APPENDIX A COMMUNITY CONSULTATION

NANISIVIK MINE HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT: SUMMARY OF RESULTS OF LIMITED COMMUNITY CONSULTATION

A consultation was conducted on-site with members of the communities of Nanisivik and Arctic Bay in September 2002 by Robert McCollough (JWEL) and Inuktitut translator Mishak Allurut. The consultation focussed on those residents who hunt and fish and would therefore be most likely to use the land in the vicinity of Nanisivik Mine after the mine has been closed and the reclamation processes has been completed. Residents offered the opportunity to be interviewed through contacting community elders and via broadcast on the community radio stations. Residents were given the choice of being interviewed by Mr. McCollough and Mr. Allurut, or by Mr. Allurut alone. Interviews were conducted in either English or Inuktitut (most were in Inuktitut) and copies of the interview questions were provided in both languages (see Appendix A-1). A total of 16 people were interviewed, 13 of whom were Inuit and lived in Arctic Bay (see list of participants in Appendix A-2). Responses to questions were recorded on questionnaire forms and these responses are compiled and provided in Appendix A-2. One interview was not completed, and has not been counted as part of the limited community consultation process.

Interviews in Nanisivik were conducted on September 23 and 24, 2002. At the request of the residents, interviews took place in their location of choice, rather than at the hotel or hamlet office. Some wanted to be interviewed in their home and others wanted to be interviewed in a traditional manner in the carving and skinning huts away from the home.

Most of the people interviewed were elders, most of whom had been born and raised in Inuit camps on the land and were brought to Arctic Bay to live by the government collection program. Typically, anyone under 30 to 35 years of age would not hunt or fish for livelihood and would rarely go hunting or fishing for sport.

APPENDIX A-1 COMMUNITY QUESTIONNAIRE (English and Inuktitut)

	Location of Interview:					
	Interviewer:					
HIGH ARCTIC COMMUNITY QUESTIONNAIRE						
CanZinco Ltd. retained Jacques Whitford Environment Limited to conduct a risk assessment of its Nanisivik Mine near Arctic Bay on Baffin Island (see attached map). This study will be used to provide working guidelines for the final reclamation of the Nanisivik site to ensure that the environment and people using the site are protected. To conduct this assessment, it is important that we gain a clear understanding of the expected type and amount of activity from your community in these areas once CanZinco Ltd. has shut down its operation. By providing answers to the following questionnaire, you are enabling the study team to incorporate information that most accurately reflects Inuit lifestyle.						
	This infor		-		ntended for the use of the er purposes. Thank you	
Section 1 – Participar	ıt Informat	tion				
Date:						
Name of Participant:						
Age:		_	Community:			
Sex:		Male			Female	

Section 2 – Hunting and Fishing Patterns

Do you hunt or fish for	food?			
hunting		yes		no
fishing		yes		no
How often do you hunt	for food?			
		times per week		times per year
		times per month		rarely
How often do you fish f	for food?			
		times per week		times per year
		times per month		rarely
Where do you travel to	hunt/fish	for food?		
Do you ever hunt near N	Nanisivik'	?		
		yes	no)
If yes, where do you hu	nt in the l	Nanisivik area		
How often do you hunt	near Nan	isivik?		
		times per week		_ times per year
		times per month		_ rarely

What animals do you hunt near	Nanisivik?		
Do you ever fish near Nanisivik	?		
	yes		no
If yes, where do you fish near N	Janisivik? (please ma	rk on map)	
How often do you fish near Nan	nisivik?		
	_ times per week		 times per year
	_ times per month		 rarely
What fish do you catch there?			
How often do you eat wild game	e?		
	_ times per week		 times per year
	_ times per month		 rarely
How often do you eat wild game	e from the Nanisivik	area?	
	_ times per week		 _ times per year
	_ times per month		 _ rarely

How much of this meat	do you u	sually eat for a single meal?		
		small serving		
		medium serving		
		large serving		
How often do you eat fi	sh?			
		times per week		_ times per year
		times per month		_ rarely
How often do you eat fi	sh from t	he Nanisivik area?		
		times per week		_ times per year
		times per month		_ rarely
How much of these fish	do you u	sually eat for a single meal?		
		small serving		
		medium serving		
		large serving		
Section 3 – Land Use				
Once the mine has been	closed, h	now many times a season would yo	u visit Na	nisivik?
-		times per winter		times per summer
<u>-</u>		times per spring		times per fall
Once the mine has been	closed, ł	now long would you stay per visit?		
		a few hours		14 to 30 days
		1 to 3 days		30 to 90 days
	П	3 to 14 days		more than 90 days

Once CanZinco Ltd. rei	moves its	facilities, would you be:			
		less likely to visit Nanisivik?			
		equally likely to visit Nanisivik?			
		more likely to visit Nanisivik?			
How many times a seas	on do yo	u visit Nanisivik now?			
		times per winter		times per summer	
		times per spring		times per fall	
How long do you typica	ally stay _l	per visit?			
		a few hours		14 to 30 days	
		1 to 3 days		30 to 90 days	
		3 to 14 days		more than 90 days	
While staying at Nanisi	vik, when	re would you camp? (please mark	on map)		
What type of shelter do	you use	while camping?			
Does the shelter have an	n open flo	oor?			
		yes		no	
When you travel to hun	t or fish,	do you ever bring anyone else with	ı you?		
		yes		no	

What are the ages of the people that come along?					
_	0 to 6 months		12 years to 20 years		
_	6 months to 5 years		20 years and above		
_	5 years to 12 years				
How do the others who do not hunt or fish spend their time while out on the land?					
Do you bring food from	home when you travel to hu	nt or fish?			
	yes		no		
Where do you get your v	vater from when you travel to	o hunt or fish?			
Section 4 – Ecological K	Knowledge				
What animals or birds w common would these be	ould you expect to find aroun?	nd the townsite or min	ne areas and how		
Animals / Birds	rarely present	usually present	always present		

What fish or marine animal these be?	s would you expect to find	near the dock and how	common would
Fish / Animals	rarely present	usually present	always present
Are there any other tradition that we should know about		places around the mine	e, townsite, or dock

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:

Dr. David Rae (Jacques Whitford Environment Limited) at (506) 457-3200 or

Dr. Malcolm Stephenson (Jacques Whitford Environment Limited) at (506) 457-3200.

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בי כ $^{\circ}$ כ $^{\circ}$ כ $^{\circ}$ כ $^{\circ}$ S $^{\circ}$ Dr. David Rae (Jacques Whitford Environment Limited) at (506) 457-3200 or

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APPENDIX A-2 LIST OF PARTICIPANTS

HIGH ARCTIC COMMUNITY QUESTIONNAIRE

The following is a list of those members of the community who took part in the limited community consultation process in Arctic Bay and Nanisivik.

Names of Participants	Community	
1. Mishak Allurut	Arctic Bay	
2. Muktar Akumalik	Arctic Bay	
3. Isaac Shooyouk	Arctic Bay	
4. Olayuk Kigutikajuk	Arctic Bay	
5. Ikey Kigutikajuk	Arctic Bay	
6. Ipeelee Koonoo	Arctic Bay	
7. Jotah Muckpa	Arctic Bay	
8. Joeli Qamaninq	Arctic Bay	
9. Joanasie Akumalik	Arctic Bay	
10. Wayne Osborne	Nanisivik	
11. Frank McDermott	Nanisivik	
12. Kudloo Ettuk	Arctic Bay	
13. Atagotak Ipeelee	Arctic Bay	
14. Aimo Muckpaloo	Arctic Bay	
15. Koonoo Oyukuluk	Arctic Bay	
16. Tommy Kilabuk	Arctic Bay	Survey incomplete and not counted.

Note: only those community members who identified themselves as hunters and/or fishermen were asked to complete the survey.

APPENDIX A-3 COMPILED RESPONSES TO HIGH ARCTIC COMMUNITY QUESTIONNAIRE AND MAPS

Compiled Responses to Questionnaire

Question Response		Comments	
9	SECTION 1 – DEMOGR	RAPHICS	
Age	40 - 73 years old		
Sex	13 Male 2 Female		
Community	13 Arctic Bay 2 Nanisivik		
SECTION	2 – HUNTING AND FIS	SHING PATTERNS	
Do you hunt for food?	14 Yes 1 No		
How often do you hunt for food?	1 - 7 times per week(4) 4 - 30 times per month(2) 200 - 365 times annually (1) rarely	 "Depending on weather, often." "As often as possible." (5) "Weekends hunter" "Spring & Summer, 3x/mo & 16x/yr" "In Winter more often, and Spring" "Depends on gas & supplies & time" "In good weather, every day, 0 in bad weather." 	
Do you fish for food?	14 Yes 1 No (but this respondent indication)	ated later on that they do fish near Nanisivik)	
How often do you fish for food?	7 times per week(2)times per month 1 - 6 times annually(3) rarely	 "Spring & Summer, 4x/wk for 3 months" "Winter only" "October – June (Fall – Spring)" "As often as possible" (3) "Every chance I get" (2) "Spring & Fall – on fresh ice" "Seasonal – Fall & Spring & Summer" 	
Where do you travel to hunt/fish for food?	Admiralty Inlet	7) 1) 1)	
Do you ever hunt near Nanisivik?	14 Yes 1 No		
If yes, where specifically do you hunt in the Nanisivik area?	 "Close by - sand and across sa Strathcona Sound (5) "Out past Kuhulu Lake and o		
How often do you hunt near Nanisvik?	$\frac{1-2}{4}$ times per week(2) $\frac{4}{2}$ times per month(1) $\frac{2-9}{2}$ times annually(2) rarely	 One month in each Spring & Fall 2 months / year "Less than in the past" "As often as possible" (2) In Winter (6x/month) "Seasonal – Fall" "Mainly in Winter" "In Winter more often, rarely in summer" "Used to be every day when there was caribou but not anymore" "Not as much as before because no or hardly any seals left" 	

Question	Response	Comments
What animals do you hunt near Nanisivik?	Ptarmigan Rabbit Goose Fox Seal (Bearded, Ringed, and Hamal Caribou Narwhal	(6) (4) (5) arp)(11) (4)
Do you ever fish near Nanisivik?	14 Yes 1 No	
If yes, where specifically do you fish in the Nanisivik area?	Kuhulu Lake"On other side of Sound in lak"Across Nanisivik"	res"
How often do you fish near Nanisivik?	1 time per week(1)times per month 2 - 6 times annually(2) rarely(2)	 "In the Fall only" "In the Fall, depends on weather" "In Fall" "Seasonal – Fall" "Only in the Fall" "For two years in the Fall" "Fall for about 2 weeks" "Used to be in the Fall"
What fish do you catch near Nanisivik?	Land-locked Char(11)Arctic Char(2)	
How often do you eat wild game?	<u>4 – 7</u> times per week(13) <u>1</u> time per month(1) _ times annually rarely(1)	Staple diet (7x/wk) (11)"Whenever available"
How often do you eat wild game from the Nanisivik area?	times per week1 time per month(1) times annually rarely(7) never(1)	 "Periodically, when seals or ptarmigan are caught" "Whenever available. Whenever they catch some" "When he gets some yes" "Depends on if I catch one" "Only when he catch one near Nanisivik" "Seals when available" "Whenever available"
How much of this meat do you usually eat for a single meal?	5 Small serving 8 Medium serving 1 Large serving	Ate large servings when younger
How often do you eat fish?	$\frac{1-7}{1-2} \text{ times per week} (10)$ $\frac{1-2}{1-2} \text{ times per month} (1)$ $\frac{1}{1-2} \text{ times annually rarely}$	 Staple diet (7x/wk) (7) "Small serving" "Whenever available" "More often in the Fall" "In Spring & Fall" "When available"

Question	Response	Comments
How often do you fish from the Nanisivik area? How much of this fish do you	1 - 7 times per week	 "Spring & Fall" "When they catch it" "In the Fall" "Whenever he gets it, Fall usually" "Only in the Fall" "Only those times he caught fish" "Whenever he catches them" "4 or 5 small fish" (1)
usually eat for a single meal?	1 Large serving	TIGE
	SECTION 3 – LAND	
Once the mine has been closed, how many times a season would you visit Nanisivik?	<u>2–17</u> times per winter (2) <u>a few–14</u> times per spring (3) <u>a few–14</u> times per summer (3) <u>1–17</u> times per fall (4) never (5)	 "3x/summer for 1 week, for hunting purposes" "Nov-Dec-Jan = too dark, otherwise 2x/wk" "Depends on Husband's hunting" "Occasionally" "Only Airport" "To hunt in Strathcona Sound" "Hunting as before" "1980s did visit more often than today"
Once the mine has been closed, how long would you stay per visit?	6 a few hours 1 to 3 days 1 to 3 days 14 to 30 days 10 to 90 days more than 90 days no time	"Hunting in the area" "Across from Nanisivik"
Once CanZinco Ltd. removes its facilities, would you be:	7 less likely to visit Nanisivik? 4 equally likely to visit Nanisivik? 2 more likely to visit Nanisivik?* 2 never visit 1 N/A (because current resident)	* to salvage materials
How many times a season do you visit Nanisivik now?	never visit	 "Variable depending on the meetings" (Elders Association Meetings held in Nanisivik) "5 times in a year" "3 times in his life" "Once a week in Summer, once a month in Winter" "7/9 times/year" "Once a year" "Nov-Dec-Jan = too dark, otherwise 2x/wk" "When working every day but when not working only once in a year"

Question	Response	Comments	
How long do you typically stay per visit?	2 no time 10 a few hours 1 1 to 3 days 3 to 14 days 14 to 30 days 30 to 90 days more than 90 days currently a resident		
While staying at Nanisivik, where would you camp?	 Kuhulu Lake Along Beach Lone Island English Bay Graveyard Point Cape Strathcona Traditional camps Across from Nanisivik Nanisivik dock area 		
What type of shelter do you use while camping?	Tent Igloo (in winter)	· · ·	
Does the shelter have an open floor?	• Igloo (in winter)(2) 10 Yes 4 No		
When you travel to hunt or fish, do you ever bring anyone else with you?	14 Yes 1 No		
What are the ages of the people that come along?	0 to 6 months 6 months to 5 years 5 years to 12 years 12 years to 20 years 20 years and above	"In Spring – all children come, in Winter – only wife"	
How do the others who do not hunt or fish for food spend their time while out on the land?	 Collecting blueberries, plants, seaweeds Preparing/drying fish Skinning/drying hides Sewing clothes/mending tents Cooking meals "Involved in hunting facilities" "They are involved in hunting" "All involved" (3) "All involved in hunting" "All hunt & fish" "All engaged in hunting activity" "No comment" "Social time" "Most or all people are involved in activities related to hunting or fishing." 		

Question	Response	Comments		
Do you bring food home when yo	<i>u</i> <u>15</u> Yes	"Limited"		
travel to hunt or fish?	No	"Once in a while"		
Where do you get your water from when you hunt or fish?	something." "Where I can fin "Not bring water "Not bring water "Winter-snow, so "Bring water/or of "Water from hun "Streams" "Gets water from "River, snow, lal "I get my water fom "Get water from	from the hunting" gets water from the camp area" ammer-bring own or multi year ice for very campsite where there's water" ting area" a campsite" tee" from the land/campsite" the land" g water but mostly drink from hunting area or melt snow"	water"	
SECTION 4 – ECOLOGICAL KNOWLEDGE				
What animals or birds would you	expect to find around the	townsite or mine areas and how comm	on would these be?	
Animals/Birds	Rarely Present	Usually Present	Always Present	

Ptarmigan* 1+1(summer)+1(seasonal) Goose 1+1(summer) Snow Goose 1(in season) Fox (Arctic) 3 5 Killdeer 1 (1950s) 1 (present day) Raven* Seagull 3+1(seasonal) 1+1(in season) • • Gyr Falcon Duck* 1(seasonal) 1 Small birds** 1(seasonal) Fulmar* 1 Loon* 1 1(in season) Jaeger* Snowy Owl* Oldsquaws 1(in season) Snow Bunting* 1(in season) Rabbit/Arctic Hare 9 Wolf (at landfill) Caribou 2 + 1(when they were here) Lemming 1 Weasel

What fish or animals would you expect to find near the dock and how common would these be? Animals/Birds **Rarely Present Usually Present Always Present** Seal⁺ 9+1(summer)+1(summer and winter) 4+1(summer) Ringed Seal Bearded Seal 1+1(seasonal) Harp Seal*+ 3+1(seasonal)+1(summer) 1(summer) Narwhals⁺ 3 6 + 2(summer) + 1(seasonal)Cod 3 1+1(summer) Sculpin 1 1

* Listed, but no occurrence given on survey (* = once, ** = twice)

Animals/Birds	Rarely Present	Usually Present	Always Present
• Whale	1		
Killer Whale	1		
Bow Head Whale	1		
Mussels / Clams		1	
• Krill		1	
Polar Bear*	1		
• Fulmar*+			
• Birds		1	
• Fox		1	

^{*} Listed, but no occurrence given on survey

Are there any other traditional resources or important places around the mine that we should know about?

- "Eskimo Beach"
- "Wants to keep the roads for hunting"
- "Old campsites (Eskimo Beach)"
- "Dock area"
- "General hunting area"
- "Gateway to Pond Inlet"
- "Wants to keep the roads for hunting"
- "Gateway to Caribou hunting area (across sand)"
- "Would like to see the tent rings, traditional marks/cairns on land to be carefully preserved"
- "Surveying markers be kept"
- "Any bones of animals being visible should be kept on top not cover the bones"
- "Floe edge is also a main hunting area in spring for seal, whale, walrus, and water birds"
- Even when the tailings pond is covered since the global warming might melt and the contents of the tailings might seep out
- "Wants Nanisivik company to recognize Arctic Bay for contribution of mine development"

Other Notes:

- "No real hunting grounds close to Nanisivik. To hunt caribou we travel with Inuit for hours out on the land to the south. People travel south to hunt geese and gather eggs."
- "There used to be a seal breeding ground in Strathcona Sound, not now, seals all gone, no more babies."
- "There is a place where the narwhals beached themselves in the past. There are lots of skeletons of whales in this one location and the story is that they beached themselves."
- "He noticed, once when he climbed a mountain (King George's Mtn) he noticed where the soot fell in a circular pattern very obvious to him, around the mine. Once across the Sound, the snow was clean. The hunters' common sense could tell them the mine was contaminated land."
- "Refused to eat seals caught in Strathcona Sound because of abnormality"

⁺ Specified March – October (seasonal – ice breakup & ice free times)



APPENDIX B

TOXICITY PROFILES



APPENDIX B: TOXICITY PROFILES

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1.0 TOXICITY PROFILES

1.1 Human Health Toxicity Profiles

1.1.1 Cadmium

1.1.1.1 Assessment of Carcinogenicity

Epidemiological studies demonstrate increased incidence of lung cancer in workers exposed to cadmium via the inhalation route; however, the studies did not control for factors such as smoking and simultaneous exposures to other metals so the causal relationship is somewhat controversial. Oral exposure to cadmium has not been associated with cancer in humans or animals.

The United States Environmental Protection Agency has classified cadmium as a probable human carcinogen (Group B2) when inhaled, based on limited human and sufficient animal data (US EPA 1985). Health Canada (1996a, b) has classified cadmium as a Group II carcinogen.

1.1.1.2 Susceptible Populations

Populations which may be unusually susceptible to cadmium exposure are those with a genetic predisposition to lower inducibility of metallothionein, the enzyme which sequesters cadmium. Dietary deficiencies which lead to depleted levels of calcium or iron in individuals may result in increased absorption of cadmium from the gastrointestinal tract. Infants and children may have increased uptake of cadmium via the gastrointestinal tract and higher concentrations of cadmium in the bone.

1.1.1.3 Selection of Toxicity Values

ATSDR (1998) has developed a chronic oral minimum risk level (MRL) of $0.2 \,\mu g/kg$ -day for cadmium. The chronic MRL is derived from a No Observed Adverse Effect Level (NOAEL) of $2.1 \,\mu g/kg$ -day from a study of cadmium accumulation in the kidneys of Japanese farmers living in an area of Japan with highly elevated cadmium levels. An uncertainty factor of 10 was used to account for variability in the human population.

The United States Environmental Protection Agency has developed oral reference doses for cadmium for food and water. The RfD $_0$ for food is 1.0 μ g/kg-day and for water is 0.5 μ g/kg bw-day (US EPA 1994a). The highest cadmium level in the human kidney which does not produce proteinuria (excretion of low weight molecular proteins into the urine) has been determined to be 200 μ g Cd/g of wet kidney cortex. A toxicokinetic model was used to determine the level of chronic oral exposure that would result in a cadmium kidney concentration of 200 μ g Cd/g of wet kidney cortex. The toxicokinetic model assumes that 0.01% of the body cadmium kidney burden is eliminated daily and that absorption of

cadmium from food and water are 2.5% and 5% respectively. The NOAELs for chronic cadmium exposure were determined to be 5.0 and 10 μ g/kg-day for food and water, respectively. An uncertainty factor of 10 to account for human variability was applied to the NOAELs to develop the reference doses for food and water.

The Joint Expert Committee on Food Additives (WHO 1993) proposed that the total daily intake of cadmium should not exceed 1 μ g/kg bw-day. This intake was designed to keep the cadmium levels in the renal cortex below 50 μ g/g, and assumed an absorption rate for dietary cadmium of 5% and a daily excretion rate of 0.005% of body burden.

Health Canada (1996a) has adopted the 1 μ g/kg bw-day value as a provisional tolerable daily intake (PTDI) for both children and adults.

The US EPA (1994a) has developed an inhalation unit risk of $1.8 \times 10^{-3} \, (\mu g/m^3)^{-1}$. This unit risk is based on lung and upper respiratory tract cancers in cadmium production workers (Thun *et al.*, 1985). The air concentration at the 10^{-5} lifetime cancer risk level (1-in-100,000) is $0.006 \, \mu g/m^3$.

The World Health Organization (WHO 2000) has an annual guideline value (noncancer) of $0.005 \,\mu\text{g/m}^3$. Health Canada (1996b) has calculated a TC_{05} of $5.1 \,\mu\text{g/m}^3$. This TC_{05} can be converted to an inhalation unit risk value of $9.8 \times 10^{-3} \,(\mu\text{g/m}^3)^{-1}$ using a target risk value of 1×10^6 , and converted to an inhalation slope factor using an inhalation rate of $16.2 \,\text{m}^3$ /day and a body weight of $70.7 \,\text{kg}$.

The estimate of non-cancer oral potency from Health Canada (PTDI) will be used to assess potential hazards associated with oral exposures to cadmium in this report. The Health Canada TC_{05} provides a more conservative unit risk estimate of the potency of inhaled cadmium. Both studies appear to be based on the same data. Therefore the inhalation unit risk derived from the Health Canada TC_{05} will be used to assess potential cancer risks associated with inhalation exposures to cadmium. The toxicity values used to assess the potential risks associated with ingestion and inhalation exposure to cadmium are summarized in Table B-1.

Table B-1 Selected Toxicity Values for Cadmium

Route of Exposure	TRV	Toxicological Basis	Source Agency			
Non-Cancer Effects	Non-Cancer Effects					
Ingestion – toddler and adult	1 μg/kg bw-day	Kidney damage in humans	Health Canada, 1996a			
Inhalation	NA					
Cancer Effects						
Ingestion	NA					
Inhalation	$9.8 \times 10^{-3} (\mu g/m^3)^{-1}$	Lung cancer in cadmium workers	Health Canada, 1996b			

The biological end-point of greatest concern differs between oral and inhalation exposures. Therefore, the potential hazards associated with oral exposures and the potential cancer risks associated with inhalation exposures to cadmium will be assessed separately.

1.1.1.4 Bioavailability of Cadmium

Oral Route of Exposure

Oral absorption of cadmium in humans generally is reported to be very low (1 to 7 percent) (ATSDR 1997). The total retention of cadmium in the bodies of humans has been measured following ingestion of radioactive cadmium. About 25% of a dose of cadmium administered mixed with food to 5 healthy adults was retained after 3-5 days, but retention decreased to about 6% after about 20 days (Rahola *et al.* 1973). Similar results were obtained with 14 healthy adults, with an average of 4.6% of cadmium chloride in water taken with a meal retained in the body 1-2 weeks after a simultaneously administered fecal marker (trivalent chromium) had been completely excreted (McLellan *et al.* 1978). The influence of chemical complexation of cadmium on human absorption was evaluated in seven volunteers who ingested brown crab meat (hepatopancreas) that had been labeled with radioactive cadmium chloride by prior feeding of the crabs (Newton *et al.* 1984). Whole-body counting was used to evaluate uptake. Whole-body retention in the volunteers ranged from 1.2 to 7.6% with a mean of 2.7% (Newton *et al.* 1984), only slightly lower than the values of 4.6-6% obtained using dissolved cadmium ion (McLellan *et al.* 1978; Rahola *et al.* 1973). These results indicated that, in general, cadmium absorption from food is not dependent on chemical complexation.

Evidence that the bioavailability of cadmium in soil may be reduced compared to the bioavailability of soluble cadmium forms is available from a limited number of studies. Several studies have reported reduced oral bioavailability of a soluble cadmium form, cadmium chloride, mixed with soil (Griffin *et al.* 1990; Schilderman *et al.* 1997). For cadmium in weathered soil, data are available for soil from a single site (the site of a former zinc smelter) that has been evaluated in vivo in rats (Schoof and Freeman 1995; PTI 1994). A relative cadmium bioavailability estimate of 33 percent was obtained based on comparison of liver and kidney tissue concentrations in animals fed rodent chow mixed with soil, versus those fed rodent chow mixed with cadmium chloride.

Dermal Route of Exposure

An in vitro study of dermal absorption in human cadaver skin of cadmium chloride mixed with soil yielded an estimate of 0.02 to 0.07 percent absorption based on cadmium in receptor fluid (Wester *et al.* 1992). An additional 0.06 to 0.13 percent of the dose was retained in the skin. The US EPA default value of 1.0 percent for dermal absorption of cadmium compounds from soil is more than 10 times higher than the maximum percent of the cadmium chloride dose reaching the receptor fluid and 5 times

higher than the maximum combined percent dose in receptor fluid and skin. Dermal absorption of cadmium from weathered soils may be even lower.

Selected Values

Based on the above review, oral bioavailability is set at 10% to ensure a protective assessment. Dermal bioavailability is set at the US EPA default of 1% which is likely to overestimate dermal absorption.

1.1.2 Lead

1.1.2.1 Assessment of Carcinogenicity

Epidemiological studies of occupationally exposed adults were not able to demonstrate an increase in cancers among an exposed population compared to a control group. The International Agency for Research on Cancer considers the overall evidence of lead carcinogenicity in humans to be inadequate. The US EPA (1993) has classified lead as a probable human carcinogen based on sufficient animal evidence but did not recommend derivation of a quantitative estimate of oral carcinogenic risk due to a lack of understanding of the toxicological and pharmacokinetic characteristics of lead. Neurobehavioural effects of lead in children were considered to be the most relevant endpoints in determining a toxicity value.

CCME (1992) classified lead as Group IIIB – possibly carcinogenic to man (inadequate data in humans, limited evidence in animals) according to the classification scheme of the Environmental Health Directorate of Health and Welfare Canada. Chemicals classified in Group IIIB are treated as non-carcinogens and are evaluated against a Tolerable Daily Intake (TDI), based on a NOAEL.

1.1.2.2 Susceptible Populations

There is a very large database that documents the effects of acute and chronic lead exposure in adults and children. Extensive summaries of the human health effects of lead are available from a number of sources including Health Canada (1996c), the US EPA Integrated Risk Information System (IRIS) database and the Association for Toxic Substances and Disease Registry (ATSDR). These reviews show that infants, young children up to the age of six, and pregnant women (developing fetuses) are the most susceptible.

1.1.2.3 Selection of Toxicity Values

The WHO (1986) established a provisional tolerable weekly intake (PTWI) for children of 25 μg/kg bw, equivalent to approximately 3.57 μg/kg bw-day from all sources. This PTWI was established based on metabolic studies in infants (Ryu *et al.* 1983; Ziegler *et al.* 1978) showing that a mean daily intake of 3-

 $4 \mu g$ Pb/kg body weight was a NOAEL and was not associated with an increase in blood lead levels or in the body burden of lead. WHO more recently extended this PTWI to all age groups to protect other sensitive population groups, such as women of child-bearing age.

The Ontario Ministry of Environment and Energy (MOEE 1994) derived a lead intake of concern (IOC) for a toddler of 3.7 μ g/kg bw-day based on a blood lead level of concern of 10 μ g/dL, in close agreement with the values derived by the WHO. The OMOE applied an uncertainty factor of 2 to derive an IOC for populations of 1.85 μ g/kg bw-day, intended to represent a maximum intake not to be exceeded. As noted by MOEE, there was no scientific rationale for the size of the uncertainty factor, only judgement based on the quality and quantity of data.

Health Canada (1996c) adopted the WHO toxicity value for the derivation of the CCME Soil Quality Guidelines. The toxicity values used to assess the potential risks associated with ingestion and inhalation exposure to lead are summarized in Table B-2.

Table B-2 Selected Toxicity Values for Lead

Route of Exposure	TRV	Toxicological Basis	Source Agency			
	Non-Cancer Effects					
Ingestion	3.57 µg/kg bw-day	Blood lead level in young children	Health Canada (1996c)			
Inhalation	3.57 µg/kg bw-day	Blood lead level in young children	Health Canada (1996c)			
Cancer Effects						
Ingestion	NA					
Inhalation	NA					

1.1.2.4 Bioavailability of Lead

Oral Route of Exposure

Gastrointestinal absorption of lead varies with the age, diet, and nutritional status of the subject, as well as with the chemical species and the particle size of lead that is administered (ATSDR 1993). Age is a well-established determinant of lead absorption; adults typically absorb 7 to 15 percent of lead ingested from dietary sources, while estimates of lead absorption from dietary sources in infants and children range from 40 to 53 percent. Estimates derived from dietary balance studies conducted in infants and children (ages 2 weeks to 8 years) indicate absorption of approximately 40–50% of ingested lead (Alexander *et al.* 1974; Ziegler *et al.* 1978).

Soil lead absorption has been studied in rats, swine, and humans. The swine model has been used to test soils from numerous sites. A physiologically based extraction method is also well developed (Ruby *et al.* 1993, 1996; Medlin 1997) and is undergoing detailed validation studies. The studies in rats and swine have indicated that absorption of lead from soil will vary with the source of the lead, ranging from

near zero to greater than 50 percent absolute bioavailability (*i.e.*, relative bioavailability of 1.0, or more compared to soluble lead forms) (Casteel *et al.* 1997; Dieter *et al.* 1993; Freeman *et al.* 1992, 1996; Schoof *et al.* 1995; US EPA 1996b-e, 1998a-e). On average, the results of these studies support the use of a default assumption that 30 percent of an oral lead dose is absorbed from soil (*i.e.*, relative bioavailability of 0.6). A study in adult humans indicates that absolute lead bioavailability from a mining-area soil varies from approximately 3 to 26 percent, depending on how recently the test subject had eaten (Maddaloni *et al.* 1998).

The absorption of lead in soil is less than that of dissolved lead. In US EPA's childhood lead model, it is assumed that 50 percent of an oral lead dose is absorbed from food and water, while 30 percent of a soil lead dose is assumed to be absorbed. Thus, the default assumption for lead is that the relative bioavailability of soil lead compared to soluble lead forms is 0.6 (i.e., 30 percent divided by 50 percent) (US EPA 1994b). Adult subjects who ingested soil (particle size less than 250 µm) from the Bunker Hill NPL site absorbed 26% of the resulting 250 µg/70 kg body weight lead dose when the soil was ingested in the fasted state and 2.5% when the same soil lead dose was ingested with a meal (Maddaloni et al. 1998). Additional evidence for a lower absorption of soil-borne lead compared to dissolved lead is provided from studies in laboratory animal models. In immature swine that received oral doses of soil from one of four NPL sites (75 or 225 µg Pb/kg body weight), bioavailability of soil-borne lead ranged from 50% to 82% of that of a similar dose of highly water soluble lead acetate (Table 2-5) (Casteel et al. 1997; US EPA 1996a, 1996b, 1996c). If the relative bioavailability of soil-borne lead (soil/acetate) in immature swine is indicative of the relative bioavailability in human children, and if the absolute bioavailability of water soluble lead in humans children is 50%, as the Alexander et al. (1974) and Ziegler et al. (1978) studies would suggest, then the absolute bioavailability of soil-borne lead in human children predicted from the swine studies would range from 25 to 41%.

Dermal Route of Exposure

Limited information is available regarding absorption after dermal exposure in humans. Dermal absorption of inorganic lead compounds is reported to be much less significant than absorption by inhalation or oral routes of exposure, because of the greatly reduced dermal absorption rate (US EPA 1986). Following skin application of ²⁰³Pb-labeled lead acetate in cosmetic preparations (0.1 mL of a lotion containing 6 mmol lead acetate/L or 0.1 g of a cream containing 9 mmol lead acetate/kg) to 8 male volunteers for 12 hours, absorption was ~0.3%, but expected to be 0.06% during normal use of such preparations (Moore *et al.* 1980). Most of the absorption took place by 12 hours of exposure. The US EPA default value of for dermal absorption of lead compounds from soil is 1%.

Based on the above review, absolute oral bioavailability for lead through food ingestion is set at 50% to ensure a protective assessment for young children. In accordance with the US EPA recommendations and the above review, the relative bioavailability of lead from soil is 60%, yielding an absolute soil bioavailability of 30%. Dermal bioavailability is set at the US EPA default of 1% which is likely to overestimate dermal absorption.

1.1.3 **Zinc**

1.1.3.1 Assessment of Carcinogenicity

Epidemiological studies of workers exposed to zinc have not shown a relationship between zinc exposure and the development of cancer (ATSDR 1994). Animal studies have also not shown a link between inhalation, oral or dermal exposure to zinc and an increase in the incidence of cancers (ATSDR 1994). Based on inadequate evidence in humans and animals, US EPA classified zinc as Class D; not classifiable as to human carcinogenicity (US EPA 1992). Health Canada (1996b) and CCME (1987) do not consider zinc as a human carcinogen.

1.1.3.2 Susceptible Populations

There is no specific information regarding the existence of human subpopulations that are sensitive to the toxic effects of zinc; however there is limited indication that people who are malnourished or have marginal copper status may be more susceptible to excessive zinc exposures than the average population (ATSDR 1994).

1.1.3.3 Selection of Toxicity Values

CCME considers zinc to be an essential nutrient and has not developed an oral TDI to account for environmental exposures to zinc (CCME 1987).

The US EPA has developed an RfD $_{o}$ of 300 μ g/kg bw-day based on a clinical study of females taking zinc as a dietary supplement (US EPA 1992). The study reported a decrease in the level of erythrocyte superoxide dismutase in study participants. An uncertainty factor of 3 was applied to the NOEL from the study to account for sensitive members of the population (US EPA 1992).

Neither the US EPA nor Health Canada has developed reference concentration values for inhalation exposures to zinc. The oral reference dose developed by the US EPA has been selected for assessing the potential hazards associated with oral exposures to zinc. The toxicity values used to assess the potential risks associated with ingestion and inhalation exposure to zinc are summarized in Table B-3.

Table B-3 Selected Toxicity Values for Zinc

Route of Exposure	TRV	Toxicological Basis	Source Agency
	No	on-Cancer Effects	
Ingestion	300 μg/kg bw-day	Decreased erythrocyte SOD	US EPA (1992)
Inhalation	NA	NA	NA
		Cancer Effects	
Ingestion	NA		
Inhalation	NA		

1.1.3.4 Bioavailability of Zinc

Oral Route of Exposure

Several studies have measured oral absorption rates of zinc in humans. Absorption ranged from 8% to 81% following short-term exposures to zinc supplements in the diet; differences in absorption are probably due to the type of diet (amount of zinc ingested, amount and kind of food eaten) (Aamodt *et al.* 1983; Hunt *et al.* 1991; Istfan *et al.* 1983; Reinhold 1991; Sandstrom and Abrahamson 1989; Sandstrom and Cederblad 1980; Sandstrom and Sandberg 1992). The body's natural homeostatic mechanisms control zinc absorption from the gastrointestinal tract (Davies 1980). Persons with adequate nutritional levels of zinc absorb approximately 20-30% of all ingested zinc. Those who are zinc-deficient absorb greater proportions of administered zinc (Johnson *et al.* 1988; Spencer *et al.* 1985).

Dermal Route of Exposure

Dermal absorption of zinc occurs, but its mechanism is not clearly defined. Studies are very limited regarding the absorption of zinc through the skin. Historically, zinc oxide has been used clinically to promote the healing of burns and wounds (Gordon *et al.* 1981). Absorption has been observed in burn patients treated with gauze dressings containing zinc oxide (Hallmans 1977).

Selected Values

Based on the above review, oral bioavailability is set at 80% to ensure a protective assessment. Dermal bioavailability is set at the US EPA default of 1% which is likely to overestimate dermal absorption.

1.1.4 References for Human Health Toxicity Profiles

Aamodt R.L., W.F. Rumble and R.I. Henkin. 1983. Zinc absorption in humans: Effects of age, sex, and food. In: Inglett G, ed. The Nutritional Bioavailability of Zinc. The American Chemical Society, pp. 61-82. Washington, DC.

- ATSDR (Agency for Toxic Substances and Disease Registry). 1993. Toxicological Profile for Lead. U.S. Department of Health and Human Services, Public Health Service, ATSDR, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry). 1994. Toxicological Profile for Zinc. U.S. Department of Health and Human Services, Public Health Service, ATSDR, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Toxicological Profile for Cadmium. U.S. Department of Health and Human Services, Public Health Service. ATSDR, Atlanta, GA.
- Alexander F.W., B.E. Clayton and H.T. Delves. 1974. Mineral and trace-metal balances in children receiving normal and synthetic diets. Q.J. Med. 43: 89-111.
- Casteel, S.W., R.P. Cowart, C.P. Weis, G.M. Henningsen, E. Hoffman, W.J. Brattin, R.E. Guzman, M.F. Starost, J.T. Payne, S.L. Stockham, S.V. Becker, J.W. Drexler, and J.R. Turk. 1997. Bioavailability of Lead to Juvenile Swine Dosed with Soil from the Smuggler Mountain NPL Site of Aspen, Colorado. Fund. Appl. Toxicol., 36: 177-187.
- CCME (Canadian Council of Ministers of the Environment). 1987. Guidelines for Canadian Drinking Water Quality. Supporting documentation for Zinc.
- CCME (Canadian Council of Ministers of the Environment). 1992. Guidelines for Canadian Drinking Water Quality. Supporting Document for Lead.
- Davies NT. 1980. Studies on the absorption of zinc by rat intestine. Br. J. Nutr. 43: 189-203.
- Dieter, M.P., H.B. Matthews, R.A. Jeffcoat, and R.F. Moseman. 1993. Comparison of Lead Bioavailability in F344 Rats Fed Lead Acetate, Lead Oxide, Lead Sulfide, or Lead Ore. Journal of Toxicology and Environmental Health, 39, 79-93, 1993.
- Freeman, G.B., J.D. Johnson, J.M. Killinger, S.C. Liao, P.I. Feder, A.O. Davis, M.V. Ruby, R.L. Chaney, S.C. Lovre, and P.D. Bergstrom. 1992. Relative Bioavailability of Lead from Mining Waste Soil in Rats. Fund. Appl. Toxicol. 19: 388-398.
- Freeman, G.B., J.A. Dill, J.D. Johnson, P.J. Kurtz, F. Parham, and H.B. Matthews. 1996. Comparative Absorption of Lead from Contaminated Soil and Lead Salts by Weanling Fischer 344 Rats. Fund.. Appl. Toxicol. 33: 109-119.
- Gordon E.F., R.C. Gordon and D.B. Passal. 1981. Zinc metabolism: Basic, clinical, and behavioral aspects. J. Pediatr. 99: 341-349.

- Griffin, S.R., R. Rubenstein, S. Irene, C. DeRosa, and H. Choudhury. 1990. Bioavailability in Rats of Metals Adsorbed to Soils. U.S. Environmental Protection Agency, Washington, DC, Hazleton Laboratories, America, Inc. Poster presented at the Society of Toxicology 29th Annual Meeting, Miami Beach, FL. February 12-16, 1990. Poster paper no. 623.
- Hallmans, G. 1977. Treatment of burns with zinc tape: A study of local absorption of zinc in humans. Stand. J. Plast. Reconstr. Surg. 11: 155-161.
- Health Canada. 1996a. Canadian Soil Quality Guidelines for Contaminated Sites. Human Health Effects: Inorganic Cadmium. The National Contaminated Sites Remediation Program.
- Health Canada. 1996b. Health-based tolerable daily intakes/ concentrations and tumorigenic doses/concentrations for priority substances. ISBN 0-662-24858-9.
- Health Canada. 1996c. Canadian Soil Quality Guidelines for Contaminated Sites. Human Health Effects: Inorganic Lead. The National Contaminated Sites Remediation Program.
- Hunt J.R., G.I. Lykken and L.K. Mullen. 1991. Moderate and high amounts of protein from casein enhance human absorption of zinc from whole wheat or white rolls. Nutrition Research 11: 413-418.
- Istfan N.W., M. Jang horbani V.R. Young. 1983. Absorption of stable ⁷⁰Zn in healthy young men in relation to zinc intake. Am. J. Clin. Nutr. 38: 187-194.
- Johnson P.E., J.R. Hunt and N.V. Ralston. 1988. The effect of past and current dietary Zn intake on Zn absorption and endogenous excretion in the rat. J. Nutr. 118: 1205-1209.
- Maddaloni, M., P. Goodrum, G. Diamond, W. Manton, C. Blum, N. LoLacono, and J. Graziano. 1998. Metal-Weighted Bioavailability of Soil-Borne Lead in Adults: Use of Empirical Data Analyzed by Stable Isotope Dilution, and Probabilistic Worker Exposure Scenarios. Poster presented at the Society of Toxicology, 37th Annual Meeting in Seattle, WA.
- McLellan JS, P.R. Flanagan and M.J. Chamberlain. 1978. Measurement of dietary cadmium absorption in humans. J. Toxicol. Environ. Health 4: 131-138.
- Medlin, E.A. 1997. An In Vitro Method for Estimating the Relative Bioavailability of Lead in Humans. M.S. Thesis. Department of Geological Sciences, University of Colorado at Boulder, CO.
- MOEE (Ministry of Environment and Energy). 1994. Scientific Criteria Document for Multimedia Environmental Standards Development Lead. Ontario MOEE.

- Moore M.R., P.A. Meredith and W.S. Watson. 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.
- Newton D., P. Johnson and A.E. Lally. 1984. The uptake by man of cadmium ingested in crab meat. Hum. Toxicol. 3: 23-28.
- PTI (PTI Environmental Services). 1994. Remedial Investigation Report: National Zinc Site Remedial Investigation and Feasibility Study, Volume 1. Prepared for City of Bartlesville, Cyprus Amax Minerals Company, and Salomon, Inc.
- Rahola T., R.K. Aaran and J.K. Miettenen. 1973. Retention and elimination of 115mCd in man. In: Health physics problems of internal contamination. Budapest: Akademia 213-218.
- Reinhold J.G., B. Faradji and P. Abadi. 1991. Decreased absorption of calcium, magnesium, and phosphorous by humans due to increased fiber and phosphorous consumption as wheat bread. Nutr. Rev. 49: 204-206.
- Ruby, M.V., A. Davis, T.E. Link, R. Schoof, R.L. Chaney, G.B. Freeman, and P. Bergstrom. 1993. Development of an In Vitro Screening Test to Evaluate the In Vivo Bioaccessibility of Ingested Mine-Waste Lead. Environ. Sci. Technol., 27: 2870-2877.
- Ruby, M.V., A. Davis, R. Schoof, S. Eberle, and C.M. Sellstone. 1996. Estimation of Lead and Arsenic Bioavailability Using a Physiologically Based Extraction Test. Environ. Sci. Technol., 30: 422-430.
- Ryu, J.E., E.E. Ziegler, S.E. Nelson, and S.J. Fomon. 1983. Dietary intake of lead and blood lead concentration in early infancy. Am. J. Dis. Child. 137: 886-891.
- Sandstrom B. and A. Cederblad. 1980. Zinc absorption from composite meals: II. Influence of the main protein source. Am. J. Clin. Nutr. 33: 1778-1783.
- Sandstrom B. and H. Abrahamson. 1989. Zinc absorption and achlorhydria. Eur. J. Clin. Nutr. 43: 877-879.
- Sandstrom B. and A.S. Sandberg. 1992. Inhibitory effects of isolated inositol phosphates on zinc absorption in humans. Journal of Trace Elements and Electrolytes in Health and Disease 6: 99-103.
- Schilderman, P.A.E.L., E.J.C. Moonen, P. Kembers, and J.C.S. Kleinjans. 1997. Bioavailability of Soil-Adsorbed Cadmium in Orally Exposed Male Rats. Environ. Health Perspect. 105: 234-238.

- Schoof, R.A. and G.B. Freeman. 1995. Oral Bioavailability of Lead and Cadmium in Soil from a Smelter Site. Poster presented at the Seventh International Congress of Toxicology, Seattle, WA. July 3-6, 1995.
- Schoof, R.A., M.K. Butcher, C. Sellstone, R.W. Ball, J.R. Fricke, V. Keller, and B. Keehn. 1995. An Assessment of Lead Absorption from Soil Affected by Smelter Emissions. Environ. Geochem. Health 17(4): 189-199.
- Spencer H, L. Kramer and D. Osis. 1985. Zinc metabolism in man. J. Environ. Pathol. Toxicol. Oncol. 5: 265-278.
- Thun, M.J., T.M. Schnorr, A.B. Smith and W.E. Halperin. 1985. Mortality among a cohort of U.S. cadmium production workers: An update. J. Natl. Cancer Inst. 742: 325-333.
- US EPA (United States Environmental Protection Agency). 1985. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium. Addendum to the Health Assessment Document for Cadmium (EPA 600/B- B1-023). EPA 600/B-83-025F.
- US EPA (United Sates Environmental Protection Agency). 1986. Air quality criteria for lead. Research Triangle Park, NC: US EPA, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. EPA 600/8-83-028F.
- US EPA (United States Environmental Protection Agency). 1992. Integrated Risk Information System (IRIS). Substance Files for Zinc and compounds. URL: http://cfpub.epa.gov/iris/
- US EPA (United States Environmental Protection Agency). 1993. Integrated Risk Information System (IRIS). Substance Files for Lead and compounds. URL: http://cfpub.epa.gov/iris/
- US EPA (United States Environmental Protection Agency). 1994a. Integrated Risk Information System (IRIS). Substance Files for Cadmium and compounds. URL: http://cfpub.epa.gov/iris/
- US EPA (United States Environmental Protection Agency). 1994b. Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. EPA/540/R-93/081. Office of Emergency and Remedial Response, Washington, DC.
- US EPA (United States Environmental Protection Agency). 1996a. Soil Screening Guidance: Technical Background Document. EPA/540/R-95/128. Office of Solid Waste and Emergency Response, Washington, DC.

- US EPA (United States Environmental Protection Agency). 1996b. Bioavailability of Lead in Slag and Soil Samples from the Murray Smelter Superfund Site. Document Control No. 04800-030-0163. US EPA Region VIII, Denver, CO.
- US EPA (United States Environmental Protection Agency). 1996c. Bioavailability of Lead in Soil Samples from the New Jersey Zinc NPL Site, Palmerton, Pennsylvania. Document Control No. 04800-030-0159. US EPA Region VIII, Denver, CO.
- US EPA (United States Environmental Protection Agency). 1996d. Bioavailability of Lead in Soil Samples from the Jasper County, Missouri, Superfund Site. Document Control No. 04800-030-0161. US EPA Region VIII, Denver, CO.
- US EPA (United States Environmental Protection Agency). 1996e. Bioavailability of Lead in Soil Samples from the Smuggler Mountain NPL Site, Aspen, Colorado. US EPA Region VIII, Denver, CO.
- US EPA (United States Environmental Protection Agency). 1996f. Integrated Risk Information System (IRIS). Substance Files for Zinc and compounds.
- US EPA (United States Environmental Protection Agency). 1998a. Bioavailability of Lead in Soil and Mine Waste from the California Gulch NPL Site, Leadville, Colorado. Document Control No. 04800-030-0178. US EPA Region VIII, Denver, CO.
- US EPA (United States Environmental Protection Agency). 1998b. Bioavailability of Lead in a Slag Sample from the Midvale Slag NPL Site, Midvale, Utah. Document Control No. 04800-030-0166.
- US EPA (United States Environmental Protection Agency). 1998d. Bioavailability of Lead in Unweathered Galena- Enriched Soil. Document Control No. 04800-030-0171. US EPA Region VIII, Denver, CO.
- US EPA (United States Environmental Protection Agency). 1998e. Bioavailability of Lead in Paint. Document Control No.04800-030-0170. US EPA Region VIII, Denver, CO.
- Wester, R.C., H.I. Maibach, L. Sedik, J. Melendres, S. Dizio, and M. Wade. 1992. "*In Vitro* Percutaneous Absorption of Cadmium from Water and Soil into Human Skin." Fund. Appl. Toxicol., 19: 1-5.
- WHO (World Health Organization). 1986. Principles for evaluating health risks from chemicals during infancy and early childhood; the need for a special approach. Environmental Health Criteria 59. WHO, Geneva, Switzerland.

- WHO (World Health Organization). 1993. Evaluation of certain food additives and contaminants: forty-first report of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). WHO Technical Report Series No. 837. WHO, Geneva, Switzerland.
- WHO (World Health Organization). 1998. Cadmium. guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2 Health criteria and other supporting information. WHO, Geneva, Switzerland. pp. 195-201.
- WHO (World Health Organization). 2000. Guidelines for air quality. WHO, Geneva, Switzerland.
- Ziegler, E.E., B.B. Edwards, R.L. Jensen, K.R. Mahaffey and S.J. Fomon. 1978. Absorption and retention of lead by infants during infancy and early childhood: The need for a special approach. Ped. Res. 12: 29-34.

1.2 Ecological Health Toxicity Profiles

1.2.1 Cadmium

Cadmium (Cd) is a eavy metal that does not appear to be biologically essential to wildlife. Background concentrations of cadmium in Canadian soils range from not detectable to as high as 8.1 mg/kg (CCME 1999). In Ontario, for surface soils from rural parklands remote from pollution point-sources, the 98th percentile for cadmium was 0.71 mg/kg (MOEE 1993).

Cadmium is a known teratogen, carcinogen, and a probable mutagen in some terrestrial and aquatic biota (Hoffman *et al.* 1995). Exposure to low concentrations of cadmium may result in adverse effects in a wide range of ecological receptors. Although biomagnification of cadmium in the food chain has not been demonstrated, cadmium does bioaccumulate in invertebrate and vertebrate species, microorganisms, and plants.

The bioavailability of cadmium is dependent on its chemical form as well as other environmental conditions such as pH, clay content, particle size, hardness, and organic carbon. The free ionic form of cadmium is more toxic to aquatic biota than cadmium that is complexed with dissolved organic matter or with soluble particulate matter or colloidal matter. Soils and sediments tend to attract cadmium, due to the low solubility and volatility of cadmium. Based on its persistence in the environment and its toxicity to wildlife, cadmium contamination may present a significant threat to ecosystems at all levels of biological organization.

Cadmium can be present in soil as free cadmium compounds or in solution as the Cd²⁺ ion dissolved in interstitial water (ATSDR 1993). The speciation of cadmium in soils is dependent on the soil geochemistry. For soil types ranging from sand to silty clay loam, the adsorption of Cd²⁺ is highly

dependent on pH. As pH increases, so too does cadmium sorption. Under acidic conditions, cadmium is more likely to move within the soil matrix and into other media. Cadmium tends to sorb more strongly to organic matter than to metal oxides, such as iron, aluminum and manganese oxides. Movement of cadmium within soils is also affected by wind transport, leaching and uptake by terrestrial organisms. In acidic soils, the release of Cd²⁺ and its uptake by plants is favored (ATSDR 1993).

In the aqueous environment, cadmium is relatively mobile, and can exist as the hydrated ion, as inorganic complexes, and as organic complexes. Cadmium tends to complex with dissolved organic carbon and also in association with colloidal and particulate matter.

Terrestrial plants and animals have been shown to be adversely affected by exposure to cadmium. In terrestrial plants, uptake of cadmium has been demonstrated, although uptake rates and translocation of cadmium are variable (CCME 1999). The lowest soil cadmium concentrations at which toxicity to plants have been observed are 2.5 and 4 mg/kg (CCME 1999).

Adverse effects of cadmium ingestion have been observed in various mammals and bird species, and include reduction of food intake and growth rate, impaired reproduction and mortality (Environment Canada 1996). Animals have a limited ability to eliminate cadmium from their bodies, and cadmium has been shown to accumulate primarily in the liver and renal cortex.

Many studies have demonstrated uptake of cadmium by earthworms in contaminated soils, and adverse effects to chronic earthworm endpoints were indicated by cadmium concentrations ranging from 25 mg/kg to 108 mg/kg soil (summarized in Sample *et al.* 1997). The bioconcentration factor from soil to earthworms has been shown to be negatively correlated with soil pH, presumably because the lowering of pH leads to increased desorption of metal cations, such as cadmium, thereby increasing bioavailability.

In laboratory rats, reduced growth has been observed from a dietary exposure of 7.15 mg/kg (Weigel at al. 1984 in Sample *et al.* 1997). Reproductive effects on rats have been associated with drinking water exposures as low as 1 mg/kg CdCl₂, and in mice from drinking water exposures of 10 mg/L Cd (as CdCl₂)(summarized in Sample *et al.* 1997). For dietary exposures, reproductive effects on rats have been observed from exposures as low as 0.125 mg/kg CdCl₂ over four generations (Wills *et al.* 1981 in Sample *et al.* 1997). For exposure via oral gavage, the lowest observed adverse effects level for reproduction in rats was reported to be 10 mg/kg body weight per day (Sutoe *et al.* 1980 in Sample *et al.* 1996). Reproductive effects in avian species have also been reported. Mallard duck hens orally exposed to cadmium at 19 mg/kg-day for 90 days exhibited suppressed egg production (White and Finley 1978).

The soil quality guideline for cadmium for the protection of ecological health has been determined to be 10 mg/kg (based on soil contact) by CCME (1999).

1.2.2 Copper

Copper (Cu) is a metal that occurs naturally in rock, soil, water, sediment, air, and biota. Copper can occur in four oxidation states (Cu, Cu⁺, Cu²⁺ and Cu³⁺), although Cu²⁺ is the most common state, particularly in oxidizing conditions. Copper is an essential nutrient for all living organisms; however, long-term exposure to elevated or deficient levels of copper may result in adverse effects in plants and animals (CCME 1999).

Copper is used widely in the manufacturing industry and occurs in a wide range of mineral deposits as both a primary and secondary mineral. Most copper occurs in the form of sulphide minerals. The average copper concentration in Canadian soil is estimated to be 20 mg/kg, with a range from 2 to 100 mg/kg (BCMELP 1992). In surface soils from rural parkland remote from pollution point-sources, the 98th percentile was 41 mg/kg for rural parkland (MOEE 1993).

In soils, most copper is in mineral form or strongly bound to particles, and therefore has low bioavailability. However, copper may be solubilized and thereby made more available for uptake by plants and animals. Factors that influence the availability of copper in soils are pH, cation exchange capacity (CEC), organic matter content, reduction-oxidation potential and the presence of metal oxides (e.g., as iron, manganese and aluminum oxides). Copper is more mobile in acidic soils. In general, copper will be immobilized in soils with high CEC and organic matter content. The CEC of soils is influenced by pH and clay and organic matter content. Copper is specifically adsorbed by iron, manganese and aluminum oxides, and binds more strongly that most other metals. However, under reducing conditions, copper bound to these metal oxides will be solubilized.

Copper has been demonstrated to be potentially toxic to plants and animals. Toxicity to terrestrial plants has been shown to occur at concentrations ranging from 100 mg/kg to 200 mg/kg copper in soil (summarized in Sample *et al.* 1997). Copper bioaccumulates in plants to a small degree.

Animals have the ability to regulate internal concentrations of copper. Nevertheless, adverse reactions by wildlife have been shown to occur in association with high copper and deficient copper concentrations.

Studies have shown that earthworms are adversely affected by copper concentrations in soil as low as 68 mg/kg, and also show that the organic content of the soil is a strong determinant of copper availability and toxicity to litter invertebrates (summarized in Sample *et al.* 1997). In mink, the lowest observed adverse effect level was estimated to be 15 mg/kg-day, based on an increased mortality rate in offspring of mink fed a diet supplemented with copper (Sample *et al.* 1996). In chicks, dietary copper levels from 588 to 1176 mg/kg for 10 days resulted in growth effects, with 500 mg/kg estimated to be the minimum toxic level, corresponding to a lowest observed adverse effect level of 61.7 mg/kg-day (Mehring *et al.* 1960 in Sample *et al.* 1996).

The soil quality guideline for copper for the protection of ecological health has been determined to be 63 mg/kg (based on soil contact) by CCME (1999).

1.2.3 Lead

Lead is a non-essential and highly toxic heavy metal. Natural environments rarely contain lead in its elemental form. However, lead is present in low concentrations throughout the environment as a result anthropogenic inputs (e.g., from animal wastes, coal residues, incineration of municipal wastes, waste waters and automobile emissions) and natural sources (e.g., volcanoes, forest fires, sea salt, geologic weathering). In Canada, mean background levels of lead in soils remote from ore bodies have been estimated to range from 12 to 25 mg/kg (CCME 1999). In surface soils from rural parklands remote from pollution point-sources, the 98th percentile was 45 mg/kg (MOEE 1993). Near lead emissions sources such as smelters, lead concentrations in excess of 700 mg/kg in soil have been found (CCME 1999).

In soils, lead can pose a threat if it is mobile and moves to surface or groundwaters, or into biota. Lead form and mobility in soils is influenced by pH, soil texture and organic matter. The dissolved form of lead in soils is Pb²⁺, and therefore the cation exchange capacity (CEC) of the soil can decrease its mobility in the short term (CCME 1999). Soil erosion by wind or water is a mechanism by which lead can be mobilized from soil to contaminate the surrounding environment.

The most toxic forms of lead are considered to be the organolead compounds, the most important of which are tetramethyl and tetraethyl lead. Most of the lead that is ingested by biota is rapidly egested (Eisler 1988). By contrast, inhaled lead is quickly absorbed into the bloodstream (ATSDR 1993). Lead has been shown to bioconcentrate and, as a result, older organisms tend to have the highest body burdens. However, lead does not appear to biomagnify in the food chain (Eisler 1988).

In plants, significant adverse effects are generally only seen at relatively high lead levels. For example, root and shoot growth was reduced in red spruce when exposed to 150 mg/kg lead in soil (Seiler and Paganelli 1987). Uptake and accumulation rates in plants are related to soil pH and vary among and within species. The decline of some European spruce forests has been attributed to excessive concentrations of atmospheric lead. Reported effects include inhibition of plant growth, and reductions in germination, seed viability, and rates of photosynthesis and transpiration (Hoffman *et al.* 1995).

In animals and birds, lead acts to inhibit enzymes necessary for normal biological function. In mammals, lead toxicity may affect the hematological system, the brain and nervous system, learning and behavior, and reproduction (Hoffman *et al.* 1995).

In rats, oral doses of 80 mg/kg-day have been estimated to be the lowest observed level associated with reproductive impairment (Azar et al. 1973 in Sample et al. 1996). In birds, reproductive and

developmental effects include decreases in egg hatching success at an estimated lowest observed adverse effect concentration of 11.3 mg/kg-day via oral exposures in Japanese quail (Edens *et al.* 1976 in Sample *et al.* 1996). Earthworms accumulate lead, but total lead concentrations in earthworms are almost always well below that in soil (CCME 1999). Earthworms, like all soil invertebrates, cycle soil through their bodies, but assimilate low net amounts of lead compared with other trace elements, such as cadmium (CCME 1999).

The soil quality guideline for lead for the protection of ecological health has been determined to be 300 mg/kg (based on soil contact) by CCME (1999).

1.2.4 Silver

Silver is a naturally occurring element in the earth's crust at an average concentration of approximately 0.1 mg/kg, and is generally present at low concentrations (Purcell and Peters 1998). Although there are natural deposits, silver is primarily found on land where it has been deposited because of human activity, in the atmosphere as a result of smelting and coal burning activities, and in aquatic systems as a result of discharges from mining, industry or sewage treatment plants. In Canada, the average silver content in soils remote from mineralized zones has been reported to be 0.30 mg/kg (Boyle 1968 in ATSDR 1990).

Silver occurs in four oxidation states: Ag, Ag^+ , Ag^{2+} and Ag^{3+} , with Ag and Ag^+ being the most common. Most silver minerals are compounds of silver with sulfur or the homologues and neighbours on the periodic chart. In soils, silver is primarily found in sulfide form in association with iron, lead or tellurides. In surface waters, silver can be found in a number of forms: monovalent ion, sulfide, bicarbonate, sulfate salts, or adsorbed onto organic or inorganic materials. Many of these forms are insoluble and as a result, their bioavailability is not great. The potential for effects in the environment is dependent on the chemical form of silver.

The mobility of silver in the environment depends on its physical and chemical form, as well as physicochemical conditions in surrounding media (e.g., soil, sediment, and water). The majority (>94%) of silver released into the environment is expected to remain in the soil or wastewater sludge at the emission site (Ratte 1999). The remainder will be transported via air and water. In water, silver is found as a free monovalent ion, as part of a chloride or sulfide compound, or adsorbed onto particulate matter. The bioavailability of silver in soils is largely dependent on environmental factors such as drainage, oxidation-reduction potential, pH, and organic matter content. Silver partitions preferentially into the particulate phase and forms a variety of organic-inorganic solids. The bioaccumulation of silver in soil appears to be low (Ratte 1999).

Toxicity of silver occurs mainly in the aqueous phase and depends on the concentration of free Ag⁺ ions. Studies on silver toxicity are almost all based on the free ion, but this does not represent the form most likely to be encountered in the environment. Most recent studies of silver compounds conclude that they

are much less toxic than silver ions (Purcell and Peters 1998). In soil, the toxicity of silver, even at high total concentrations, is very low (Ratte 1999).

In higher plants and fungi, silver is only expected to accumulate in contaminated areas, such as mine tailings and soils amended with sewage sludge that contains silver. The sensitivity of terrestrial plant species to silver exposure varies, and includes effects on growth and germination to sensitive species at a concentration of 7.5 mg/L silver nitrate (Ratte 1999).

Silver toxicity in terrestrial animals has been investigated mainly in laboratory bioassays. Oral exposure to high doses of silver in drinking water killed rats and dermal exposures resulted in reduced weight gain in guinea pigs. The lowest observed adverse effects level for rats was 362 mg/kg-day (death; two week exposure period)(Walker 1971 in ATSDR 1990) and for guinea pigs was 137.13 mg/kg-day (reduced weight gain; eight week exposure period)(Wahlberg 1965 in ATSDR 1990). There is no substantial potential for bioaccumulation in mammals (Ratte 1999). Birds have been shown to bioaccumulate silver in their livers (Eisler 1996 in Ratte 1999). Silver toxicity to terrestrial invertebrates has been investigated mainly using earthworms. Neither carcinogenic nor mutagenic effects are suggested from silver exposure. No studies demonstrating adverse chronic effects to reproductive and developmental endpoints in mammals have been identified (ATSDR 1990; Ratte 1999).

Although no Canadian soil quality guideline has been developed to date for silver for the protection of ecological health, the Ontario Ministry of Environment and Energy has developed a guideline of 20 mg/kg in soil (MOEE 1993).

1.2.5 Zinc

Zinc is a naturally-occurring common metallic element. In Canada, the mean level of zinc in soils is reported to be 74 mg/kg, with average concentrations in various regions ranging from 54 to 81 mg/kg (CCME 1999). In surface soils from rural parklands remote from pollution point-sources, the 98th percentile was 120 mg/kg (MOEE 1993). The continental crustal zinc content is reported to be 52 mg/kg in the upper layer (Wedepohl 1995). Zinc is primarily used for galvanizing and in the manufacture of brass and bronze and for heating and cooling system components (CCME 1999).

In the environment, zinc occurs primarily in the Zn²⁺ oxidation state. Zinc tends to strongly react with organic and inorganic compounds and forms stable combinations with many organic substances and a wide range of biochemical compounds. Zinc is an essential element and is naturally used in many biochemical compounds. In soils, zinc is highly reactive and forms both soluble and insoluble organic complexes. Zinc can also be adsorbed to clay or metallic oxides and may also be part of the parent soil material. Soil pH has been shown to be a main factor in controlling zinc mobility and sorption in soils; as pH decreases, zinc becomes more soluble and bioavailable to organisms. However, there are other factors that contribute to zinc mobility in soils, such as plant uptake, leaching, moisture content,

reduction-oxidation potential, and mineralization of organic matter (CCME 1999). In anaerobic soils, zinc sulfide controls the mobility of zinc (ATSDR 1994). Zinc sulfide is insoluble, and as a result, zinc mobility and bioavailability in anaerobic soils is low.

In unpolluted waters, zinc exists mostly in the hydrated divalent cationic form, and in polluted waters zinc often forms organic and inorganic complexes (ATSDR 1994). The hydrated divalent cationic form of zinc is much more toxic to aquatic biota than zinc that is complexed with dissolved organic matter or with particulate or colloidal matter. Bioconcentration of zinc in aquatic organisms is relatively high, though it is much lower in terrestrial organisms, and biomagnification does not occur in either terrestrial or aquatic food chains (ATSDR 1994).

Because zinc is an essential element, organisms can regulate internal concentrations and therefore tend to be relatively tolerant of high exposure concentrations. Adverse effects of zinc on terrestrial plants have been observed at 50 mg zinc/kg dry soil (reduction in seed yield in turnips; in CCME 1999).

Mammals have been shown to be tolerant tolerant to intake rates of zinc up to levels 100 times greater than the minimum recommended daily requirement (Eisler 1993). The primary toxic effects of zinc are on zinc-dependent enzymes that regulate RNA and DNA. Eisler (1993) reported a number of adverse reproductive, developmental and survival effects on animals resulting from chronic dietary exposures to zinc ranging from 500 to over 6000 mg/kg for rats and mice and at 3000 mg/kg for Mallard duck. Schlicker and Cox (1986, in Sample *et al.* 1996) demonstrated reproductive effects in rats at a lowest observed adverse effect level of 320 mg/kg-day zinc in diet. In birds, Stahl *et al.* (1990, in Sample *et al.* 1996) demonstrated reproductive effects to white leghorn hens from a dietary dose of 131 mg/kg-day (lowest observed adverse effects level). The lowest effect concentration reported for domestic mammals was a dietary exposure of 750 mg zinc/kg for Cheviot sheep, resulting in a 64% reduction in the number of viable offspring (in CCME 1999). For earthworms, an LC50 of 80 mg zinc/kg dry soil has been reported, however, the lethal concentration has been shown to be highly dependent upon soil pH (CCME 1999).

The soil quality guideline for zinc for the protection of ecological health has been determined to be 200 mg/kg (based on soil contact) by CCME (1999).

1.2.6 References for Ecological Health Toxicity Profiles

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. Toxicological Profile for Silver. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

- ATSDR (Agency for Toxic Substances and Disease Registry). 1993. Toxicological Profile for Cadmium (Update). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry). 1994. Toxicological Profile for Zinc (Update). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.
- BCMELP (British Columbia Ministry of Environment, Lands and Parks). 1992. Toxicology of copper and chromium for contaminated sites. Ref. No. 107-10/grf92-1. Environmental Protection Division, Victoria, BC.
- CCME (Canadian Council of Ministers of the Environment). 1999. Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health. Canadian Environmental Quality Guidelines, Canadian Council of Ministers of the Environment, Winnipeg, MN.
- Efroymson, R.A., B.E. Sample and G.W. Suter. 2001. Uptake of inorganic chemicals from soil by plant leaves: Regressions of field data. Environ. Toxicol. Chem. 20: 2561-2571.
- Eisler, R. 1988. Lead Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Department of the Interior, Fish and Wildlife Service Biological Report 85 (1.14).
- Eisler, R. 1993. Zinc Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service, Biological Report 10, Contaminant Hazards Reviews Report 25.
- Environment Canada. 1996. Canadian soil quality guidelines for cadmium: Environmental. Supporting document Final draft. December 1996. Science Policy and Environmental Quality Branch, Guidelines Division, Ottawa, ON.
- Hoffman, D.J., B.A. Rattner, G.A. Burton Jr., and J. Cairns, Jr. 1995. Handbook of Ecotoxicology. Lewis Publishers, Boca Raton, FL. Pp. 356-391.
- MOEE (Ministry of Environment and Energy). 1993. Ontario Typical Range of Chemical Parameters in Soil, Vegetation, Moss Bags and Snow. Toronto, ON.
- Purcell, T.W. and J.J. Peters. 1998. Sources of silver in the environment. Environ. Toxicol. Chem. 17: 539-546.
- Ratte, H.T. 1999. Bioaccumulation and toxicity of silver compounds: A review. Environ. Toxicol. Chem. 18: 89-108.

- Richardson, M.E., M.R. Pivey Fox and B.E. Fry. 1974. Rathological changes produced in Japanese quail by ingestion of cadmium. J. Nutr. 104: 323-338.
- Sample, B.E., D.M. Opresko and G.W. Suter. 1996. Toxicological benchmarks for wildlife: 1996 revision. ES/ER/TM-86/R3. Oak Ridge National Laboratory, TN.
- Sample, B.E., G.W. Suter II, M.B. Sheaffer, D.S. Jones and R.A. Efroymson. 1997. Ecotoxicological profiles for selected metals and other inorganic chemicals. ES/ER/TM-210. Oak Ridge National Laboratory, TN.
- Schroeder, H.A. and M. Mitchener. 1971. Toxic effects of trace elements on reproduction of mice and rats. Archi. Environmental Health 23: 102-106.
- Seiler, J.R. and D. Paganelli. 1987. Photosynthesis and growth response of red spruce and lobolly pine to soil-applied lead and simulated acid rain. For. Sci. 33: 668-675.
- Shore, R.F. and P.E.T. Douben. 1994. The ectoxicological significance of cadmium intake and residues in terrestrial small mammals. Ecotoxicology and Environmental Safety 29: 101-112.
- Sorell, T.L. and J.H. Graziano. 1990. Effect of oral cadmium during pregnancy on maternal and fetal zinc metabolism in the rat. Toxicology and Applied Pharmacology 102: 537-545.
- Warila, J., S. Batterman and D.R. Passino-Reader. 2001. A probabilistic model for silver bioaccumulation in aquatic systems and assessment of human health risks. Environ. Toxicol. Chem. 20: 432-441.
- Wedepohl, K.H. 1995. The composition of the continental crust. Ingerson Lecture, Geochimicha et Cosmochimica Acta 59: 1217-1232.
- White, D.H. and M.T. Finley. 1978. Uptake and retention of dietary cadmium in Mallard Ducks. Environmental Research 17: 53-59.
- Will, M. and G.W. Suter II. 1995. Toxicological benchmarks for screening potential contaminants of concern for effects on terrestrial plants: 1995 revision. ES/ER/TM-85/R2. Oak Ridge National Laboratory, TN.

APPENDIX C RISK ASSESSMENT TECHNICAL INFORMATION



Calculation of Intakes from Marine Mammal Consumption

Species Consumed

Based on the community survey conducted in Arctic Bay by JWEL in September 2002, the predominant species hunted and consumed by local people were seal and narwhal. Food items included seal meat, seal liver, narwhal meat, and narwhal muktuk. Chan et al (1995) conducted a dietary exposure assessment in the community of Qikiqtarjuaq on eastern Baffin Island and found similar results. Approximately 90% of marine mammal intake in that community were from these four food items.

Metals Concentrations

Concentrations of cadmium and lead in marine mammals from the Canadian Arctic were researched in the following sources:

AMAP, 1998. AMAP Assessment Report: Arctic Pollution Issues. Arctic Monitoring and Assessment Program (AMAP), Oslo, Norway.

Canadian Arctic Contaminants Assessment Report I. 1997. Indian and Northern Affairs Canada. Ottawa.

Canadian Arctic Contaminants Assessment Report II. 2003. Indian and Northern Affairs Canada. Ottawa.

Chan, H. M., Kim, C., Khoday, K., Receveur, O., and Kuhnlein, H. V. 1995. Assessment of Dietary Exposure to Trace Metals in Baffin Inuit Food. Environ. Health Perspectives Vol. 103, No. 7-8.

Wagemann, R., Innes, S., and Richard, P.R. 1996. Overview and Regional and Temporal Differences of Heavy Metals in Arctic Whales and Ringed Seals in the Canadian Arctic. Sci. Tot. Environ. Vol. 186, pp. 41-66.

No information was available on zinc concentrations in marine mammals. Table 1 summarizes the data available from the above noted publications.

Table 1 Cadmium and Lead Concentrations in Marine Mammals (μg/g wet weight)

	CACAR I	CACAR II	AMAP 1998	Wagemann 1996	Chan 1995
		Ringe	d Seals		
Meat	Cd = 0.51, n = 1-2 Pb = 0.11, n = 1-2 Based on averages of individual samples presented by Chan et al (1995). No information on animal age, data from a single community (Qikiqtarjuaq). Data collected in 1987-1988.	01101111		Cd = 0.098, n = 35 Pb = 0.009, n = 53 Data collected from various locations across the eastern Arctic, not specific to Arctic Bay, from 1989 to 1993. Mean animal ages of 4.2 – 12.9 years.	Refer to CACAR I.
			0.008 (n = 9, age = 0-1) 0.02 (n = 1, age = 2-4) 0.05 (n = 1, age = 5-10) 0.01 (n = 1, age = 10-15) 0.03 (n = 1, age = >15) 0.011 (n = 2, age = 0-1) 0.006 (n = 10, age = 5-10) 0.005 (n = 2, age = 10-15) 0.006 (n = 1, age = >15) Data from Arctic Bay, Admiralty Inlet, and Nanisivik collected between 1974 and 1983.		

Table 1 Cadmium and Lead Concentrations in Marine Mammals (μg/g wet weight)

	CACAR I	CACAR II	AMAP 1998	Wagemann 1996	Chan 1995
Liver	Cd = 2.14	Cd = 5.13, n = 25	Cd	Cd = 11.9, n = 115	
	Pb = 0.033	Pb = 0.014, n = 25	8.35 (n = 17, age = 6.7)	Pb = 0.013, n = 140	
		, , ,	10.1 (n = 15, age = 8.2)	,	
	Comments as	Average age of	0.99 (n = 9, age = 0-1)	Data collected from	
	above.	animals was 8.1	2.09 (n = 1, age = 2-4)	various locations	
		years, data collected	6.05 (n = 1, age = 5-10)	across the eastern	
		specifically from	4.17 (n = 1, age = 10-15)	Arctic, not specific to	
		Arctic Bay in 1998-	8.73 (n = 1, age = >15)	Arctic Bay, from	
		2000.	0.25 (n = 2, age = 0-1)	1989 to 1993. Mean	
			12.68 (n = 10, age = 5-10)	animal ages of 4.2 –	
			8.97 (n = 2, age = 10-15)	12.9 years.	
			2.15 (n = 1, age = >15)		
			<u>Pb</u>		
			0.016 (n = 15, age = 8.2)		
			0.024 (n = 9, age = 0-1)		
			0.02 (n = 1, age = 2-4)		
			0.07 (n = 1, age = 5-10)		
			0.06 (n = 1, age = 10-15)		
			0.03 (n = 1, age = >15) 0.016 (n = 10, age = 5-10)		
			0.016 (ff = 10, age = 5-10) 0.023 (n = 2, age = 10-15)		
			0.014 (n = 1, age = >15)		
			,		
		Nan	Comments as above.		
Moot	No data	No data	Cd = 0.24, n = 27	Cd = 0.21, n = 56	
Meat	NO data	INO data	Pb = 0.05, n = 27	Pb = 0.008, n = 55	
			1 5 = 0.03, 11 = 21	1 0 = 0.000, 11 = 33	
			Data collected from	Data collected from	
			Admiralty Inlet in 1975. No	various locations	
			information on animal age.	across the eastern	
				Arctic, not specific to	
				Arctic Bay, from	
				1984 to 1994. No	
				information on	
				animal ages.	
Muktuk	Cd = 0.037	No data	Cd = 0.04, n = 11	Cd = 0.018, n = 48	
	Pb = 0.087		Pb = 0.27, n = 11	Pb = 0.002, n = 48	
	Comments as above		Data collected from	Data collected from	
			Admiralty Inlet in 1975. No	various locations	
			information on animal age.	across the eastern	
				Arctic, not specific to	
				Arctic Bay, from	
				1984 to 1994. No	
				information on	
				animal ages.	

Based on the above data, a selection hierarchy was developed, as follows:

- 1) specificity of data to Arctic Bay
- 2) adequacy of sample size and animal age distribution
- 3) dates of data collection

Table 2 provides the selected cadmium and lead marine mammal concentrations carried forward for the risk assessment based on the above data and selection hierarchy:

 Table 2
 Selected Marine Mammal Cd and Pb Concentrations (μg/g wet weight)

	Cadmium	Lead	Reference		
Ringed Seal					
Meat	0.075	0.016	AMAP 1998		
Liver	5.13	0.014	CACAR II 2003		
Narwhal					
Meat	0.24	0.05	AMAP 1998		
Muktuk	0.04	0.27	AMAP 1998		

Dietary Consumption

CACAR II reports that the daily consumption of country foods by Inuit people is 194 g/day and 245 g/day for females (20 – 40 years old) and males (20 – 40 years old), respectively. The sexes combined daily consumption rate of country foods is therefore 220 g/day. Richardson (1997) indicates that on a per-body-weight basis, consumption rates remain consistent across age groups. Therefore, the daily country food consumption rate for a 16.5 kg toddler is calculated to be 54.5 g/day, as follows:

Adult rate (220 g/day) \div 66.6 kg = 3.3 g/kg-day x toddler body weight (16.5 kg) = 54.5 g/day

Chan et al (1995) report that 52% (28 g/day) of country food consumption for Inuit children (3 – 12 years old) comes from marine mammal ingestion and provide a breakdown of each marine mammal food item, as follows:

ringed seal meat - 34.6%
narwhal muktuk - 8.2%
narwhal meat - 0.8%
ringed seal liver - 0.6%

Based on these percentages, the intake of each of the four marine mammal food types in g/day is as follows:

ringed seal meat - 21.8 g/day
narwhal muktuk - 5.3 g/day
narwhal meat - 0.6 g/day
ringed seal liver - 0.3 g/day
Total - 28 g/day

Marine Mammal Estimated Daily Intake

Based on the cadmium and lead concentrations in Table 2, the calculated consumption rates, and a toddler body weight of 16.5 kg, the dietary estimated daily intake from marine mammal consumption is provided in Table 3.

Table 3 Marine Mammal Cd and Pb Estimated Daily Intakes (µg/kg-day)

	Cadmium	Lead
Ringed Seal		
Meat	0.0991	0.0211
Liver	0.0933	0.0003
Narwhal		
Meat	0.0087	0.0018
Muktuk	0.0128	0.0867
TOTAL	0.2139	0.1099

Lead/Zinc - Residential

Compound	TDI	EDI	Ci	WGi site	WGi bkgd	WGi tot	THQ	BSC	AF i	AF d	AF s	AF g	Hazard Quotient	CCME (Res)
														(mg/kg)
Lead	0.00357	0.0004923	699	5.17	1.03	1.86	1	31	0.3	1	0.01	0.5	1.000	140
Zinc	0.3	0	10773	48.39	31.86	35.17	0.2	29	0.8	1	0.01	0.8	0.199	200

<u>Parameter</u>	Definition (units)	Default Value	Reference
Ci =	Soil concentration (mg/kg)	site-specific	
WGi site =	Wild game tissue concentration (mg/kg)	site-specific	
WGi bkgd =	Background wild game tissue concentration (mg/kg)	regional	
WGi tot =	Weighted wild game tissue concentration (mg/kg)	calculated	
TDI =	reference dose - oral (mg/kg bw-day)	chemical specific	bold = CCME; underline = IRIS
EDI =	estimated daily intake (multimedia exposure assessment) (mg/kg bw-day)	chemical specific	CCME (1-4 yrs)
SAF =	soil allocation factor (unitless)	chemical specific	
BW =	body weight (kg)	16.5	CCME (1-4 yrs)
BSC =	background soil concentration (mg/kg)	chemical specific	Site specific
AFg =	absorption factor for gut (unitless)	chemical specific	
AF i =	absorption factor for soil (unitless)	chemical specific	
AF d =	absorption factor for lung (unitless)	chemical specific	
AF s =	absorption factor skin (unitless)	chemical specific	EPA Region 3
GR =	wild game ingestion rate (kg/day)	0.018	Richardson (1997)
IR =	soil ingestion rate (kg/day)	0.00008	CCME (1-4 yrs)
DR =	soil inhalation rate (kg/day)	6.417E-09	calculated
SR =	soil dermal contact rate (kg/day)	0.000301	calculated
ET i =	exposure term for soil ingestion pathway (unitless)	0.334	Site Specific [(122 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET d =	exposure term for soil inhalation pathway (unitless)	0.334	Site Specific [(122 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET s =	exposure term for soil dermal contact pathway (unitless)	0.334	Site Specific [(122 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET wg =	exposure term for wild game ingestion pathway (unitless)	1.000	Site Specific [(365 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
TSP =	total suspended particulate matter in air (kg/m3)	6.9E-10	calculated
IR air =	daily inhalation rate (m3/day)	9.3	CCME (1-4 yrs)
SA =	skin surface area (cm2/day)	3010	CCME (1-4 yrs)
M =	soil to skin adherence factor (mg/cm2)	0.1	CCME (1-4 yrs)
FRP=	fraction respirable particles	1	US EPA
Pe =	particulate emission rate (kg/m2/s)	6.9E-13	Atlantic RBCA
L =	Length of soil parallel to wind (m)	1000	Site Specific
Uair =	Average wind speed (m/s)	1	Site Specific
ht =	Height of air breathing zone (m)	1	Site Specific

Lead/Zinc - Hunting

Compound	TDI	EDI	Ci	WGi site	WGi bkgd	WGi tot	THQ	BSC	AF i	AF d	AF s	AF g	Hazard Quotient	CCME (Res)
														(mg/kg)
Lead	0.00357	0.0004923	1052	6.39	1.03	2.10	1	31	0.3	1	0.01	0.5	0.999	140
Zinc	0.3	0	23224	51.09	31.86	35.71	0.2	29	0.8	1	0.01	0.8	0.199	200

<u>Parameter</u>	<u>Definition (units)</u>	Default Value	Reference
Ci =	Soil concentration (mg/kg)	site-specific	
WGi site =	Wild game tissue concentration (mg/kg)	site-specific	
WGi bkgd =	Background wild game tissue concentration (mg/kg)	regional	
WGi tot =	Weighted wild game tissue concentration (mg/kg)	calculated	
TDI =	reference dose - oral (mg/kg bw-day)	chemical specific	bold = CCME; underline = IRIS
EDI =	estimated daily intake (multimedia exposure assessment) (mg/kg bw-day)	chemical specific	CCME (1-4 yrs)
SAF =	soil allocation factor (unitless)	chemical specific	
BW =	body weight (kg)	16.5	CCME (1-4 yrs)
BSC =	background soil concentration (mg/kg)	chemical specific	Site specific
AFg =	absorption factor for gut (unitless)	chemical specific	
AF i =	absorption factor for soil (unitless)	chemical specific	
AF d =	absorption factor for lung (unitless)	chemical specific	
AF s =	absorption factor skin (unitless)	chemical specific	EPA Region 3
GR =	wild game ingestion rate (kg/day)	0.018	Richardson (1997)
IR =	soil ingestion rate (kg/day)	0.00008	CCME (1-4 yrs)
DR =	soil inhalation rate (kg/day)	6.417E-09	calculated
SR =	soil dermal contact rate (kg/day)	0.000301	calculated
ET i =	exposure term for soil ingestion pathway (unitless)	0.164	Site Specific [(60 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET d =	exposure term for soil inhalation pathway (unitless)	0.164	Site Specific [(60 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET s =	exposure term for soil dermal contact pathway (unitless)	0.164	Site Specific [(60 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET wg =	exposure term for wild game ingestion pathway (unitless)	1.000	Site Specific [(365 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
TSP =	total suspended particulate matter in air (kg/m3)	6.9E-10	calculated
IR air =	daily inhalation rate (m3/day)	9.3	CCME (1-4 yrs)
SA =	skin surface area (cm2/day)	3010	CCME (1-4 yrs)
M =	soil to skin adherence factor (mg/cm2)	0.1	CCME (1-4 yrs)
FRP=	fraction respirable particles	1	US EPA
Pe =	particulate emission rate (kg/m2/s)	6.9E-13	Atlantic RBCA
L =	Length of soil parallel to wind (m)	1000	Site Specific
Uair =	Average wind speed (m/s)	1	Site Specific
ht =	Height of air breathing zone (m)	1	Site Specific

Cadmium - Residential

Compound	TDI	EDI	Ci	WGi site	WGi bkgd	WGi tot	THQ	BSC	AF i	AF d	AF s	AF g	Hazard Quotient	CCME (Res)
														(mg/kg)
Cadmium	0.001	0.0006734	34	0.48	0.08	0.16	1	0.71	0.1	1	0.01	0.1	1.000	140

<u>Parameter</u>	Definition (units)	Default Value	Reference
Ci =	Soil concentration (mg/kg)	site-specific	
WGi site =	Wild game tissue concentration (mg/kg)	site-specific	
WGi bkgd =	Background wild game tissue concentration (mg/kg)	regional	
WGi tot =	Weighted wild game tissue concentration (mg/kg)	calculated	
TDI =	reference dose - oral (mg/kg bw-day)	chemical specific	CCME
EDI =	estimated daily intake (multimedia exposure assessment) (mg/kg bw-day)	chemical specific	CCME (1-4 yrs)
SAF =	soil allocation factor (unitless)	chemical specific	
BW =	body weight (kg)	16.5	CCME (1-4 yrs)
BSC =	background soil concentration (mg/kg)	chemical specific	Site specific
AFg =	absorption factor for gut (unitless)	chemical specific	
AF i =	absorption factor for soil (unitless)	chemical specific	
AF d =	absorption factor for lung (unitless)	chemical specific	
AF s =	absorption factor skin (unitless)	chemical specific	EPA Region 3
GR =	wild game ingestion rate (kg/day)	0.018	Richardson (1997)
IR =	soil ingestion rate (kg/day)	0.00008	CCME (1-4 yrs)
DR =	soil inhalation rate (kg/day)	6.417E-09	calculated
SR =	soil dermal contact rate (kg/day)	0.000301	calculated
ET i =	exposure term for soil ingestion pathway (unitless)	0.334	Site Specific [(122 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET d =	exposure term for soil inhalation pathway (unitless)	0.334	Site Specific [(122 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET s =	exposure term for soil dermal contact pathway (unitless)	0.334	Site Specific [(122 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET wg =	exposure term for wild game ingestion pathway (unitless)	1.000	Site Specific [(365 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
TSP =	total suspended particulate matter in air (kg/m3)	6.9E-10	calculated
IR air =	daily inhalation rate (m3/day)	9.3	CCME (1-4 yrs)
SA =	skin surface area (cm2/day)	3010	CCME (1-4 yrs)
M =	soil to skin adherence factor (mg/cm2)	0.1	CCME (1-4 yrs)
FRP=	fraction respirable particles	1	US EPA
Pe =	particulate emission rate (kg/m2/s)	6.9E-13	Atlantic RBCA
L =	Length of soil parallel to wind (m)	1000	Site Specific
Uair =	Average wind speed (m/s)	1	Site Specific
ht =	Height of air breathing zone (m)	1	Site Specific

Cadmium - Hunting

Compound	TDI	EDI	Ci	WGi site	WGi bkgd	WGi tot	THQ	BSC	AF i	AF d	AF s	AF g	Hazard Quotient	CCME (Res)
														(mg/kg)
Cadmium	0.001	0.0006734	52	0.59	0.08	0.18	1	0.71	0.1	1	0.01	0.1	1.000	14

<u>Parameter</u>	Definition (units)	Default Value	Reference
Ci =	Soil concentration (mg/kg)	site-specific	
WGi site =	Wild game tissue concentration (mg/kg)	site-specific	
WGi bkgd =	Background wild game tissue concentration (mg/kg)	regional	
WGi tot =	Weighted wild game tissue concentration (mg/kg)	calculated	
TDI =	reference dose - oral (mg/kg bw-day)	chemical specific	CCME
EDI =	estimated daily intake (multimedia exposure assessment) (mg/kg bw-day)	chemical specific	CCME (1-4 yrs)
SAF =	soil allocation factor (unitless)	chemical specific	
BW =	body weight (kg)	16.5	CCME (1-4 yrs)
BSC =	background soil concentration (mg/kg)	chemical specific	Site specific
AFg =	absorption factor for gut (unitless)	chemical specific	
AF i =	absorption factor for soil (unitless)	chemical specific	
AF d =	absorption factor for lung (unitless)	chemical specific	
AF s =	absorption factor skin (unitless)	chemical specific	EPA Region 3
GR =	wild game ingestion rate (kg/day)	0.018	Richardson (1997)
IR =	soil ingestion rate (kg/day)	0.00008	CCME (1-4 yrs)
DR =	soil inhalation rate (kg/day)	6.417E-09	calculated
SR =	soil dermal contact rate (kg/day)	0.000301	calculated
ET i =	exposure term for soil ingestion pathway (unitless)	0.164	Site Specific [(60 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET d =	exposure term for soil inhalation pathway (unitless)	0.164	Site Specific [(60 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET s =	exposure term for soil dermal contact pathway (unitless)	0.164	Site Specific [(60 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
ET wg =	exposure term for wild game ingestion pathway (unitless)	1.000	Site Specific [(365 d/yr x 24 hr/d) / (365 d/yr x 24 hr/d)]
TSP =	total suspended particulate matter in air (kg/m3)	6.9E-10	calculated
IR air =	daily inhalation rate (m3/day)	9.3	CCME (1-4 yrs)
SA =	skin surface area (cm2/day)	3010	CCME (1-4 yrs)
M =	soil to skin adherence factor (mg/cm2)	0.1	CCME (1-4 yrs)
FRP=	fraction respirable particles	1	US EPA
Pe =	particulate emission rate (kg/m2/s)	6.9E-13	Atlantic RBCA
L =	Length of soil parallel to wind (m)	1000	Site Specific
Uair =	Average wind speed (m/s)	1	Site Specific
ht =	Height of air breathing zone (m)	1	Site Specific

Risk Specific Soil Quality Guideline for Human Health for Non-Threshold (carcinogenic) Substances

Cadmium - Inhalation exposure, Town Area

SFi **BSC SQC-adult SQC-adjusted CCME** Compound AF d 1/mg/kg-d mg/kg (mg/kg) (mg/kg) (mg/kg) Cadmium 4.3E+01 0.71 443 368 14 1

CHECK:

IILCR			
Cs = 4.74			
1.29E-08			

Parameter	Definition (units)	Default Value	Reference
SF i =	inhalation slope factor [1/(mg/kg-day)]	chemical specific	Health Canada
TR =	target risk	1.00E-06	CCME
BW =	body weight (kg)	70.7	CCME
BSC =	background soil concentration	chemical specific	Ontario Typical Range - rural parkland
AF d =	absorption factor for lung (unitless)	chemical specific	US EPA
$\mathbf{DR} =$	soil inhalation rate (kg/day)	1.1178E-08	calculated
ET d =	exposure term for soil inhalation pathway (unitless)	0.334	Site Specific (24 hrs/d x 122 d/yr x 70 yrs) / (24 hrs/d x 365 d/yr x 70 yrs)
IR air =	daily outdoor inhalation rate, outdoor (m3/day)	16.2	CCME
TSP =	ambient total suspended particulate matter in air (kg/m3)	6.9E-10	calculated
DRadj =	age adjusted soil inhalation rate (m3-yr/kg-d)	19.3	Age adjusted $(0-0.5 \text{ yrs}) + (0.5-5 \text{ yrs}) + (5-11 \text{ yrs}) + (12-19 \text{ yrs}) + (20-70 \text{ yrs})$
$\mathbf{EF} \mathbf{d} =$	exposure frequency for soil inhalation (d/yr)	122	Site Specific 122 d/yr x (24 hrs/d / 24 hrs/d)
ATc =	averaging time for carcinogens (d)	25550	CCME

Risk Specific Soil Quality Guideline for Human Health for Non-Threshold (carcinogenic) Substances

Cadmium - Inhalation exposure, General Mine Area

CHECK:

Compound	SFi	BSC	AF d	SQC-adult	SQC-adjusted	CCME
	1/mg/kg-d	mg/kg		(mg/kg)	(mg/kg)	(mg/kg)
Cadmium	4.3E+01	0.71	1	900	748	14

IILCR
Cs = 5.75
7.69E-09

Parameter	<u>Definition (units)</u>	Default Value	Reference
CE:	11.1.4	1	Harld Comple
SF i =	inhalation slope factor [1/(mg/kg-day)]	chemical specific	Health Canada
TR =	target risk	1.00E-06	CCME
BW =	body weight (kg)	70.7	CCME
BSC =	background soil concentration	chemical specific	Ontario Typical Range - rural parkland
AF d =	absorption factor for lung (unitless)	chemical specific	US EPA
$\mathbf{DR} =$	soil inhalation rate (kg/day)	1.1178E-08	calculated
ET d =	exposure term for soil inhalation pathway (unitless)	0.164	Site Specific (24 hrs/d x 60 d/yr x 70 yrs) / (24 hrs/d x 365 d/yr x 70 yrs)
IR air =	daily outdoor inhalation rate, outdoor (m3/day)	16.2	CCME
TSP =	ambient total suspended particulate matter in air (kg/m3)	6.9E-10	calculated
DRadj =	age adjusted soil inhalation rate (m3-yr/kg-d)	19.3	Age adjusted (0-0.5 yrs) + (0.5-5 yrs) + (5-11 yrs) + (12-19 yrs) + (20-70 yrs)
$\mathbf{EF} \ \mathbf{d} =$	exposure frequency for soil inhalation (d/yr)	60	Site Specific 60 d/yr x (24 hrs/d / 24 hrs/d)
ATc =	averaging time for carcinogens (d)	25550	CCME

NANISIVIK HUMAN HEALTH RISK ASSESSMENT SAMPLE CALCULATION LEAD INTAKES IN THE TOWN SITE

SQRO Calculation

Hazard Quotient (HQ) =
$$\frac{CDI}{(TDI - EDI)}$$

where:

CDI = chronic daily intake

= sum of all site-specific intakes

TDI = tolerable daily intake EDI = estimated daily intake

= sum of all non-site related ambient intakes

When HQ = 1, the soil concentration = SQRO

Wild Game Ingestion

Calculation of Wild Game Tissue Concentration

 $WG_{conc} \ (mg/kg) \ = \ (GF_{site} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{soil})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_{bkgd})))) \ x \ WWCF)) + (GF_{bkgd} \ x \ ((\ EXP(-0.6114 + 0.5181(LN(C_$

where:

WG_{conc} = 1.86 (wild game tissue concentration, empirical regression equation from Sample et. al. 1998)

GF_{site} = 0.2 (fraction of wild game consumed by local residents that was caught on the Nanisivik mine site)

C_{soil} = 700 mg/kg (soil concentration set equal to the SQRO for the sample calculation)

WWCF = 0.32 (dry weight to wet weight conversion factor)

 GF_{bkgd} = 0.8 (fraction of wild game consumed by local residents that was caught in other areas) C_{bkgd} = 31 mg/kg (regional soil concentration set equal to the OTR₉₈ value for rural parkland)

Calculation of Intake from Wild Game Ingestion

$$WG_{intake} (mg/kg-d) = \underbrace{WG_{conc} \times IR_{game} \times ET_{game} \times AF_{oral}}_{BW}$$

where:

 $WG_{intake} = 1.01 \times 10^{-3}$ (intake from ingestion of wild game) $WG_{conc} = 1.86 \text{ mg/kg}$ (wild game tissue concentration)

IR_{game} = 0.018 kg/d (amount of wild game consumed daily by a toddler, Richardson 1997)

ET_{game} = 1 (exposure term for wild game ingestion)
AF_{oral} = 0.5 (bioavailability factor for dietary intakes)
BW = 16.5 kg (toddler body weight, Richardson 1997)

Drinking Water Ingestion

Calculation of Intake from Drinking Water Ingestion

$$DW_{intake} (mg/kg-d) = DW_{conc} \times IR_{water} \times ET_{water} \times AF_{oral}$$

where:

 $DW_{intake} = 1.09 \times 10^{-4}$ (intake from ingestion of drinking water)

DW_{conc} = 0.006 mg/L (EPC for Nanisivik town water supply, 1996-2001)

IR_{water} = 0.6 L/d (consumption rate for a toddler CCME, 2001)

ET_{water} = 1 (exposure term for drinking water ingestion)
AF_{oral} = 0.5 (bioavailability factor for dietary intakes)
BW = 16.5 kg (toddler body weight, Richardson 1997)

Soil/Dust Ingestion

Calculation of Intake from Soil/Dust Ingestion in the Summer

 $SD_{sum-ing intake}$ (mg/kg-d) = $C_{soil} \times IR_{soil ing} \times ET_{soil ing sum} \times AF_{soil ing}$

BW

where:

 $SD_{sum-ing\ intake} = 3.4 \times 10^{-4}$ (intake from soil/dust ingestion in the summer)

C_{soil} = 700 mg/kg (soil concentration set equal to the SQRO for the sample calculation)

IR_{soil ing} = 0.00008 kg/d (CCME default soil ingestion rate)

ET_{soil ing sum} = 0.334 (exposure term for summer soil ingestion = (24 hours/day x 122 days/year)/(24 hours/day x 365 days/year))

AF_{soil ing} = 0.3 (bioavailability factor for soil ingestion) BW = 16.5 kg (toddler body weight, Richardson 1997)

Calculation of Intake from Soil/Dust Ingestion in the Winter

IR_{dust} = SA_{finger} x DA x FR_{soil} x FR_{indoor} x WSF x FME x ET_{dust} x 10⁻⁶ kg/mg

where:

 $IR_{dust} = 3.86 \times 10^{-6} \text{ kg/day (dust ingestion rate)}$

SA_{finger} = 8.75 cm²/event (skin surface area of ½ of one finger, OMOE 1996)

DA = $0.1 \text{ mg/cm}^2 \text{ (dermal loading, CCME 2001)}$

FR_{soil} = 1 (fraction of dust that comes from soil, Hawley 1985) FR_{indoor} = 0.7 (fraction of outside dust levels indoors, Hawley 1985)

WSF = 0.1 (factor to account for winter snow cover and permafrost limiting soil dust re-suspension)

FME = 9 events/hour (number of mouthing events per hour, OMOE 1996)

ET_{dust} = 7 hours/day (hours of exposure per day, OMOE 1996)

 $SD_{wint-ing intake}$ (mg/kg-d) = $C_{soil} \times IR_{dust} \times ET_{dust ing wint} \times AF_{soil ing}$ BW

where:

 $SD_{wint-ing intake} = 3.27 \times 10^{-5}$ (intake from soil/dust ingestion in the winter)

C_{soil} = 700 mg/kg (soil concentration set equal to the SQRO for the sample calculation)

 IR_{dust} = 3.86 x 10⁻⁶ kg/d (calculated above)

ET_{dust ing wint} = 0.666 (exposure term for winter dust ingestion = (24 hours/day x 243 days/year)/(24 hours/day x 365 days/year))

 $AF_{soil ing}$ = 0.3 (bioavailability factor for soil ingestion) BW = 16.5 kg (toddler body weight, Richardson 1997)

Soil/Dust Dermal Contact

Calculation of Intake from Soil/Dust Dermal Contact in the Summer

 $SD_{summ derm intake}$ (mg/kg-d) = $C_{soil} \times SA_{body} \times SAF \times ET_{derm summ} \times AF_{dermal} \times 10^{-6}$ kg/mg

where:

 $SD_{summ derm intake} = 4.27 \times 10^{-5}$ (intake from soil/dust dermal contact in the summer)

C_{soil} = 700 mg/kg (soil concentration set equal to the SQRO for the sample calculation)

RW/

 SA_{body} = 3,010 cm²/day (skin surface area, CCME 2001)

SAF = 0.1 mg/cm^2 (soil to skin adherence factor, CCME 2001)

ET_{derm summ} = 0.334 (exposure term for summer soil contact = (24 hours/day x 122 days/year)/(24 hours/day x 365 days/year))

AF_{dermal} = 0.01 (bioavailability factor for soil dermal contact, US EPA 1992)

BW = 16.5 kg (toddler body weight, Richardson 1997)

Calculation of Intake from Soil/Dust Dermal Contact in the Winter

= C_{soil} x SA_{hands} x SAF x FR_{soil} x FR_{indoor} x WSF x ET_{derm wint} x AF_{dermal} x 10⁻⁶ kg/mg SD_{wint derm intake} (mg/kg-d)

where:

= 8.5 x 10⁻⁷ (intake from soil/dust dermal contact in the winter)

= 700 mg/kg (soil concentration set equal to the SQRO for the sample calculation) C_{soil}

= 430 cm²/day (skin surface area, CCME 2001) **SA**_{hands}

SAF = 0.1 mg/cm² (soil to skin adherence factor, CCME 2001) = 1 (fraction of dust that comes from soil, Hawley 1985) FR_{soil} FR_{indoor} = 0.7 (fraction of outside dust levels indoors, Hawley 1985)

WSF = 0.1 (factor to account for winter snow cover and permafrost limiting soil dust re-suspension)

= 0.666 (exposure term for winter soil contact = (24 hours/day x 243 days/year)/(24 hours/day x 365 days/year)) ET_{derm wint}

 $\mathsf{AF}_{\mathsf{dermal}}$ = 0.01 (bioavailability factor for soil dermal contact, US EPA 1992)

= 16.5 kg (toddler body weight, Richardson 1997) BW

Soil/Dust Inhalation

Calculation of Intake from Soil/Dust Inhalation in the Summer

TSP (kg/m³) Pe x L / Uair x ht

where:

TSP

 6.9×10^{-10} (total suspended particulate matter) 6.9×10^{-13} kg/m²/s (particulate emission rate, ASTM RBCA 1995) Pe

1,000 m (length of soil parallel to wind direction) L

Uair 1 m/s (average wind speed) 1 m (height of air breathing zone) Ht

= C_{soil} x FRP x TSP x IR_{inh} x ET_{inh summ} x AF_{inh} SD_{summ inh intake} (mg/kg-d)

ВW

where:

= 9.09×10^{-8} (intake from soil/dust inhalation in the summer)

= 700 mg/kg (soil concentration set equal to the SQRO for the sample calculation) C_{soil}

= 1 (fraction of the total suspended particulate that is respirable) **FRP**

= 6.9 x 10⁻¹⁰ kg/m³ (calculated above) **TSP**

= 9.3 m³/day (toddler inhalation rate, CCME 2001) IR_{inh}

0.334 (exposure term for summer soil inhalation = (24 hours/day x 122 days/year)/(24 hours/day x 365 days/year)) ET_{inh summ}

= 1 (bioavailability factor for soil inhalation) AF_{soil ing}

BW 16.5 kg (toddler body weight, Richardson 1997)

Calculation of Intake from Soil/Dust Inhalation in the Winter

C_{soil} x FRP x TSP x IR_{inh} x FR_{soil} x FR_{indoor} x WSF x ET_{inh wint} x AF_{inh} SD_{wint inh intake} (mg/kg-d)

where:

= 1.27 x 10⁻⁸ (intake from soil/dust inhalation in the winter) SD_{wint inh intake}

= 700 mg/kg (soil concentration set equal to the SQRO for the sample calculation) C_{soil}

= 1 (fraction of the total suspended particulate that is respirable) **FRP**

= $6.9 \times 10^{-10} \text{ kg/m}^3$ (calculated above) **TSP**

= 9.3 m³/day (toddler inhalation rate, CCME 2001) IR_{inh} = 1 (fraction of dust that comes from soil, Hawley 1985) FR_{soil} = 0.7 (fraction of outside dust levels indoors, Hawley 1985) FRindoor

WSF = 0.1 (factor to account for winter snow cover and permafrost limiting soil dust re-suspension) ET_{inh wint} = 0.666 (exposure term for winter soil inhalation = (24 hours/day x 243 days/year)/(24 hours/day x 365 days/year))

AF_{inh} = 1 (bioavailability factor for soil inhalation)

BW = 16.5 kg (toddler body weight, Richardson 1997)

Sum of all Intake Pathways

Intake_{total} = WG_{intake} + DW_{intake} + SD_{sum-ing intake} + SD_{summ derm intake} + SD_{summ inh intake}

 $= 1.54 \times 10^{-3} \text{ mg/kg-day}$

Calculation of Hazard Quotient

 $HQ = Intake_{total} / ((TDI - EDI) x AF_{oral})$

where:

 $AF_{oral} = 0.5$

 $HQ = 0.00154 / ((0.00357 - 0.000492) \times 0.5)$

HQ = 1

EDI = Air + Supermarket Food + Marine Mammal Consumption = 0.0066 + 0.3758 + 0.1099

= $0.4923 \mu g/kg-day$ = $4.92 \times 10^{-4} mg/kg-day$

Based on the above sample calculation, a soil concentration of 700 mg/kg results in a hazard quotient equal to 1.

Therefore the Soil Quality Remedial Objective (SQRO) for lead in the town site is 700 mg/kg.