eat fish from Little Cornwallis Island?

\_\_\_\_\_ times per day

\_\_\_\_\_ times per month

\_\_\_\_\_ times per week

\_\_\_\_\_ rarely

How much of these fish do you usually eat for a single meal?

☐ small serving

☐ medium serving

☐ large serving

### ***Section 3 - Land Use***

Once Cominco removes its facilities, would you be:

☐ less likely to visit Little Cornwallis Island?

☐ equally likely to visit Little Cornwallis Island?

☐ more likely to visit Little Cornwallis Island?

How many times a season do you visit Little Cornwallis Island now?

\_\_\_\_\_ times per winter

\_\_\_\_\_ times per summer

\_\_\_\_\_ times per spring

\_\_\_\_\_ times per fall

How long do you typically stay per visit?

- |                                    |   |
|------------------------------------|---|
| <input type="radio"/> a few hours  | <input type="radio"/> 14 to 30 days     |
| <input type="radio"/> 1 to 3 days  | <input type="radio"/> 30 to 90 days     |
| <input type="radio"/> 3 to 14 days | <input type="radio"/> more than 90 days |

Once the mine has been closed, how many times a season would you visit Little Cornwallis Island?

- |                        |                        |
|------------------------|------------------------|
| _____ times per winter | _____ times per summer |
| _____ times per spring | _____ times per fall   |

Once the mine has been closed, how long would you stay per visit?

- |                                    |   |
|------------------------------------|---|
| <input type="radio"/> a few hours  | <input type="radio"/> 14 to 30 days     |
| <input type="radio"/> 1 to 3 days  | <input type="radio"/> 30 to 90 days     |
| <input type="radio"/> 3 to 14 days | <input type="radio"/> more than 90 days |

While staying on Little Cornwallis Island, where would you camp? (please mark on map)

---

What type of shelter do you use while camping?

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Does the shelter have an open floor?

- |                           |                          |
|---------------------------|--------------------------|
| <input type="radio"/> yes | <input type="radio"/> no |
|---------------------------|--------------------------|

When you travel to hunt or fish, do you ever bring anyone else with you?

- |                           |                          |
|---------------------------|--------------------------|
| <input type="radio"/> yes | <input type="radio"/> no |
|---------------------------|--------------------------|

What are the ages of the people that come along?

- |   |  |
|---|--|
| <input type="radio"/> 0 to 6 months       | <input type="radio"/> 12 years to 20 years |
| <input type="radio"/> 6 months to 5 years | <input type="radio"/> 20 years and above   |
| <input type="radio"/> 5 years to 12 years |  |



How do the others who do not hunt or fish spend their time on the Island?

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Do you bring food from home when you travel to hunt or fish?

☐

yes

☐

no

Where do you get your water from when you travel to hunt or fish?

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**Thank you for your time. If you have any questions or concerns regarding the risk assessment and you would like to discuss them, feel free to contact either:**

**Mr. Stephen Morison,(GARTNER LEE LTD.) at (403) 262 4299 or**

**Mr. Bart Koppe (CANTOX ENVIRONMENTAL INC.) at (604) 299 4144.**





ኤፌልጋፍም ድረገጥረሕጻኑ ሊረገጥ?

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ልክጋረሕገሕጻኑ ሊረገጥ?

Q      ል/ፋ

Q      ል/ፋ/ፋ/ፋ

ፋጥፖሉኑ፣ ዴሙ ልክጋረሕገሕጻኑ (ዴጋጋጋጋ ጠጠጋጋ)

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ኤፌልጋፍም ልክጋረሕገሕጻኑ ሊረገጥ?

\_\_\_\_ ግጥሙ ለዴረፋፋፋ

\_\_\_\_ ግጥሙ ለፋፋ

\_\_\_\_ ግጥሙ ርፍፍ

\_\_\_\_ ልክጋረሕገሕጻኑ ለፋፋ

ኤፌልጋፍም ልክጋረሕገሕጻኑ ርጋጋጋ?

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ኤፌልጋፍም ድረገጥረሕጻኑ ሊረገጥ?

\_\_\_\_ ግጥሙ ድጋፍ

\_\_\_\_ ግጥሙ ለፋፋ

\_\_\_\_ ግጥሙ ለዴረፋፋፋ

\_\_\_\_ ሙጋጋጋጋጋ

ኤፌልጋፍም ድረገጥረሕጻኑ ሊረገጥ ሙጋጋጋጋጋ?

\_\_\_\_ ግጥሙ ድጋፍ

\_\_\_\_ ግጥሙ ለፋፋ

\_\_\_\_ ግጥሙ ለዴረፋፋፋ

\_\_\_\_ ሙጋጋጋጋጋ

ኔጌ ልብወለድና ርዕሴ ሙሉ ማሳሰቢያ ማረጋገጥ?

- ☐ ገደብ
- ☐ ገደብ ልብወለድ
- ☐ ልብወለድ

ኔጌ ልብወለድና ልብወለድ ማረጋገጥ?

- |                                 |                                   |
|---------------------------------|-----------------------------------|
| <input type="radio"/> ገደብ ልብወለድ | <input type="radio"/> ገደብ ልብወለድ   |
| <input type="radio"/> ገደብ ልብወለድ | <input type="radio"/> ልብወለድ ልብወለድ |

ኔጌ ልብወለድና ልብወለድ ለገደብ?

- |                                 |                                   |
|---------------------------------|-----------------------------------|
| <input type="radio"/> ገደብ ልብወለድ | <input type="radio"/> ገደብ ልብወለድ   |
| <input type="radio"/> ገደብ ልብወለድ | <input type="radio"/> ልብወለድ ልብወለድ |

ኔጌ ልብወለድና ልብወለድ ማረጋገጥ?

- ☐ ገደብ
- ☐ ገደብ ልብወለድ
- ☐ ልብወለድ

### ለ ማረጋገጥ - ማረጋገጥ ማረጋገጥ

ከገደብ ልብወለድ ልብወለድ ልብወለድ?

- ☐ ለገደብ ልብወለድ ልብወለድ?
- ☐ ለገደብ ልብወለድ ልብወለድ ልብወለድ?
- ☐ ለገደብ ልብወለድ ልብወለድ ልብወለድ?

ኔጌ ልብወለድና ልብወለድ ለገደብ ልብወለድ?

- |                                 |                                 |
|---------------------------------|---------------------------------|
| <input type="radio"/> ገደብ ልብወለድ | <input type="radio"/> ገደብ ልብወለድ |
| <input type="radio"/> ገደብ ልብወለድ | <input type="radio"/> ገደብ ልብወለድ |

ክፍል 4 ለመጠቀሙ ለጥያቄዎቹ?

- ☐ ለጥያቄው ልዩነት
- ☐ 1 ምረቃ 3 ወር ይገባል
- ☐ 3 ምረቃ 14 ወር ይገባል

- ☐ 14 ምረቃ 30 ወር ይገባል
- ☐ 30 ምረቃ 90 ወር ይገባል
- ☐ ለጥያቄው 90 ምረቃ ይገባል

በክፍል 4 ለመጠቀሙ ለጥያቄዎቹ ለጥያቄው ልዩነት?

- \_\_\_\_\_ ምረቃዎች ይገባሉ
- \_\_\_\_\_ ምረቃዎች ይገባሉ

- \_\_\_\_\_ ምረቃዎች ይገባሉ
- \_\_\_\_\_ ምረቃዎች ይገባሉ

በክፍል 4 ለመጠቀሙ ለጥያቄዎቹ ለጥያቄው ልዩነት?

- ☐ ለጥያቄው ልዩነት
- ☐ 1 ምረቃ 3 ወር ይገባል
- ☐ 3 ምረቃ 14 ወር ይገባል

- ☐ 14 ምረቃ 30 ወር ይገባል
- ☐ 30 ምረቃ 90 ወር ይገባል
- ☐ 90 ይገባል ይገባል

ለጥያቄው ልዩነት ለጥያቄው ልዩነት? (ወይንም ይገባል)

---

ለጥያቄው ልዩነት ለጥያቄው ልዩነት? ይገባል ይገባል

---



---

ለጥያቄው ልዩነት?

- ☐ ለ/4 ☐ ለ/4

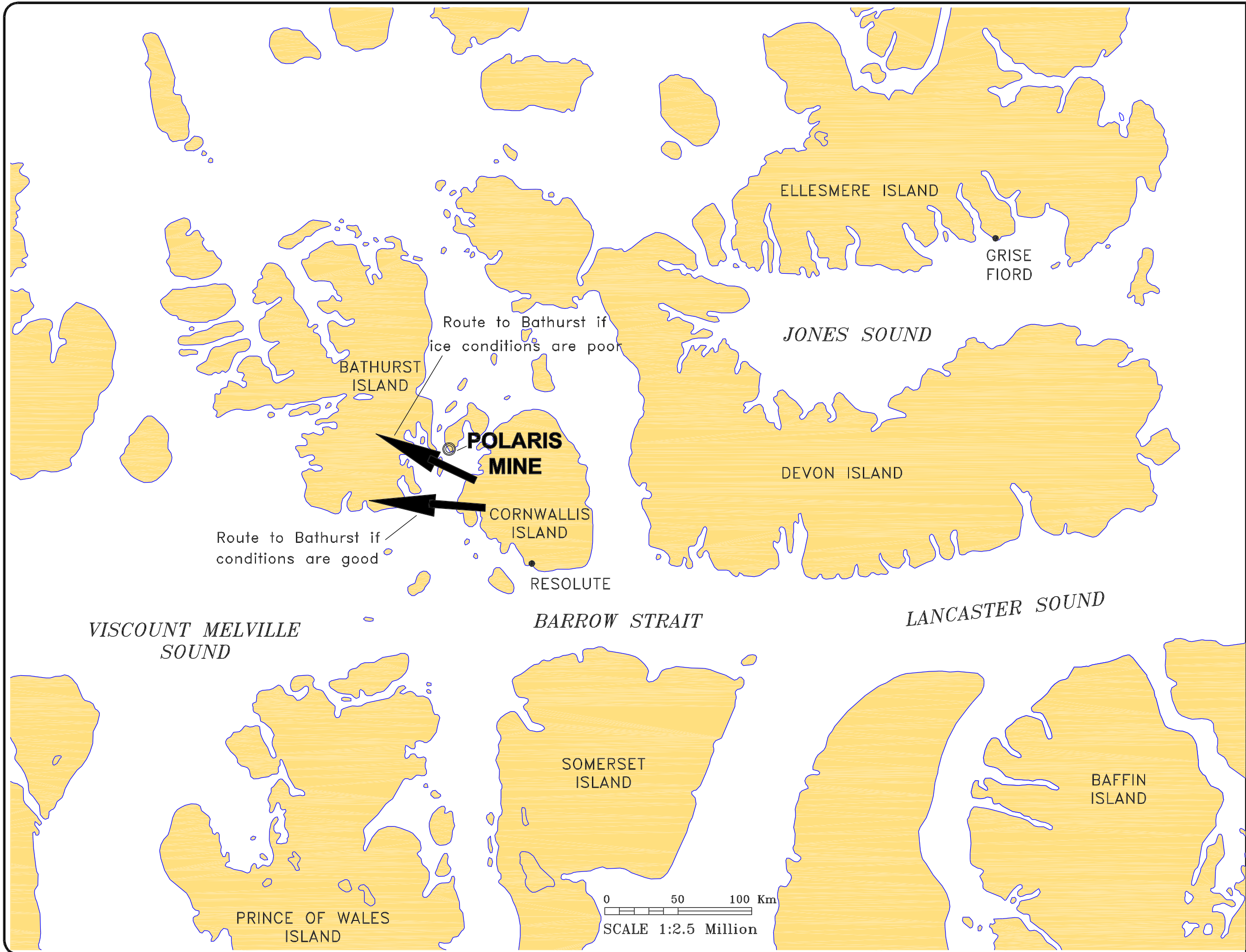
በጥያቄው ልዩነት ለጥያቄው ልዩነት? ለጥያቄው ልዩነት?

- ☐ ለ/4 ☐ ለ/4

ክፍል 4 ለመጠቀሙ ለጥያቄዎቹ?

- ☐ 0 ምረቃ 6 ወር ይገባል ☐ 12 ምረቃ 20 ወር ይገባል
- ☐ 6 ምረቃ 5 ወር ይገባል ☐ 20 ምረቃ ይገባል
- ☐ 5 ምረቃ ይገባል 12 ምረቃ ይገባል





**AREA OF INTEREST**

**KEY MAP**

**SOURCE OF DRAWING:**  
GSC MAP D1880A (1996) CD DIGITAL MAP  
**PROJECTION:**  
LAMBERT CONIC CONFORMAL  
NAD 27  
**UNITS METRES**

**DRAWING INFORMATION:**

DESIGNED BY:	AJK
REVIEWED BY:	AJK
DRAWN BY:	CPW
DATE ISSUED:	MARCH 21, 2001
PROJECT NUMBER:	20-935
FILE NAME:	20935-M3-02.DWG
REVISION:	0

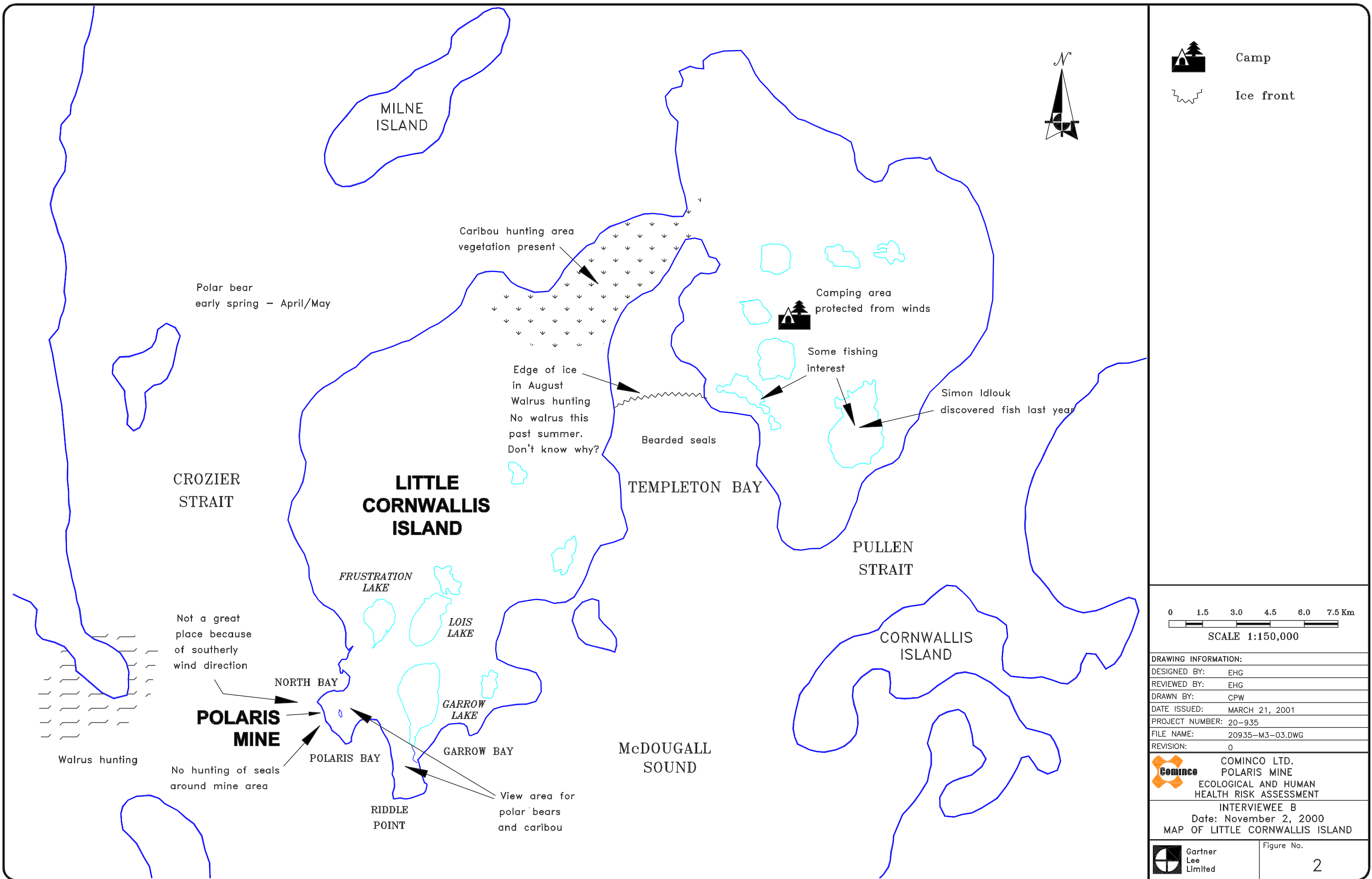
**COMINCO LTD.**  
**POLARIS MINE**  
ECOLOGICAL AND HUMAN  
HEALTH RISK ASSESSMENT



INTERVIEWEE A  
Date: November 2, 2000  
MAP OF LITTLE CORNWALLIS ISLAND

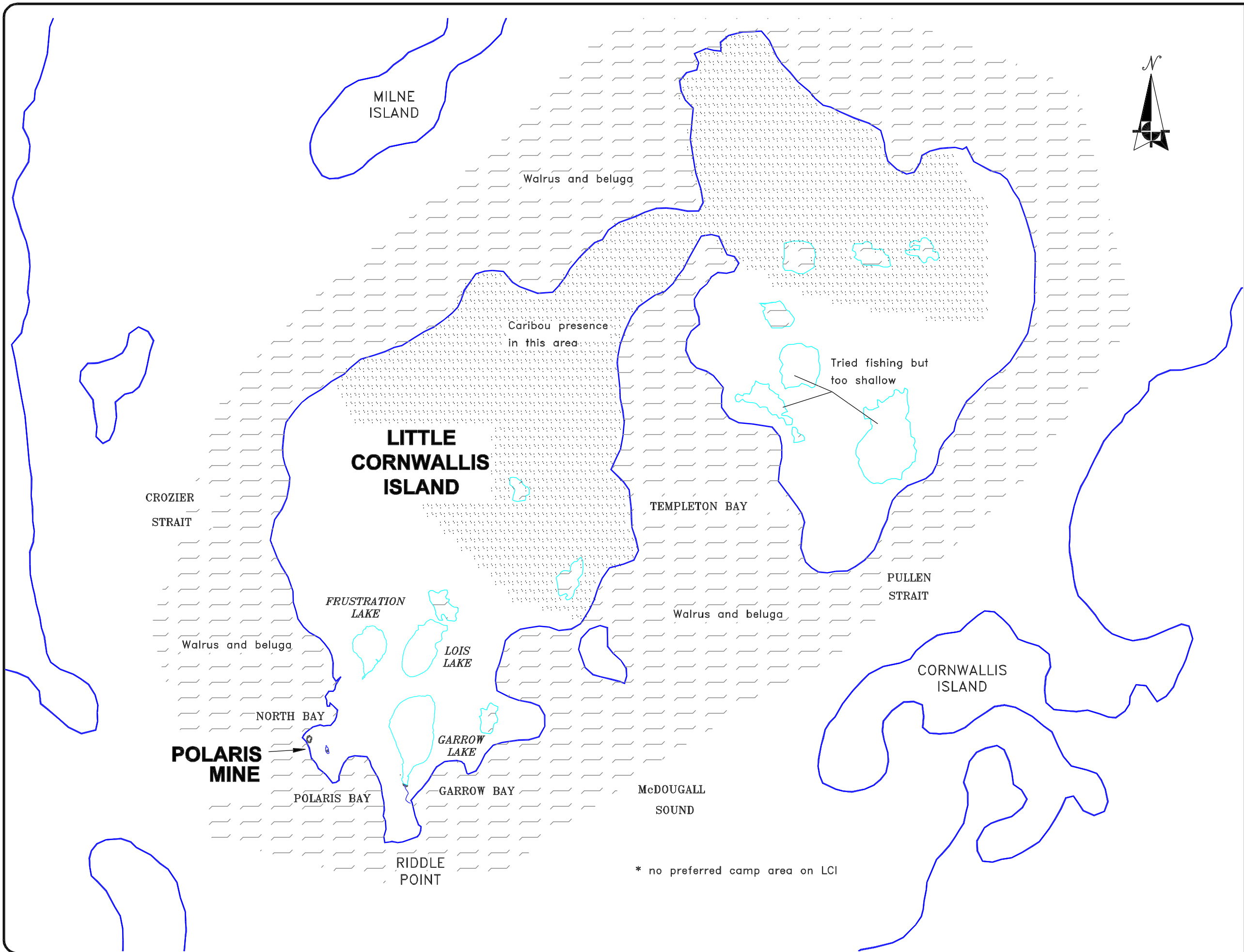
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Lee  
Limited

Figure No.  
**1**

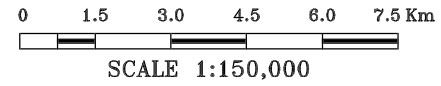





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DRAWN BY:	CPW
DATE ISSUED:	MARCH 21, 2001
PROJECT NUMBER:	20-935
FILE NAME:	20935-M3-03.DWG
REVISION:	0
 COMINCO LTD. POLARIS MINE ECOLOGICAL AND HUMAN HEALTH RISK ASSESSMENT	
INTERVIEWEE B Date: November 2, 2000 MAP OF LITTLE CORNWALLIS ISLAND	
 Gartner Lee Limited	Figure No. 2



Camp  
Ice front




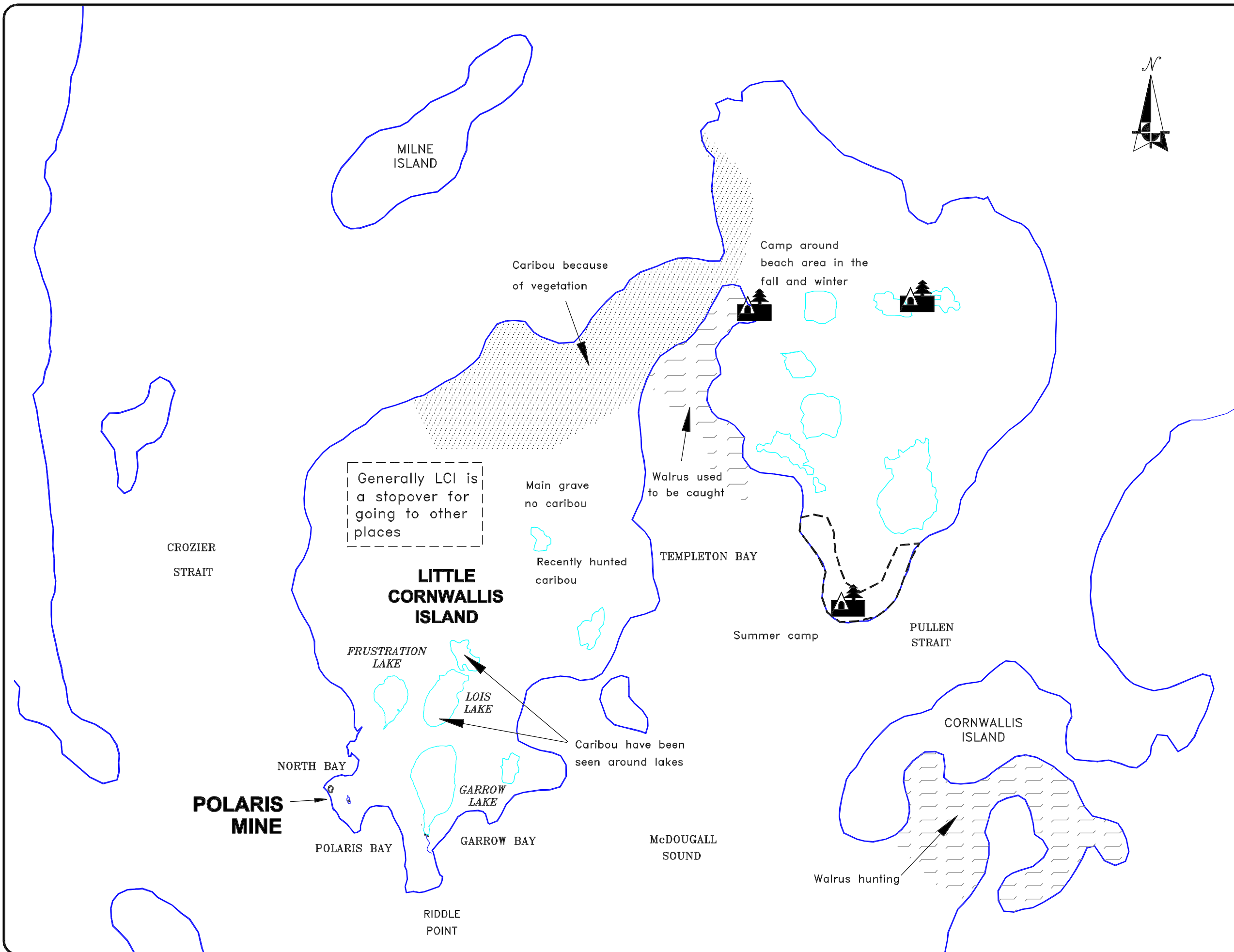
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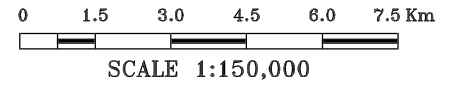
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HEALTH RISK ASSESSMENT

INTERVIEWEE C  
Date: November 2, 2000  
MAP OF LITTLE CORNWALLIS ISLAND


 Gartner Lee Limited	Figure No.  3
---	---------------------



Camp  
Ice front




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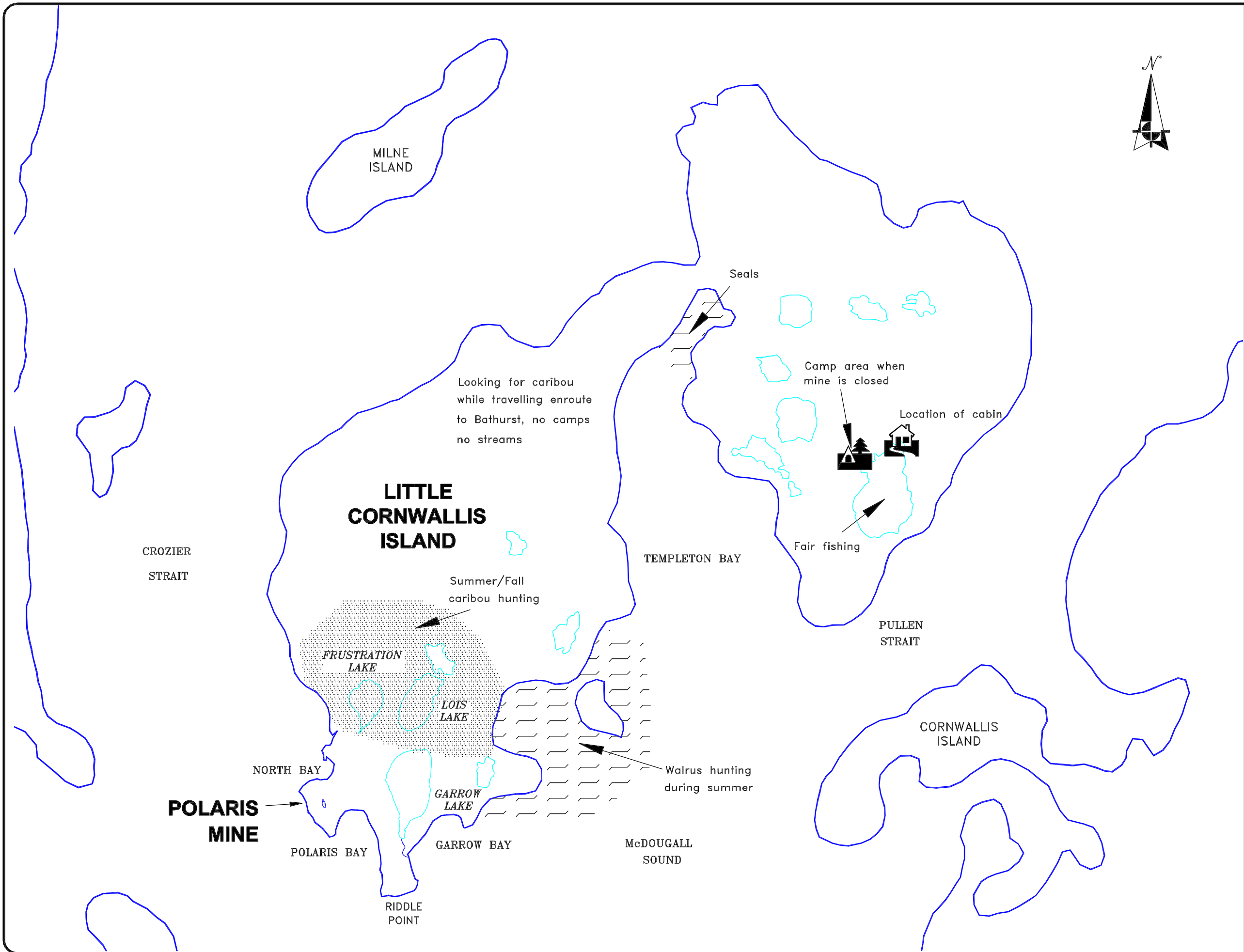
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HEALTH RISK ASSESSMENT

INTERVIEWEE D  
Date: November 2, 2000  
MAP OF LITTLE CORNWALLIS ISLAND



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Lee  
Limited

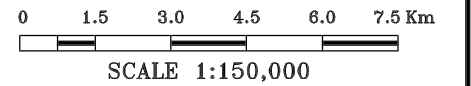
Figure No.  
4




Camp



Ice front




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DRAWN BY:	CPW
DATE ISSUED:	MARCH 21, 2001
PROJECT NUMBER:	20-935
FILE NAME:	20935-M3-01.DWG
REVISION:	0



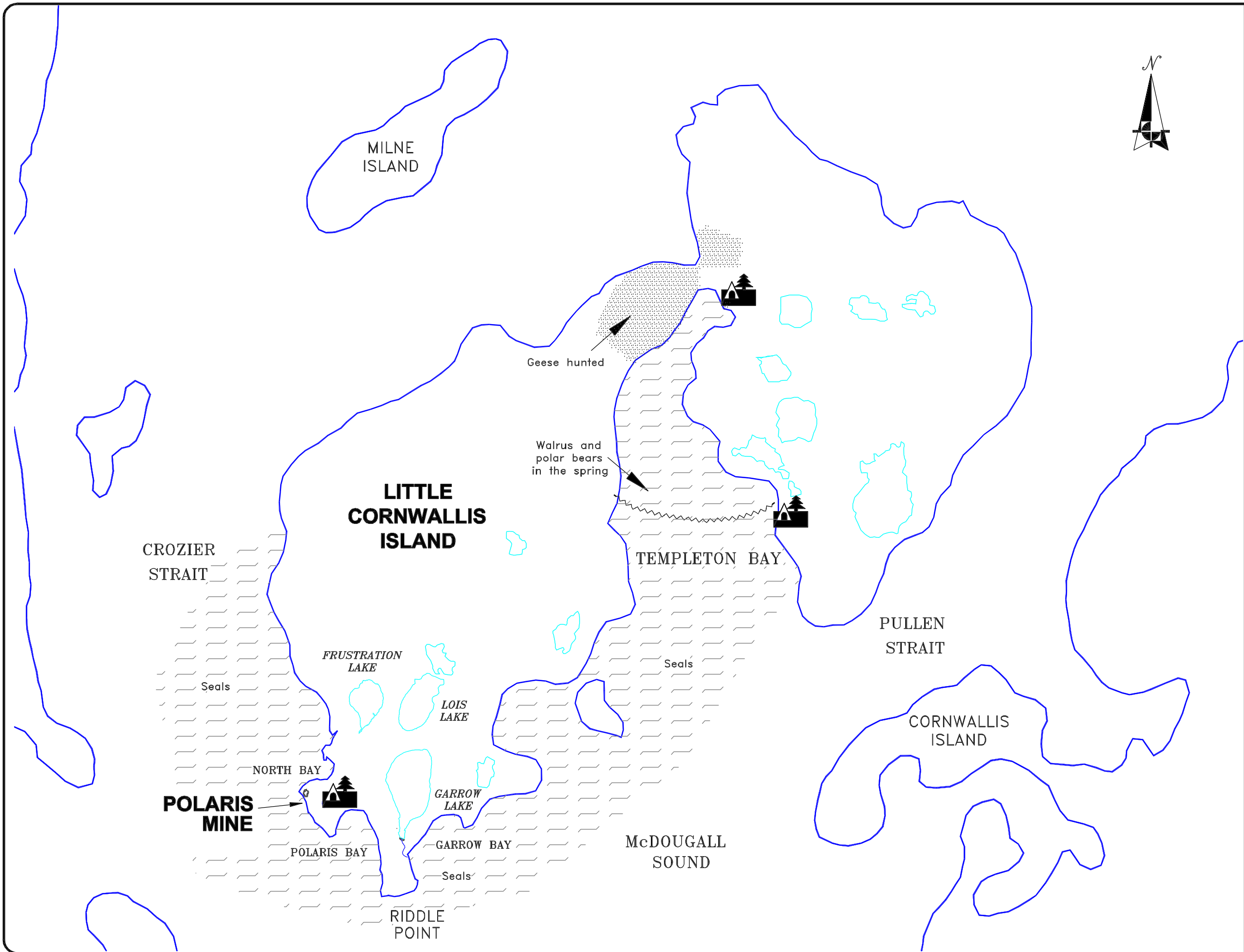
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HEALTH RISK ASSESSMENT

INTERVIEWEE E  
Date: November 2, 2000  
MAP OF LITTLE CORNWALLIS ISLAND



Gartner  
Lee  
Limited

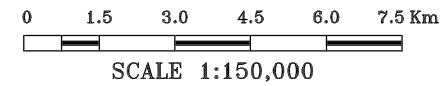
Figure No.  
5




Camp



Ice front



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DRAWN BY:	CPW
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PROJECT NUMBER:	20-935
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MAP OF LITTLE CORNWALLIS ISLAND


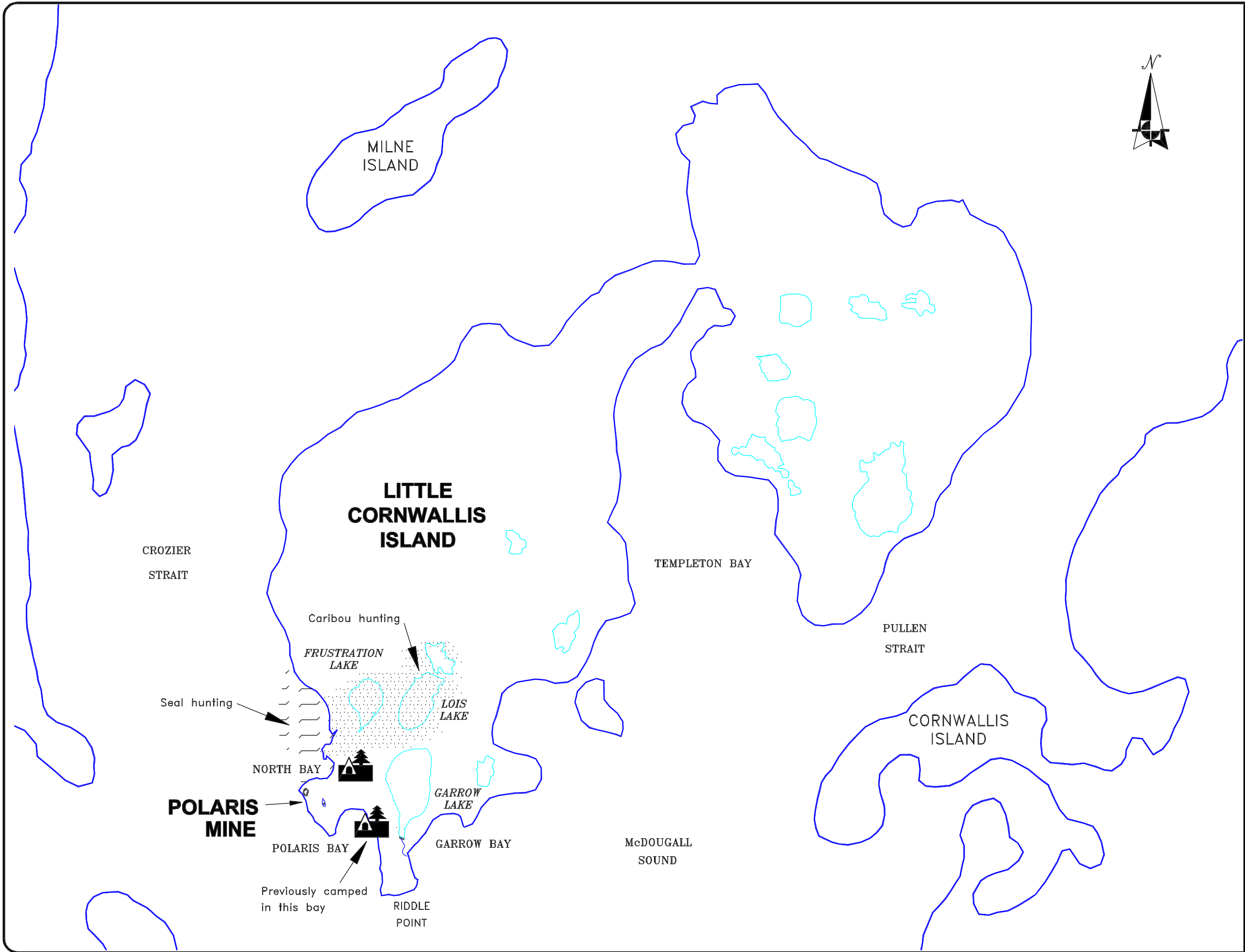
Gartner  
Lee  
Limited

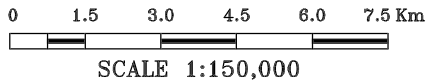
Figure No.  
**6**



Camp



Ice front



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REVIEWED BY:	EHG
DRAWN BY:	CPW
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HEALTH RISK ASSESSMENT

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Date: November 4, 2000  
MAP OF LITTLE CORNWALLIS ISLAND

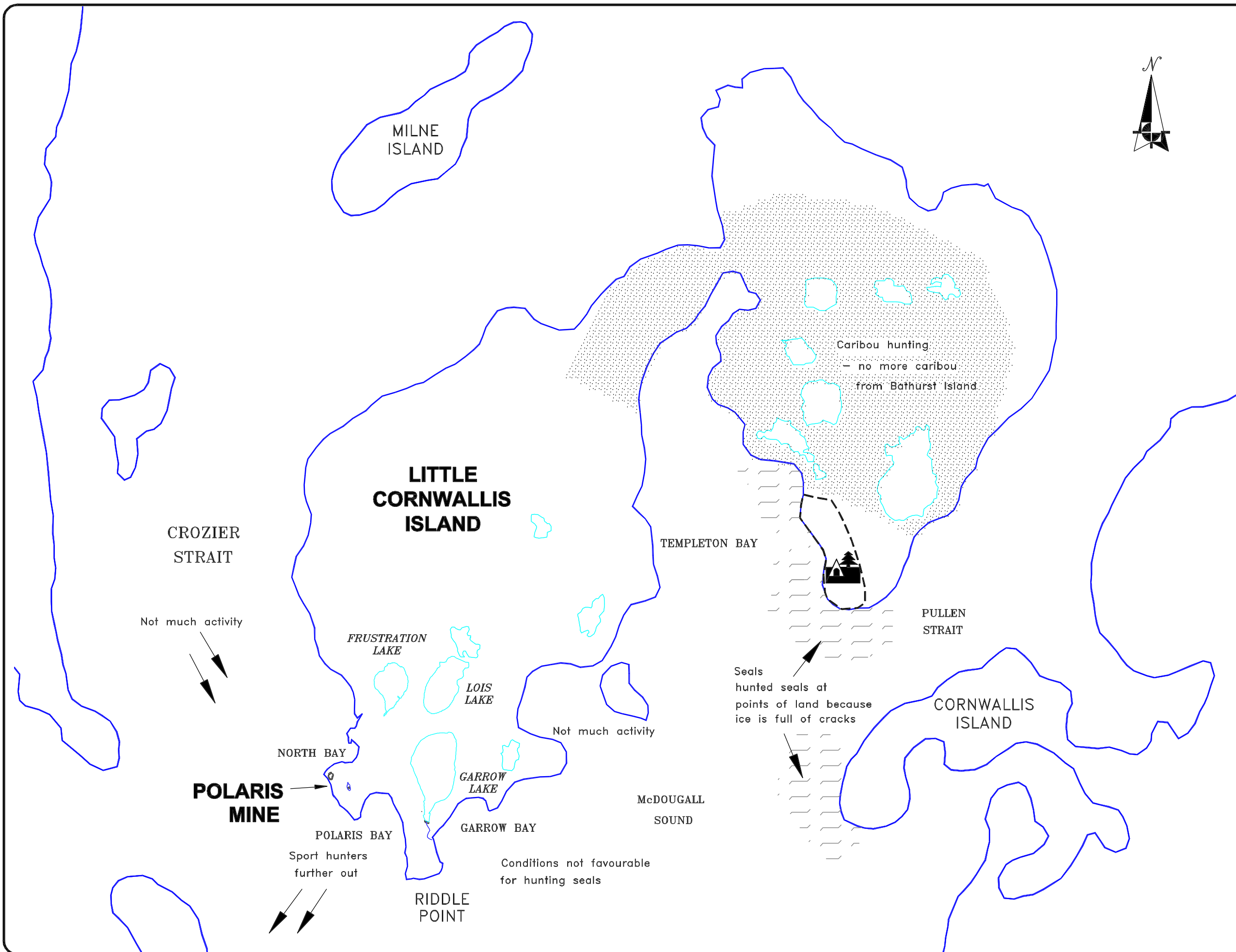


Gartner  
Lee  
Limited

Figure No.

7

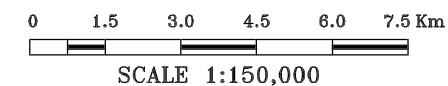




Camp



Ice front



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DRAWN BY:	CPW
DATE ISSUED:	MARCH 21, 2001
PROJECT NUMBER:	20-935
FILE NAME:	20935-M3-08.DWG
REVISION:	0



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ECOLOGICAL AND HUMAN  
HEALTH RISK ASSESSMENT

INTERVIEWEE H  
Date: November 2, 2000  
MAP OF LITTLE CORNWALLIS ISLAND



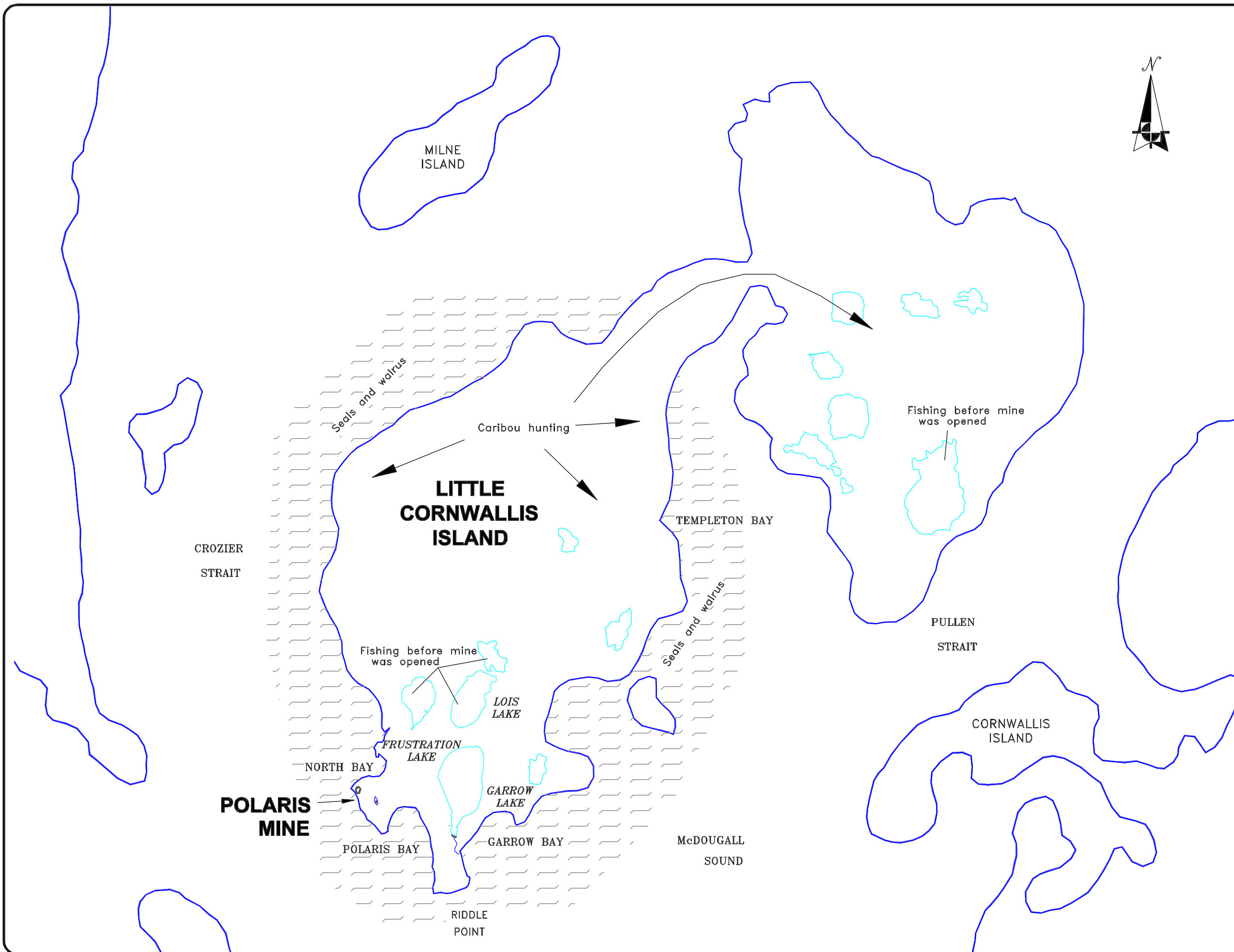
Gartner  
Lee  
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Figure No.

8



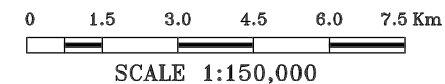





Camp



Ice front



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DESIGNED BY:	EHG
REVIEWED BY:	EHG
DRAWN BY:	CPW
DATE ISSUED:	MARCH 21, 2001
PROJECT NUMBER:	20-935
FILE NAME:	20935-M3-10.DWG
REVISION:	0

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HEALTH RISK ASSESSMENT	

INTERVIEWEE J	
Date: November 4, 2000	
MAP OF LITTLE CORNWALLIS ISLAND	



Gartner  
Lee  
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Figure No.

10